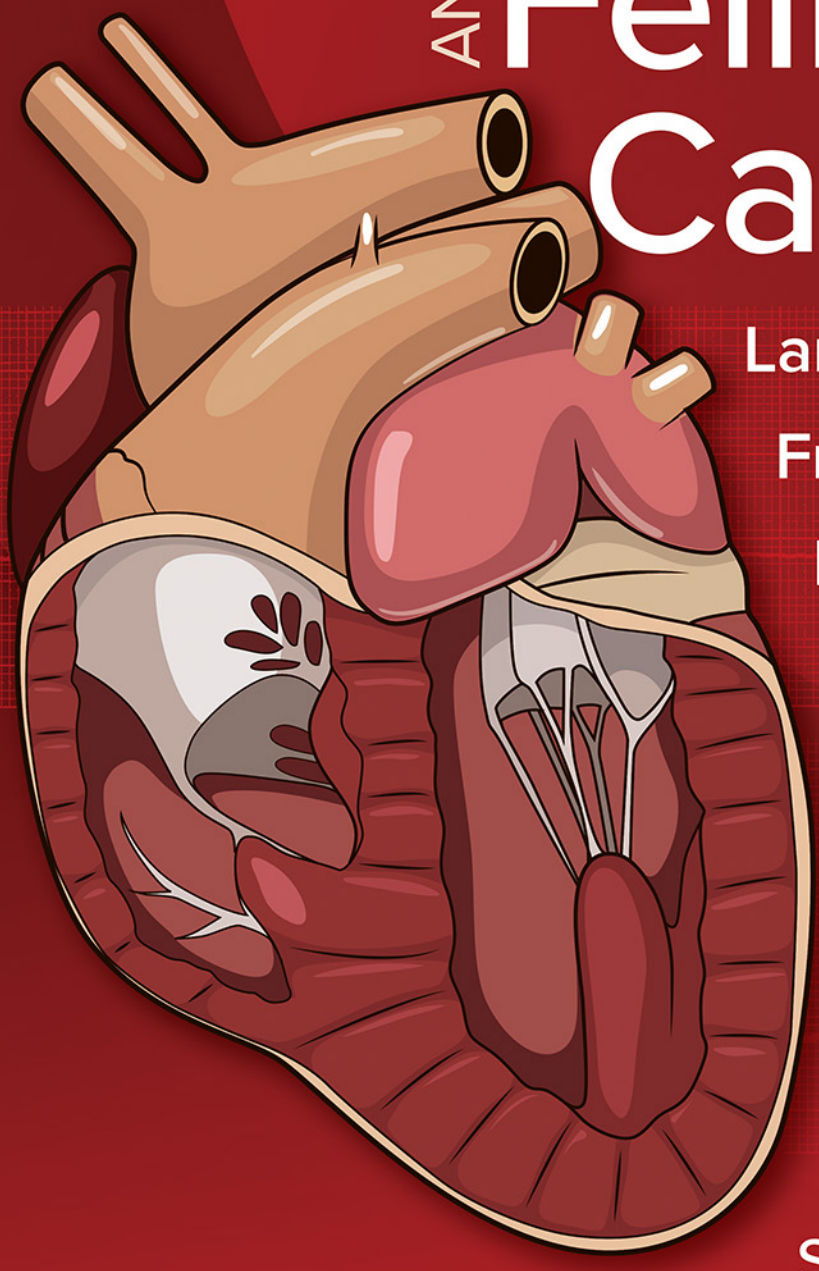
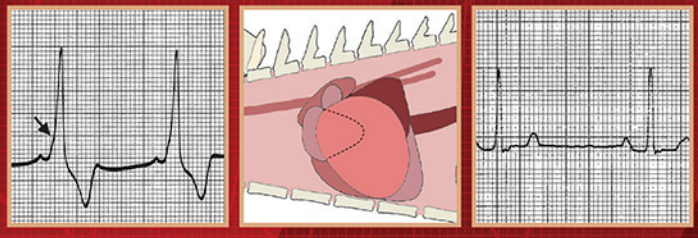
MANUAL OF Canine AND Feline Cardiology

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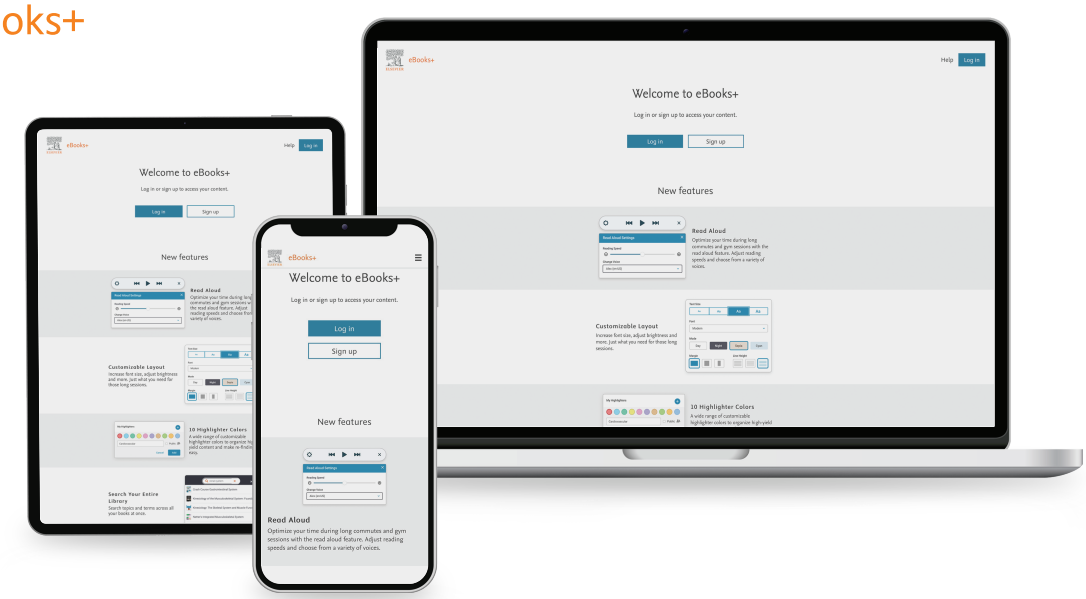


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MANUAL OF Canine AND Feline Cardiology

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MANUAL OF Canine AND Feline Cardiology

Sixth Edition



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For the many animals that have taught me and
made me the clinician and the person I am today.

Meg M. Sleeper

To my beloved family (Anna, Hannah, Sasha) both human and furry (Tobi).
Your love, support, and companionship have made this journey possible.
Thank you for being my constant sources of joy and inspiration.

Marc S. Kraus

To May, Ben, and Jade. Thanks for being a constant source of
inspiration and joy. Love always.

Francis W. K. Smith, Jr.

To my wife, Ellen who has opened in me reserves of strength
and love I never knew existed.
To my grandson, Tucker and animals who represent
the purity of life.

Larry P. Tilley

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Preface

In the last 8 years, there have been a number of excellent textbook chapters and journal articles describing new findings in the field of canine and feline cardiology. However, the amount of information is overwhelming for practicing veterinarians and veterinary students. Many veterinarians and students have stated, in past surveys, the need to keep up with cardiovascular advances in the most time-effective way. The *Manual of Canine and Feline Cardiology*, now in its sixth edition, has been updated to meet the need for a current textbook that can provide useful and practical information on cardiac disease in dogs and cats. We have worked hard to make this book a most reliable source for practicing cardiovascular medicine in dogs and cats. In this new edition, we have expanded the number of contributors, with each chapter written by an expert. We have also worked to conserve reading time by employing an easy-to-use format. This book is unique as a quick reference with consistency of presentation.

This practical approach to the diagnosis and therapy of cardiac disease will be useful to a wide audience, from the veterinary student to the fully trained cardiologist. The approach is largely clinical and includes practical and the most sophisticated methods for diagnosis and therapy.

Cardiovascular disorders in dogs and cats represent a substantial portion of diseases seen in the average veterinary practice. It is important for veterinary students and practitioners to understand the principles of diagnosis and treatment of several cardiovascular disorders.

NEW TO THIS EDITION

Since the fifth edition of this textbook was published, there has been tremendous growth in the field of canine and feline cardiology. The sixth edition has been carefully revised, edited, and updated and also has a generous amount of new figures and charts, with the majority in color.

ORGANIZATION

Section 1 describes the methods for diagnosing heart diseases in dogs and cats. The first five chapters follow the sequence that the veterinarian uses in approaching the patient: History and Physical Examination (Chapter 1), Radiology of the Heart (Chapter 2), Electrocardiography (Chapter 3), Echocardiography and Doppler Ultrasound (Chapter 4), and Special Diagnostic Techniques for the Evaluation Of Cardiac Diseases (Chapter 5).

Section 2 presents, in a step-by-step fashion, a description of the various cardiac disorders that occur in dogs and cats, starting in each chapter with general considerations (e.g., definitions, incidence, pathophysiology, etiology), history, physical examination, electrocardiography, thoracic radiography, special diagnostic techniques, differential diagnosis, and, finally, the therapeutic approach.

Section 3 includes chapters describing treatment of heart failure, the treatment of cardiac arrhythmias and conduction disturbances, and two separate chapters on cardiopulmonary arrest and resuscitation, and emergency management and critical care in cardiology. Chapters on anesthesia of the cardiac patient, cardiac surgery, pacemaker therapy, and nutrition and cardiovascular disease complete this section.

Appendix 1, Canine and Feline Breed Predilections for Heart Disease, represents the most current listing of breed-associated cardiac disorders.

Appendix 2, Cardiopulmonary Drug Formulary contains, extensive cardiopulmonary drug tables with indications, dosages, side effects, contraindications, and drug interactions.

Appendix 3, Echocardiographic Normal Values, by body weight and breed, represents an easy-to-refer-to list of normal values.

Appendix 4, Available Canine and Feline Genetic Tests for Breed-Specific Cardiac Disease.

The *Manual of Canine and Feline Cardiology*, sixth edition, will help to eliminate the aura of mystery that surrounds the diagnostic and therapeutic principles of veterinary cardiology. The teaching principles that are presented will allow even the novice to make an intelligent assessment of a cardiac case. This manual will be useful to a wide audience but comprehensive enough to serve as a reference for the advanced student and the veterinarian with expertise in cardiology.

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The completion of this manual provides a welcome opportunity to recognize many individuals who have written this textbook. The authors have board certification and are uniquely qualified to provide updates and reviews of their respective disciplines.

We would like to express our gratitude to every veterinary student, intern, and resident whom we have had the pleasure of teaching. Their curiosity and intellectual stimulation have enabled us to grow and inspired us to write this manual.

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Chapter 1

History and Physical Examination

Alexandra V. Crooks

Despite the technical nature of many cardiovascular diagnostics, such as electrocardiography, radiography, and echocardiography, history and physical examination remain the most crucial initial steps in the establishment of the correct diagnosis. Findings from a careful history and physical examination prompt the clinician to consider the probability or presence of heart disease. Results of the cardiovascular physical examination will usually allow the clinician to make a tentative diagnosis or formulate a specific differential diagnosis. The history and physical examination also provide important information regarding the stage of heart disease present, which may significantly affect therapy.

A good history and physical examination are invaluable in making a diagnosis of heart disease and helping to differentiate heart disease from pulmonary disease. Besides helping to make the diagnosis, a good history and physical examination help to determine the extent of the problem, the animal's response to previous therapy, the owner's ability to medicate the animal consistently, and the presence of other medical problems. Clinicians must obtain a thorough history and physical examination to properly diagnose and treat an animal with heart disease.

MEDICAL HISTORY

SIGNALMENT

Age

Young animals usually have congenital diseases (e.g., patent ductus arteriosus [PDA]), whereas older animals usually have acquired diseases, such as degenerative diseases (e.g., degenerative mitral valve disease) or neoplastic diseases (e.g., heart base tumor). There are exceptions because cardiomyopathies can occur in young dogs and cats (aged 6 months or younger) and older dogs and cats can have congenital heart defects that were not diagnosed when they were young (e.g., valvular dysplasia, atrial septal defect). Also cardiac disease in older animals can be modified or affected by other concurrent disease processes (e.g., collapsing trachea or other respiratory diseases and renal or liver disease).

Breed

Certain cardiac defects are more common in some breeds of animals; however, there can be regional differences in the rate of occurrence of cardiac problems. See the Appendix for a summary of some of the cardiac defects found in certain breeds of dogs and cats.

Sex

Males are more susceptible to certain cardiac diseases (e.g., small-breed male dogs to degenerative mitral valve disease, large-breed male dogs to dilated cardiomyopathy, male cats to hypertrophic cardiomyopathy). However, sick sinus syndrome occurs more commonly in the female Miniature Schnauzer, and PDA is more common in females of small-breed dogs.

Weight

The animal's weight influences several aspects of treatment, including the dose of cardiac medication, evaluation of the response to diuretic medication, and monitoring of cardiac cachexia. A Pickwickian syndrome (characterized by severe obesity, somnolence, and hypoventilation) can occur in an animal that is so obese that its ability to breathe is restricted.

Utilization of the Animal

Knowing how an animal is going to be used when giving a long-term prognosis for a cardiac disease is important. For example, hunting dogs with severe heartworm disease may not be able to hunt again after treatment. Also, some animals with congenital heart defects may have normal life spans and may make good pets; however, they should not be used for breeding purposes because the defect could be perpetuated.

The age, breed, and sex of the animal may help the clinician to make an accurate diagnosis; however, there are always exceptions to every rule. Clinicians should not ignore the fact that an animal could have an atypical problem for its age, breed, or sex.

HISTORY

- A good history will establish the presence of a cardiac problem, help to differentiate between cardiac and respiratory problems, and help to monitor the disease course and the response to therapy. It must

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be done carefully to prevent an owner from giving a misleading history. It includes several key questions, such as the reason the animal is being presented, the problems noted by the owner, the onset and duration of the problem(s), the progression of the disease, any known exposure to infectious diseases, the vaccination history, any current medications (including doses) the animal is receiving, the animal's response to any medications that have been given, and the owner's ability to give the medication(s).

- The history will also define the animal's attitude and behavior because the owner will be asked whether the animal is listless and depressed or alert and playful. Does the animal tire easily with exercise?
- The patient's history should also cover the family history of the siblings and parents, especially if congenital disease is present in the patient. The owner will be asked about the health of other pets in the household, and the history will include the results of previous tests done on the patient.
- The history covers other relevant information that may help to identify the patient's problem(s). How much water is the patient drinking, and how frequently are they urinating? Is there any vomiting/regurgitation or diarrhea? Has the patient had any seizures or syncopal episodes? What is the patient's reproductive status? Does the patient have any lameness or paresis? Is the patient coughing, sneezing, or having difficulty breathing? Does the animal rest comfortably, or is it restless? Has the patient had any previous trauma? Where is the animal housed (e.g., indoors, outdoors, fenced-in yard)? Does the animal have any diseases such as hyperthyroidism, chronic renal disease, respiratory disease, or other diseases that can affect the heart or the treatment of the animal's heart disease?
- The history should also include appetite and the type and quantity of food being fed. A detailed diet history is important in identifying issues that could be contributing to underlying heart disease (e.g., taurine deficiency) or heart failure (e.g., high salt content). The diet history should include the main foods being fed (e.g., brand and flavor of commercial pet food recipes, recipe of a home-prepared diet) as well as treats, table food, chews, supplements, and foods used to administer medications.
- Recently, veterinarians have noticed an increased incidence of dilated cardiomyopathy (DCM) in atypical dog breeds. Many of these pets are receiving diets with legumes (peas, lentils, chickpeas) as their major ingredients. Some of these dogs may have taurine-deficient cardiomyopathy. Other dogs may have normal taurine levels and develop DCM due to a separate, but still unknown, dietary factor (see Chapter 7).
- Once a problem is identified by the history, more specific questions can be asked, including what is the character of the cough, when does it occur, and what stimuli evoke it. Common presenting complaints for cardiac disease include dyspnea or tachypnea; exercise intolerance; syncope; abdominal swelling; cyanosis; anorexia or decreased appetite; poor growth or performance; and a soft, moist cough if the animal has left-sided heart failure. Coughing by itself is *not* a primary complaint of heart disease.
- Other symptoms can be associated with cardiovascular disease. Polydipsia and polyuria are common in animals that are given diuretics or that have a concurrent disease (e.g., renal disease), whereas oliguria occurs with severe left-sided heart failure. Hemoglobinuria is found with the caval syndrome of heartworm disease.
- Cardiac drugs, such as digitalis, mexiletine, or angiotensin-converting enzyme (ACE) inhibitors (e.g., enalapril), can cause anorexia, vomiting, or diarrhea. Regurgitation occurs with congenital vascular ring anomalies. Right-sided heart failure can cause intestinal edema and a protein-losing enteropathy, resulting in diarrhea. Cats with cardiomyopathy can have hemorrhagic enteritis resulting from thromboembolism of the gastric or mesenteric arteries.

Key Point

A good history will uncover all the animal's symptoms and problems, whether they are due to a cardiac problem or to another concurrent problem. Also, by the end of a good history, an astute clinician will have a good idea as to the potential causes of the animal's problems.

SPECIFIC SYMPTOMS

Coughing

- Coughing in dogs may be caused by left-sided congestive heart failure (pulmonary edema) or compression of the trachea and/or main stem bronchi secondary to heart enlargement. However, concurrent primary respiratory disease (chronic lower airway disease, heartworm disease) is also a common cause of coughing, rather than heart disease itself. Cats rarely cough, even when they have an enlarged left atrium.
- Coughing is a sudden, forced expiration and is a normal defense mechanism to clear debris from the tracheobronchial tree. It can originate from the larynx, pharynx, trachea, and bronchi but not from the smaller bronchi, bronchioles, and alveoli. A cardiac cough caused by left-sided heart failure occurs only when the pulmonary edema is peracute and severe enough that fluid accumulates in the airways. This cough is soft and moist sounding and may produce blood-tinged sputum. An enlarged left atrium (in the absence of left-sided heart failure) may contribute to

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Table 1.1 Characteristics of Coughs and Their Associated Causes in Dogs and Cats

TYPE OF COUGH	CAUSES
Acute	Tonsillitis, pharyngitis, tracheobronchitis, acute bronchitis, pleuritis, pneumonia, acute severe left-sided heart failure (dogs only)
Chronic	Heartworms, enlarged left atrium compressing trachea or main stem bronchus (dogs only), pulmonary neoplasia, asthma (cats only), chronic respiratory problems, chronic bronchitis (dogs only)
Acute onset, soft, moist that rapidly becomes worse in dogs with dyspnea	Pulmonary edema
Mild, intermittent cough, harsh, low pitched in dogs	Respiratory diseases
Loud, harsh, dry, sudden onset followed by gag in dogs	Tracheobronchitis
Honking, high pitched in dogs	Collapsing trachea or bronchi
Chronic, paroxysmal, loud, honking with excitement in dogs	Large airway disease
After drinking in dogs	Collapsing trachea, chronic tracheitis, tracheobronchitis, laryngeal paralysis, dysphagia
After eating in dogs	Pharyngeal dysfunction, megaesophagus, vascular ring anomalies, esophageal diverticula, esophageal foreign bodies, esophageal tumors
Without an inciting factor	Pulmonary or extrapulmonary disease

coughing by mechanical dorsal compression of the trachea or main stem bronchi, resulting in a cough of airway collapse. Table 1.1 lists some of the characteristic coughs in dogs and cats and their associated causes.

- Dogs and cats with pulmonary edema (cardiac or noncardiogenic) will have tachypnea or dyspnea and may have an acute onset of soft, moist coughing. Dogs with fulminant left-sided heart failure may have pink foam in the mouth and nose and be dyspneic, but they may or may not be coughing.
- Dogs with coughing as their primary problem should be radiographed and undergo other tests to find the cause because it is uncommon for coughing alone to be due to heart disease or left-sided heart failure. Dogs with a moist, productive cough may have pneumonia. Dogs with a loud, harsh, dry cough of sudden onset followed by a nonproductive gag commonly have tracheobronchitis. Dogs with a honking, high-pitched cough often have a collapsing trachea or collapsed bronchi. Small breeds of dogs with large airway disease will have a chronic, paroxysmal cough that is hard, loud, and honking and usually occurs with excitement. Dogs that cough after drinking may have cardiac disease, collapsing trachea, chronic tracheitis, tracheobronchitis, laryngeal paralysis, or other causes of dysphagia. Dogs that cough without an inciting factor may have pulmonary or extrapulmonary disease. Dogs that cough after eating have pharyngeal dysphagia, megaesophagus, vascular ring anomalies, esophageal diverticula, esophageal foreign bodies, or esophageal tumors.
- Identifying the cause of the dog's coughing can be difficult. Radiographs, echocardiograms, fluoroscopy, transtracheal wash, and bronchoscopy should be done to try to find the cause. A cough that seems to respond to a therapeutic course of furosemide does not mean that the dog had heart failure because furosemide has both antiinflammatory and antitussive properties.
- It is unusual for cats with either left- or right-sided congestive heart failure to cough; however, they will cough with heartworm disease and with respiratory diseases such as asthma. If the clinician compresses the cat's trachea and the cat has a prolonged bout of coughing, then coughing is likely to be part of the cat's problem, and underlying respiratory problems should be pursued.

Key Point

The most common cause of coughing in dogs with murmurs is respiratory disease. Coughing is *not* caused by pulmonary edema unless the dog or cat is also tachypneic or dyspneic.

Dyspnea

- Dyspnea is difficult, labored, or painful breathing. It is usually preceded by tachypnea (an increased rate of breathing), which owners may miss. It is a good idea to have the owner of a patient with cardiac disease learn to count their pet's respiratory rate at rest or sleeping. The respiratory rate should be less than 30 breaths per minute in a dog at rest or sleeping,

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and if it goes over 40 breaths per minute, then the dog has tachypnea and should be examined by a veterinarian. Respiratory rate and effort have been shown to be the most reliable indication of left-sided heart failure and pulmonary edema.

- Dyspnea will occur whenever anything increases the amount of air that must be breathed by the animal. Box 1.1 lists the problems that can cause dyspnea. The usual cardiac cause of dyspnea in dogs is left-sided heart failure, which results in pulmonary edema. The usual cardiac cause of dyspnea in cats is left-sided heart failure, which results in either pulmonary edema or pleural effusion, or both. Right-sided heart failure resulting in pleural effusion (with or without abdominal effusion) can cause dyspnea in either species. In patients with cardiac disease, dyspnea can be accompanied by stridor, which is a harsh, high-pitched respiratory sound. Other sounds include rhonchi, which sound like dry, coarse crackles. Also dyspnea can be accompanied by wheezing, which is more typical of respiratory problems than cardiac problems. Table 1.2 lists the different types of dyspnea and the associated diseases or problems.
- Different types of dyspnea can be associated with certain problems or diseases. Acute dyspnea is usually caused by pulmonary edema (cardiac and noncardiac), severe pneumonia, airway obstruction, pneumothorax, or pulmonary embolism. Chronic, progressive dyspnea is caused by left- or right-sided congestive heart failure, pericardial diseases, bronchial disease, lung diseases such as emphysema, pleural effusions, progressive anemia, and primary or secondary pulmonary neoplasia. Dyspnea at rest occurs with pneumothorax, pulmonary embolism, severe left- or right-sided heart failure, or pulmonary hypertension. Exertional dyspnea occurs after or during activity and can be associated with congestive heart failure, pulmonary hypertension, or chronic obstructive lung disease. Expiratory dyspnea is prolonged, and labored expiration is caused by lower respiratory tract obstruction or disease. Inspiratory dyspnea is prolonged, and labored inspiration is due to upper airway obstruction. Mixed dyspnea is due to severe pulmonary edema caused by left-sided heart failure or severe pneumonia.

Box 1.1 Causes of Dyspnea

Acidosis
Anemia
Central nervous system disorders
Excitement
High altitude
Hyperthyroidism (thyroid storm)
Pain
Pericardial effusions
Pleural effusions
Primary cardiac diseases causing pulmonary edema or pleural effusion
Pulmonary edema (noncardiogenic)
Pulmonary hypertension
Secondary cardiac diseases
Strenuous exercise
Thoracic wall problems (e.g., fractured ribs)

Table 1.2 Types of Dyspnea and Their Associated Diseases or Problems

TYPE OF DYSPNEA	DISEASE OR PROBLEM
Acute dyspnea	Pulmonary edema (cardiogenic and noncardiogenic), severe pneumonia, airway obstruction, pneumothorax, pulmonary embolism
Chronic, progressive dyspnea	Left- or right-sided heart failure, pericardial diseases, bronchial disease, lung diseases (e.g., emphysema), pleural effusions, progressive anemia, primary and secondary neoplasia, pulmonary hypertension
Dyspnea at rest	Pneumothorax, pulmonary embolism, severe left- or right-sided heart failure, pulmonary hypertension
Exertional dyspnea	Heart disease (e.g., dilated cardiomyopathy) or chronic obstructive lung disease, pulmonary hypertension
Expiratory dyspnea	Lower respiratory tract obstruction or disease
Inspiratory dyspnea	Upper airway obstruction
Mixed dyspnea	Pulmonary edema caused by left-sided heart failure or severe pneumonia
Orthopnea	Severe pulmonary edema, pericardial effusion, pleural effusion, diaphragmatic hernia, pneumothorax, severe pulmonary disease
Paroxysmal dyspnea	Arrhythmias (e.g., bradycardia or tachycardia)
Simple dyspnea or tachypnea	Fever, fear, pain, or excitement

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- Orthopnea means that dyspnea occurs when the animal is lying down but not when standing. In dogs, orthopnea typically includes elevation and stretching of the neck and abducting the forelimbs. In cats, orthopnea may include sitting in sternal recumbency with flexed pelvic limbs and extended thoracic limbs, the so-called sphinx pose. It is associated with severe pulmonary edema, pleural effusion, pericardial effusion, pneumothorax, diaphragmatic hernia, and severe respiratory problems. Paroxysmal dyspnea means that the dyspnea comes and goes. It can be associated with arrhythmias that cause either bradycardia or tachycardia. Simple dyspnea, or polypnea, is an increased rate of respiration caused by fever, fear, pain, or excitement. Cats with severe hyperthyroidism can also be dyspneic.
- Dyspnea that improves when treated with diuretics is suggestive of left-sided heart failure as the cause. Dyspnea that improves when treated with bronchodilators, antibiotics, or steroids is suggestive of respiratory disease as the cause.

Key Point

Dyspnea is a sign of significant cardiac, respiratory, or other systemic problems. It requires immediate diagnostic tests to identify the cause of the dyspnea so that specific therapy can be started. However, all tests should be done with minimal stress to the animal because these patients are fragile and stress could be fatal. Monitoring the animal's respiratory rate and effort at rest or during sleep is the most sensitive indicator of dyspnea and the onset of heart failure.

Hemoptysis

- Hemoptysis is coughing up of blood. It is uncommon in animals because they usually swallow their sputum. Hemoptysis is a sign of severe pulmonary disease. The causes of hemoptysis are listed in Box 1.2. Cardiac causes of hemoptysis include severe pulmonary edema (e.g., ruptured chordae tendineae) and severe heartworm disease, usually with pulmonary embolism.

Key Point

Hemoptysis is a sign of a very serious underlying abnormality in the lungs, which may be caused by either a severe cardiac or respiratory problem.

Syncope

- Syncope is a loss of consciousness caused by inadequate cerebral blood flow. It can recur and is usually brief. Syncope can be hard to differentiate from seizures. Animals usually fall over suddenly, get back up quickly, and are normal before and after the syncopal episode. Box 1.3 lists the causes of syncope in dogs and cats.
- In a dog with no other cardiac problems, syncope may be associated with severe bradycardias (e.g., third-degree heart block or sick sinus syndrome) or with marked sustained tachycardias (e.g., atrial or ventricular tachycardias) that are usually paroxysmal (i.e., they come and go with abrupt onset and termination).
- Small dogs with chronic, severe mitral regurgitation can have syncopal episodes following excitement (vasovagal or neurocardiogenic syncope) or coughing (vagally mediated syncope). Dogs with severe subaortic stenosis, pulmonic stenosis, pulmonary hypertension, or tetralogy of Fallot can have arrhythmias associated with their ventricular hypertrophy and myocardial hypoxia. These arrhythmias will cause syncope. Syncope can also occur in cats with severe hypertrophic cardiomyopathy due to

Box 1.2 Causes of Hemoptysis

Acute and chronic bronchitis
Chronic pulmonary granulomas
Clotting disorders
Disseminated intravascular coagulopathy
Lung abscesses
Lung lobe torsions
Oral or other neoplasia
Pulmonary embolism
Pulmonary fungal infections
Pulmonary neoplasia
Respiratory foreign bodies
Severe heartworm disease with pulmonary embolism
Severe pneumonia
Severe pulmonary edema (e.g., from ruptured chordae tendineae)
Trauma with severe pulmonary contusions

Box 1.3 Causes of Syncope in Dogs and Cats

Disease with poor cardiac output (e.g., dilated cardiomyopathy)
Severe bradycardia (e.g., complete heart block, sick sinus syndrome)
Severe sustained tachycardias (e.g., atrial or ventricular tachycardia)
Severe hypertrophic cardiomyopathy (usually with dynamic outflow obstruction or systolic anterior motion of the mitral valve) in cats
Systemic hypotension including arteriolar dilator therapy
Severe pulmonary hypertension
Severe subaortic stenosis
Severe pulmonic stenosis
Vagally mediated syncope (e.g., tussive syncope)
Vasovagal (neurocardiogenic) syncope
Tetralogy of Fallot

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either an arrhythmia or left ventricular outflow tract obstruction. Animals with poor cardiac output caused by dilated cardiomyopathy can have syncope, especially if they also have arrhythmias such as atrial fibrillation or ventricular premature beats that further reduce their cardiac output. Cardiac medications such as vasodilators, especially arterial dilators, can result in systemic hypotension, which can also cause syncope.

Key Point

Syncope must be distinguished from seizures with a careful history and physical examination. Having the owner videotape an episode can also help the clinician distinguish between the two. Further tests, such as Holter monitoring or event monitoring, may be necessary to determine whether an arrhythmia is causing the syncope.

Weakness and Exercise Intolerance

- Weakness and exercise intolerance are nonspecific signs of heart disease. Many diseases, such as severe anemia, systemic diseases, metabolic diseases (e.g., hyperadrenocorticism), drug toxicities, and severe respiratory diseases, can cause these signs. See Box 1.4 for causes of weakness and exercise intolerance.
- Because most animals do not exercise extensively, weakness and exercise intolerance are uncommon presenting complaints. Some owners think their animals are slowing down because of old age and not because of heart disease or other problems.
- Both complaints can be an early sign of decompensated heart failure because the heart cannot pump enough blood to the muscles on account of myocardial dysfunction (e.g., dilated cardiomyopathy or advanced mitral valve disease), obstruction to left ventricular outflow (e.g., subaortic stenosis or hypertrophic obstructive cardiomyopathy), inadequate ventricular filling (e.g., arrhythmias, pericardial diseases, hypertrophic cardiomyopathy), and decreased arterial oxygen (e.g., pulmonary edema or pleural effusion).

Box 1.4 Causes of Weakness and Exercise Intolerance

- Cardiac disease with myocardial dysfunction (e.g., dilated cardiomyopathy)
- Cardiac disease with obstruction to left ventricular outflow (e.g., subaortic stenosis, hypertrophic obstructive cardiomyopathy)
- Decreased arterial oxygen (e.g., pulmonary edema, pleural effusion, or other pulmonary diseases)
- Inadequate ventricular filling (e.g., arrhythmias, pericardial diseases)
- Drug toxicities
- Severe anemia
- Severe metabolic disease
- Severe respiratory diseases
- Severe systemic diseases

Key Point

Distinguishing between exercise intolerance caused by heart disease and other causes is important.

Ascites

- Ascites is an accumulation of fluid in the abdomen. Ascites caused by cardiac problems is caused by either the inability of the right side of the heart to pump the blood presented to it or to pericardial disease, which prevents the blood from entering the right side of the heart. In either case, venous pressure increases and eventually fluid leaks out of the vessels into the abdominal cavity, causing the ascites.
- Ascites is seen in dogs with right-sided heart failure because of acquired diseases (e.g., tricuspid regurgitation caused by chronic valvular heart disease, advanced heartworm disease, dilated cardiomyopathy, pericardial effusions, restrictive pericarditis) and congenital heart defects (e.g., tricuspid dysplasia, large ventricular septal defect, large atrial septal defect). See Fig. 1.1 for an example of ascites and Box 1.5 for a list of causes of ascites in dogs. Ascites is less common in cats but occasionally can be seen with tricuspid dysplasia or advanced cardiomyopathy.
- Large amounts of ascites will put pressure on the diaphragm, resulting in tachypnea or dyspnea. Ascites associated with right-sided heart failure is usually a transudate or protein-rich (modified) transudate and accumulates slowly.



Fig. 1.1 Notice the severe ascites in this dog with right-sided heart failure caused by severe pulmonic stenosis and severe tricuspid dysplasia.

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Box 1.5 Causes of Ascites in Dogs

- Advanced heartworm disease
- Dilated cardiomyopathy
- Large atrial septal defect
- Large ventricular septal defect
- Pericardial effusion
- Constrictive pericarditis
- Obstruction of caudal vena cava by clot or mass
- Tricuspid dysplasia
- Tricuspid regurgitation caused by chronic valvular heart disease

Key Point

Decompensated heart diseases that result in ascites (e.g., pericardial effusion, dilated cardiomyopathies, heartworm disease) may not have an associated murmur. Thus, whenever ascites occurs, right-sided heart failure must be included in the differential diagnosis.

Cyanosis

- Cyanosis is blue-tinged mucous membranes of the gums, tongue, eyes, ears, and other areas and is associated most commonly with right-to-left shunting congenital heart defects. Occasionally it is seen with severe left-sided heart failure or severe respiratory disease. It is rarely seen with abnormal hemoglobin production. Cyanosis is an insensitive way of detecting hypoxemia in dogs and cats because the oxygen saturation must be very low to cause it, and animals have darker mucous membranes, which makes cyanosis harder to detect until it is severe. See Fig. 1.2 for an example of cyanosis.
- Right-to-left shunting cardiac defects such as tetralogy of Fallot result in low oxygen saturation and high deoxygenated hemoglobin levels, which make affected animals cyanotic. These patients also have polycythemia, and the elevated number of red blood cells has increased amounts of reduced hemoglobin, which contributes to the cyanosis. Cyanosis becomes worse with exercise because the peripheral vascular resistance will decrease, although the pulmonary vascular pressure is unchanged so more deoxygenated venous blood will go systemically.

Key Point

Cyanosis is a late finding in severe cardiac disease, except in right-to-left shunting congenital heart defects, so it is an insensitive indicator of membrane oxygenation and cardiac function.

Weight Loss

- Weight loss occurs in dogs with chronic, severe heart failure, particularly right-sided heart failure.

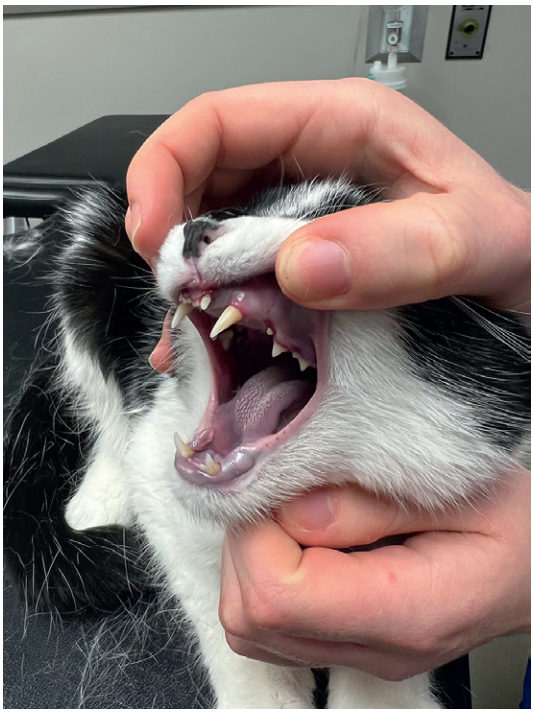


Fig. 1.2 Cat with cyanotic mucous membranes due to congenital heart disease (tetralogy of Fallot or pulmonary atresia). (Courtesy of Dr. Eva Larouche-Lebel.)

Weight loss in cats is usually associated with hyperthyroidism, chronic kidney disease, or infiltrative bowel disease, although cats with chronic heart failure can also lose weight. Cardiac cachexia is the loss of total body fat and lean body mass, especially skeletal muscle, despite a normal appetite and adequate therapy for the underlying heart disease. The loss of body condition in some dogs with dilated cardiomyopathy can be rapid (Fig. 1.3). See Box 1.6 for a list of the problems that contribute to cardiac cachexia in dogs.

- Weight loss is associated with ascites, malabsorption, congestion of the abdominal organs, lymphangiectasia, increased energy that is used for breathing, and medications that cause anorexia and vomiting. Dogs with ascites may have mild discomfort from the fluid, which makes them reluctant to eat. Also, the ascites and congested liver will compress the stomach so that the animal feels full after eating only a small amount of food. The fluid also restricts gastric emptying. Finally, if an unpalatable diet is fed, the animal is even more reluctant to eat and does not consume enough calories to keep from losing weight. Malabsorption caused by congestion of the intestines resulting from ascites also contributes to weight loss.
- Right-sided heart failure causes congestion of the pancreas, which may decrease its function of secreting enzymes for digestion, resulting in maldigestion.

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Fig. 1.3 Great Dane with cardiac cachexia caused by dilated cardiomyopathy.

Box 1.6 Problems That Contribute to Cardiac Cachexia in Dogs

Chronic heart failure
Ascites
Cardiac medications causing anorexia and vomiting
Electrolyte imbalance causing anorexia
Increased energy use by the body
Increased tumor necrosis factor
Malabsorption
Maldigestion
Protein-losing enteropathy

Also right-sided heart failure causes systemic venous and lymphatic hypertension, resulting in a secondary intestinal lymphangiectasia that produces a protein-losing enteropathy.

- Animals with right-sided heart failure and ascites have an increased effort of breathing and increased myocardial oxygen consumption that result from the decreased cardiac output. These two problems result in an increased use of energy and therefore calories by the heart and lungs. The activation of the sympathetic nervous system, which also results from the decreased cardiac output, causes increased energy use by the rest of the body.
- Some cardiac medications, such as digoxin, mexiletine, and ACE inhibitors, can cause anorexia or vomiting. The anorexia and vomiting also contribute to weight loss.
- Worsening renal function caused by diuretic medications may also be a contributing factor. Digoxin also has a direct effect on the small bowel, where it inhibits sugar and amino acid transport.
- Electrolytes, especially sodium and potassium, are adversely affected by diuretics, ACE inhibitors, and digoxin. When potassium levels are abnormal, they contribute to anorexia and therefore weight loss.
- Dogs with chronic congestive heart failure have increased tumor necrosis factor, which inhibits the activity of lipoprotein lipase (hydrolyzes chylomicrons) and therefore interferes with the conversion of triglycerides to free fatty acids. This also will contribute to the animal's weight loss.

Key Point

Weight loss and cardiac cachexia are caused by multiple factors. It is important to make sure that an animal with ascites is being treated appropriately for heart failure and that the animal's digoxin levels, electrolytes, and renal function are being monitored. It is also important to calculate the animal's caloric requirement and make sure it is eating enough to meet its requirements (at least 60 kcal/kg daily). Special high-caloric diets and multiple small meals may be needed to ensure adequate caloric intake. The use of Puppy Chow, which is high in calories with a lower volume of food, has helped some patients maintain or gain weight. Appetite stimulants have been tried with varying success.

Paresis

- Cats with acute posterior paresis (Fig. 1.4) or paresis of one front leg (Fig. 1.5) often have thromboembolism resulting from cardiomyopathy. The thrombi tend to form in the dilated left atrium or left ventricle, and pieces break off and lodge in the distal aorta or another artery. When the thrombus lodges in the aortic bifurcation, the cat will exhibit severe pain in the first few hours after the embolism, and the distal limbs will be cold and may be slightly swollen. The pads on the rear feet will be cyanotic (Fig. 1.6). The cat's pulses cannot be detected, and the nails on the affected legs do not bleed when cut short.
- Acute posterior paresis is rare in dogs but it has been associated with emboli from severe vegetative

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Fig. 1.4 Cat with a saddle thrombus resulting from a left atrial thrombus caused by hypertrophic cardiomyopathy.



Fig. 1.5 Cat with a front leg thrombus resulting from a left atrial thrombus caused by hypertrophic cardiomyopathy.

endocarditis of the aortic or mitral valve. Shifting leg lameness in dogs has also been associated with bacterial endocarditis. Hindlimb weakness or collapse may be associated with reverse (right-to-left) PDA, which delivers poorly oxygenated blood to the caudal half of the body.

Key Point

Paresis in the rear limbs of the cat is usually caused by emboli that form in the left atrium, resulting from left atrial enlargement that occurs with cardiomyopathies. Shifting leg lameness or posterior paresis in the dog can be caused by emboli from vegetations on either the mitral or aortic valve. Hindlimb weakness or collapse in the dog can be caused by a reverse PDA.



Fig. 1.6 Comparison of the front and rear leg footpads in a cat with a saddle thrombus. Note the purple color of the rear leg caused by the complete occlusion of blood flow to the rear limbs secondary to the saddle thrombus and constriction of the collateral circulation.

Box 1.7 Reasons That an Animal Will Not Lie Down

Diaphragmatic hernia
Pneumothorax
Severe pericardial effusion
Severe pleural effusion
Severe pulmonary edema
Severe respiratory disease

PHYSICAL EXAMINATION

OBSERVATION

- The animal's attitude and behavior can give clues to the severity and kind of problems that the animal is having. Note whether the animal is depressed or alert, listless, or active. An animal that refuses to lie down may have severe pulmonary edema, pleural effusion, pericardial effusion, pneumothorax, diaphragmatic hernia, or respiratory disease (Box 1.7). An animal that stands with elbows abducted and head extended with open-mouth breathing and flared nostrils has severe respiratory distress and needs immediate therapy.
- The rate, rhythm, and effort of respirations can help determine the underlying problem. Tachypnea and panting are usually caused by excitement; however, expiratory dyspnea usually indicates lower airway disease, inspiratory dyspnea usually indicates upper airway disease, and mixed dyspnea can be caused by severe pulmonary edema or severe pneumonia. See Box 1.1 for the causes of dyspnea in animals.
- Noting the nature and type of coughing is helpful if it occurs during the course of the physical examination. An animal that has a history of coughing and is not dyspneic is unlikely to have cardiac failure as the cause of coughing.

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- The presence of dependent ventral edema can give the clinician an idea of the source of the animal's problem. If edema is present in the neck, head, and forelimbs only, then it usually indicates an obstruction of the cranial vena cava or a mediastinal mass. If edema is present in the entire body, then pleural effusion with or without ascites is usually present. Other causes of edema (e.g., hypoproteinemia) should also be considered.
- An elevated temperature may occur with an infectious disease or subacute bacterial endocarditis. A subnormal temperature can occur with low cardiac output caused by severe heart failure in dogs or cats or with a saddle thrombus in cats.

Key Point

By observing an animal, a clinician can quickly determine the presence and severity of a respiratory or cardiac problem. Animals with severe dyspnea should be handled gently to avoid stress and should be treated immediately.

HEAD

- The eyes should be examined for changes that could indicate systemic diseases. Hypertension in cats will cause decreased pupillary responses due to retinal detachment or hemorrhage. A fundic examination may reveal papilledema (swelling of the optic disc) along with hemorrhage or retinal detachment. Retinal hemorrhages also can occur with polycythemia and bacterial endocarditis.
- Central retinal degeneration occurs in about one-third of cats with dilated cardiomyopathy caused by taurine deficiency. The areas of degeneration are horizontal, linear, and hyperreflective.
- The ears have no significant changes associated with the cardiovascular system. However, cyanosis can sometimes be recognized by an evaluation of the color of the pinna.
- Examine the nose for signs of disease and patency. Check each patient for any asymmetry and swellings, especially in the nasal and throat areas because these swellings could be obstructing airways and causing respiratory distress.
- The mucous membrane color and perfusion in the mouth should be noted. A perfusion time of greater than 2 seconds suggests decreased cardiac output; however, most animals with congestive heart failure have normal mucous membrane color until their heart failure is severe. Because of this, mucous membrane color and perfusion are insensitive ways to evaluate adequate circulation.
- Cyanosis of the mucous membranes is caused by hypoventilation or poor diffusion across the alveoli produced by multiple different factors (see the previous section).



Fig. 1.7 Hyperemic mucous membranes in a dog with polycythemia caused by tetralogy of Fallot.

- Hyperemic mucous membranes (dark red to muddy) may indicate an increased packed cell volume (polycythemia), which can result from a chronic right-to-left vascular shunt (Fig. 1.7).
- Pale mucous membranes indicate anemia or poor perfusion. The mucous membranes of the mouth should be compared with the posterior membranes (e.g., vagina, prepuce) because differential cyanosis can occur in a right-to-left shunting PDA.

Key Point

Mucous membrane color and perfusion are insensitive signs of cardiac function, so when they are abnormal, the animal's cardiac or respiratory problem is severe.

NECK

- The jugular pulse should be evaluated while the animal is standing with its head in a normal position. Any pulse going over one-third of the way up the neck is abnormal and can be due to any of several factors. See Box 1.8 for the causes of abnormal jugular pulses. Arterial pulses can mimic a jugular pulse; however, when light pressure is applied to the area of the jugular pulse, the arterial pulse will continue, whereas the jugular pulse will stop.
- Abnormal jugular pulses occur in right-sided heart failure caused by tricuspid regurgitation or dilated cardiomyopathy, when the right ventricle contracts and the blood flows back into the right atrium and up the jugular veins because of the insufficient tricuspid valve. Abnormal jugular pulses also occur with pulmonic stenosis and pulmonary hypertension when the right atrium contracts against a hypertrophied, noncompliant right ventricle so that only some of the blood enters the right ventricle and the rest goes back up the jugular veins.
- With heartworm disease, abnormal jugular pulses occur because there is both a stiff, hypertrophied

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Box 1.8 Causes of Abnormal Jugular Pulses

- Arrhythmias
- Heartworm disease
- Pulmonary hypertension
- Pulmonic stenosis
- Right-sided heart failure (e.g., tricuspid regurgitation, dilated cardiomyopathy)

right ventricle and tricuspid regurgitation, which both contribute to the jugular pulses.

- Arrhythmias such as second- or third-degree heart block or premature beats cause abnormal jugular pulses because the sequence of atrial and ventricular activation is disrupted. The right atrium contracts against a closed tricuspid valve, sending blood back up the jugular veins.
- The entire jugular vein can be distended, indicating increased systemic venous pressure caused by right-sided heart failure, pericardial disease, or obstruction of the cranial vena cava (e.g., heart base tumor). About 70% of dogs with right-sided heart failure have distended jugular veins, but they are rare in cats. Cats with left-sided heart failure may have distended jugular veins only when lying down, and the jugular veins return to normal when they sit or stand. See Box 1.9 for causes of jugular distension.
- Mediastinal masses such as lymphosarcoma can compress the cranial vena cava, causing a distended jugular vein; however, they also usually cause pleural effusion and head and neck edema.
- The hepatojugular reflex is a distension of the jugular veins that occurs when the abdomen is compressed for 10 to 30 seconds. It is caused by the increased return of blood. Because the right side of the heart is not normal and cannot handle the increased venous return, the blood from the cranial vena cava cannot enter the heart and the jugular veins become distended. This reflex is present with both right- and left-sided heart failure and indicates increased blood volume in the peripheral venous system caused by an inability of the heart to circulate the blood properly.
- A central venous pressure can be obtained in animals with distended jugular veins. It is most useful in the diagnosis of constrictive pericardial disease, where less invasive tests have not confirmed the diagnosis.

Key Point

Jugular vein distention can be present with both right- and left-sided heart failure but may be present in only 70% of these cases. Abdominal ultrasound is a sensitive, noninvasive method of determining increased venous pressure by detecting distended hepatic veins; however, the ultrasound confirms only the presence of increased venous pressure and not the cause.

Box 1.9 Causes of Distended Jugular Veins

- Right-sided heart failure (e.g., tricuspid regurgitation, dilated cardiomyopathy)
- Pericardial diseases
- Heart base tumor
- Mediastinal mass

TRACHEAL PALPATION

- The trachea should be palpated for abnormalities such as collapsing, masses, or increased sensitivity. This part of the examination is best postponed until after auscultation of the thorax because a cough may be elicited that makes auscultation difficult.
- Lymph nodes should be palpated to see if they are enlarged. The thyroids may be enlarged in cats with hyperthyroidism (thyroid slip). An old dog with a mass near the larynx and an associated thrill usually has thyroid carcinoma with an arteriovenous fistula causing the thrill (a murmur that can be felt as well as heard).

Key Point

Palpation of the neck can reveal primary or secondary problems that affect the heart and can mimic heart disease (collapsing trachea).

THORACIC PALPATION

- The apex beat (sometimes called the point of maximum intensity [PMI]) is where the cardiac impulse is felt strongest on the chest wall. It should be on the left side of the thorax between the fourth and sixth intercostal spaces. Shifting of the PMI is caused by cardiac enlargement, masses displacing the heart, collapsed lung lobes that allow the heart to shift, diaphragmatic hernias, pleural effusions associated with collapsed lung lobes or fibrin, or right lateral recumbency, which causes the heart to fall to the right. Pectus excavatum, a deformity of the sternum, can also shift the heart to the right (Box 1.10).
- A decreased intensity of the heartbeat or heart sounds may be caused by obesity, pleural effusion, pericardial effusion, thoracic masses, severe pneumothorax, severe emphysema, diaphragmatic hernias, decreased left ventricular contractility with decreased cardiac output, or arrhythmias, which decrease cardiac filling and therefore cardiac output (Box 1.11).
- There can be an increased intensity of the PMI or heart sounds in young, thin animals or in animals with increased heart rates or with hyperdynamic states such as anemia, hyperthyroidism, or fever. Sometimes the heart appears to be beating on the chest wall. The heart is not actually hitting the chest

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Box 1.10 Causes of Shifting of the Palpable Apex Beat or Point of Maximum Intensity

Cardiac enlargement
Collapsed lung lobes on the right
Diaphragmatic hernias
Pectus excavatum
Lying down in right lateral recumbency
Masses displacing the heart
Pleural effusions

Box 1.11 Causes of Changes in Heart Sounds

Decreased intensity caused by:
- Arrhythmias
- Decreased left ventricular cardiac output
- Diaphragmatic hernias
- Obesity
- Pleural or pericardial effusions
- Severe emphysema
- Severe pneumothorax
- Thoracic masses

Increased intensity caused by:
- Hyperdynamic states (e.g., anemia, hyperthyroidism, fever)
- Increased heart rates
- Young, thin animals

wall; the appearance is caused by the increased wall tension of the thoracic wall for an indeterminate reason. It does not mean that the heart is contracting normally or better than normal.

- Loud cardiac murmurs can be palpated as thrills, which are caused by vibrations caused by blood flow. A thrill is always located where a murmur is loudest (murmur PMI).

Key Point

It is important to locate both the PMI of the heartbeat and the presence of thrills caused by heart murmurs on the thoracic wall.

ABDOMINAL PALPATION

- Abdominal palpation is done to check for ascites, the presence of fluid in the abdomen. Ascites can be caused by right-sided heart failure as well as many other causes. The presence of ascites can be difficult to ascertain in obese animals. The clinician should put one hand on one side of the abdomen and the other hand on the other side, and then tap the abdomen. If a fluid wave is felt by the hand on the opposite side of the abdomen, then ascites is present. In addition to ascites, check for signs of hepatomegaly and splenomegaly.
- The presence of ascites, splenomegaly, and hepatomegaly usually indicates right-sided heart failure caused by dilated cardiomyopathy, tricuspid regurgitation, heartworm disease, pericardial disease, or congenital heart disease. Ascites caused by right-sided heart failure is usually a transudate or protein-rich (modified) transudate on cytologic examination. Another possible cause of ascites is an obstruction of the posterior vena cava.
- Palpate the abdomen for any masses. Also palpate the kidneys because chronic renal failure can lead to systemic hypertension that can affect the heart.

Key Point

Ascites can be caused by right-sided heart failure or other heart problems, such as pericardial effusion; however, it can also be caused by other noncardiac problems. Further tests are indicated to determine the cause of ascites.

SKIN

- Palpate for evidence of edema caused by right-sided heart failure or venous obstruction.

FEMORAL PULSES

- Both femoral pulses should be felt with the dog or cat standing, and they should be compared with each other because one could be obstructed.
- It is difficult to palpate the femoral pulse in a normal cat; therefore the absence of palpable femoral pulses in a cat should not be interpreted as definite arterial obstruction.
- Partial or complete occlusion of the pulses so that they cannot be felt is usually caused by thromboembolism. In dogs, this can occur with bacterial endocarditis of the mitral or aortic valves, hyperadrenocorticism, neoplasia, and protein-losing nephropathy. Cavalier King Charles Spaniels with chronic valvular heart disease of the mitral valve sometimes lack a pulse for unknown reasons. In cats, absent pulses are associated with cardiomyopathy but can also occasionally be caused by bacterial endocarditis or extracardiac disease.
- The femoral pulse rate should be taken. Normal rates in dogs are 70 to 180 beats/min. Puppies can have a normal rate of 220 beats/min. Normal rates in cats are 160 to 230 beats/min.
- The rhythm of the pulses should be noted. There should be a pulse for every heartbeat. Pulse deficits usually indicate incomplete ventricular filling, as seen with arrhythmias. If an arrhythmia is present (e.g., atrial fibrillation), the pulse rate will be less than the heart rate due to pulse deficits.
- Arterial pulse pressure (femoral pulse quality) is the difference between the arterial systolic pressure and the diastolic pressure. Pulse quality can normally vary depending on the animal's conformation, age, hydration, heart rate, cardiac function, and level of excitement or activity.

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Table 1.3 Types of Pulses and Their Associated Causes

PULSE	CAUSE
Absent pulses	Thromboembolism
Abrupt pulses	Mitral regurgitation Ventricular septal defects
Erratic pulses	Atrial fibrillation
Hypokinetic pulses	Dilated cardiomyopathy Heart failure Hypotension Hypovolemia Subaortic stenosis
Hyperkinetic pulses	Aortic regurgitation Fear Fever Patent ductus arteriosus Severe anemia Severe bradycardia Thyrotoxicosis
Pulse deficits	Arrhythmias
Pulsus alternans	Severe dilated cardiomyopathy
Pulsus bigeminus	Arrhythmias (atrial or ventricular bigeminy)
Pulsus paradoxus	Cardiac tamponade

- The intensity of the pulse should be palpated. Normal pulses are strong and have a rapid rate of rise and fall. Table 1.3 lists the pulse quality and the cause for each type of pulse.
- Hypokinetic (weak) pulses are caused by decreased cardiac output (e.g., dilated cardiomyopathy, congestive heart failure, hypovolemia), decreased peripheral vascular resistance, increased arterial compliance, or slower rate of rise caused by delayed emptying of the left ventricle (e.g., subaortic stenosis, hypertrophic obstructive cardiomyopathy). Dogs will have normal pulses until the stroke volume is markedly decreased with severe congestive heart failure, so pulses are an insensitive indicator of cardiac output.
- Hyperkinetic (strong) pulses rise and fall quickly and are caused by large left ventricular stroke volumes with rapid diastolic runoffs (e.g., PDA, aortic regurgitation). They are called "B-B shot" or "water-hammer" pulses. Fear, fever, severe bradycardia, thyrotoxicosis, and anemia can also produce this type of pulse.
- The pulses can be abrupt or jerky with chronic mitral valve disease and ventricular septal defects as a greater volume of blood is ejected in early systole.
- Pulsus alternans occurs when the pulse is alternately weak and then strong in patients with normal sinus rhythm. It is frequently associated with severe myocardial failure (e.g., dilated cardiomyopathy).
- Pulsus bigeminus occurs when weak pulses alternate with strong pulses. This is associated with arrhythmias such as ventricular bigeminy in which a normal sinus beat alternates with a ventricular premature beat. The weak pulses occur because the premature beat causes the ventricles to contract before they are adequately filled so that a smaller-than-normal volume of blood is ejected by the left ventricle. The difference between normal and abnormal pulses may be accentuated because the ventricles have more time to fill on the normal beats, and so the normal pulses feel even stronger.
- Pulsus paradoxus is an alteration of the pulse strength during respiration caused by changes in ventricular filling. There is an increase in pulse strength on expiration and a decrease in strength on inspiration in normal animals; however, this change is exaggerated and easier to feel when cardiac tamponade is present.
- Pulses feel erratic when an animal has atrial fibrillation.

Key Point

The quality of an animal's pulse is not a good indication of the severity of its cardiac problem because only very advanced heart disease will cause weak pulses; however, pulse deficits are good indicators of the presence of an arrhythmia.

PERCUSSION

- Percussion can be used to determine the presence of masses or fluid lines, especially in the thorax. It will elicit a hollow sound (hyperresonance) over the lungs and a dull sound (hyporesonant) over solid structures.
- If an area, especially a dorsal area, in the thorax sounds hyperresonant, then pneumothorax may be present.
- If an area, especially a ventral area, sounds hyporesonant, then pleural effusion may be present.

Key Point

Percussion can be a rapid way of determining the presence or absence of pleural effusion.

STETHOSCOPE

- The main components of the stethoscope are the bell, diaphragm, tubing, and ear pieces. The bell transmits low-frequency (20–300 cycles per second [cps]) sounds when light pressure is used and high-frequency (300–1000 cps) sounds when firm pressure is applied to the thorax. It is best for hearing third and fourth heart sounds. The diaphragm attenuates low frequencies and selectively transmits high frequencies. It is best for hearing the first and second heart sounds.

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- Most stethoscopes combine the bell and diaphragm into a dual-sided, combination-style chest piece. Some stethoscopes have the bell and diaphragm as one piece. With these stethoscopes, simple fingertip pressure allows one to switch from low- to high-frequency sounds. Unlike a traditional two-sided stethoscope, there is no interruption in sound, resulting in added convenience and efficiency in auscultation. For dogs and cats that weigh less than 15 lb, a pediatric stethoscope that has a smaller headpiece should be used.
- A practical tubing length on a stethoscope is approximately 14 to 18 inches. If the tubing is too long, it will attenuate all the heart and lung sounds.
- Ear pieces should angle forward to conform to the anatomy of the ear canals. A stethoscope with variable sizes of ear pieces is ideal because each person can find the correct-size earpiece that fits comfortably in the ear canal and shuts out extraneous sounds.
- In addition to electronic amplification of heart sounds and murmurs, electronic stethoscopes allow the user to record and play back sounds at either normal or half speed. This feature is useful for judging and timing the shape or quality of murmurs in patients with tachycardia and for judging the timing of transient heart sounds such as clicks or gallops. Some models also provide the ability to record graphic representations of sounds in a digital file format (i.e., a phonocardiogram) that can be stored on a computer, possibly even becoming part of the patient's medical record. One model of electronic stethoscope is the 3M Littmann Model 3000 (Fig. 1.8). It features useful ambient noise reduction circuitry that appears to overcome most, if not all, of the problems of background noise amplification that plagued previous models.



Fig. 1.8 3M Littmann Electronic Stethoscope Model 3200. (Courtesy 3M, St. Paul, Minnesota.)

Key Point

No one stethoscope works well for everyone. Ideally, a clinician should try various stethoscopes to find the one that works best and also find the earpiece size that best fits their ear canals.

AUSCULTATION

- Auscultation of the heart is the most helpful part of the cardiac examination; it should be done carefully and systematically. The animal should be standing so that the heart is in its normal position. This avoids the problem of positional murmurs caused by the heart rubbing against the chest wall when an animal is lying down.
- Common artifacts that can be heard include respiratory clicks and murmurs, rumbles caused by shivering and twitching, and movement sounds such as crackling caused by rubbing of hair. Extraneous sounds will occur if auscultation is not performed in a quiet area.
- Be sure to auscultate the heart and lungs separately. Also auscultate the entire thorax on both sides. Heart rate and rhythm should be identified. The effects of inspiration and expiration on heart rate, rhythm, and heart sounds should be noted. Also note the presence or absence of heart sounds.
- The areas of auscultation of the heart are shown in Fig. 1.9. Table 1.4 lists the areas of auscultation.
- The pulmonic valve area is on the left side at the base of the heart. In the dog, it is between the second and fourth intercostal spaces, just above the sternum. In the cat, it is located at the second to third intercostal space, one-third to one-half of the way up the thorax from the sternum.
- The aortic valve area is on the left side at the base of the heart. In the dog, it is at the fourth intercostal space, just above the costochondral junction. In cats, it is at the second to third intercostal space, just dorsal to the pulmonic area.
- In cats and small dogs, it may be impossible to distinguish the pulmonic and aortic areas, so these two areas are referred to as the left heart base.
- The mitral valve area is on the left side. In the dog, it is at the fifth intercostal space at the costochondral junction. In the cat, it is at the fifth to sixth intercostal space, one-fourth of the way up the thorax from the sternum. In cats and small dogs, this area may also be referred to as the left heart apex.
- The tricuspid valve area is on the right side. In the dog, it is at the third to fifth intercostal space near the costochondral junction. In the cat, it is at the fourth or fifth intercostal space, at a level opposite

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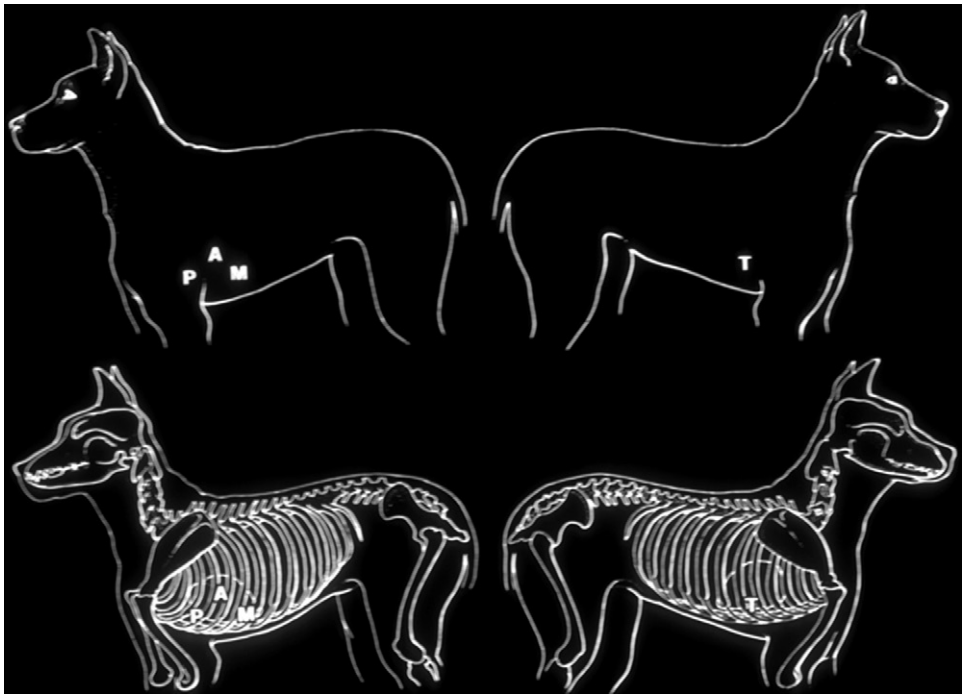


Fig. 1.9 Areas of auscultation in the dog. *M* is the mitral valve area, *A* is the aortic valve area, *P* is the pulmonic valve area, and *T* is the tricuspid valve area. (From Gompf RE: The clinical approach to heart disease: history and physical examination. In Fox PR, editor: *Canine and feline cardiology*, New York, 1988, Churchill Livingstone.)

Table 1.4 Areas of Auscultation in the Dog and Cat

STRUCTURE	LOCATION
Mitral valve (left apex)	Dog—left side, fifth intercostal space at costochondral junction Cat—left side, fifth to sixth intercostal space near sternum
Aortic valve (left base)	Dog—left side, fourth intercostal space just above costochondral junction Cat—left side, second to third intercostal space, just dorsal to pulmonic area
Pulmonic valve (left base)	Dog—left side, between second and fourth intercostal space, just above sternum Cat—left side, second to third intercostal space, one-third of the way up from sternum
Tricuspid valve (right apex)	Dog—right side, third to fifth intercostal space near costochondral junction Cat—right side, fourth to fifth intercostal space near sternum

the mitral area. It may also be referred to as the right heart apex.

- Note the areas in which murmurs are loudest (PMI) and where they radiate. Often, murmurs have an area where they are heard the loudest (PMI) and other areas where they are softer (referred to as areas of radiation).
- Alternative areas of auscultation include the thoracic inlets for radiation of the murmur of subaortic stenosis and the left axillary area for murmurs of PDA.

Key Point

Auscultate the lungs and heart separately to avoid missing or confusing any abnormal sounds. Auscultate all valve areas in all animals. Congenital heart defects have been missed when clinicians listen only to the mitral valve area in a young animal.

NORMAL HEART SOUNDS

- Heart sounds are caused by the abrupt acceleration or deceleration of blood and the vibrations of the heart and vessels.
- The first heart sound (S_1) (Fig. 1.10) is caused by passive closure of the mitral (left atrioventricular) and tricuspid (right atrioventricular) valves, resulting in the sudden acceleration and deceleration of blood. It has four parts that can be seen on a phonocardiogram.
- S_1 is longer, louder, duller, and lower pitched than the second heart sound (S_2). It is loudest over the mitral and tricuspid areas and in young, thin animals and those with high sympathetic tone (e.g., fear), tachycardia, systemic hypertension, anemia, or mitral regurgitation. The intensity of S_1 decreases because of obesity, pleural or pericardial effusion, thoracic masses, diaphragmatic hernias,

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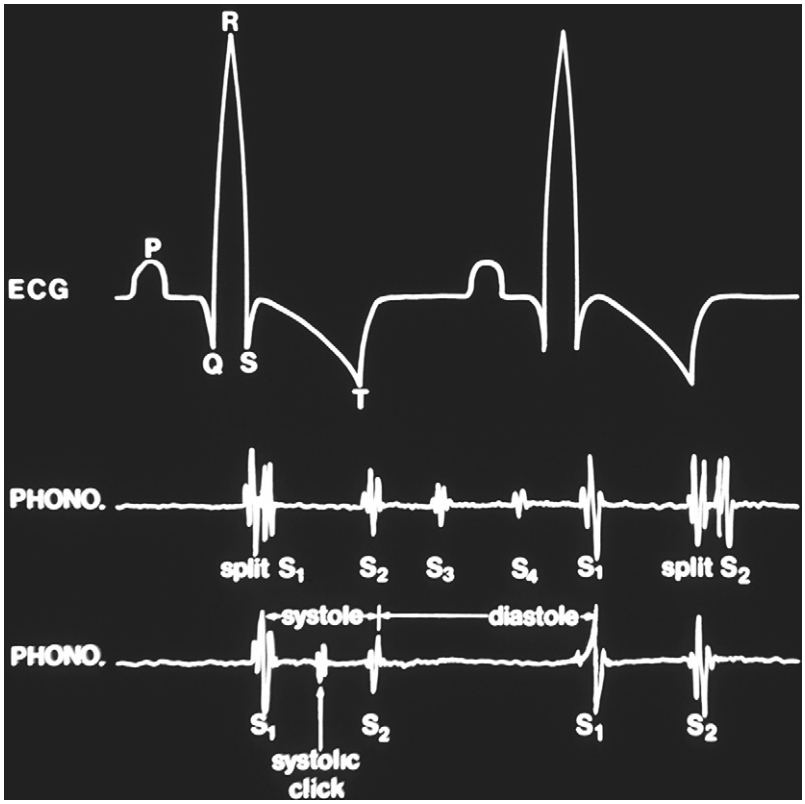


Fig. 1.10 Heart sounds and their relationship to the ECG. The first heart sound is S_1. The second heart sound is S_2. The third heart sound is S_3, and the fourth is S_4. (From Gompf RE: The clinical approach to heart disease: history and physical examination. In Fox PR, editor: *Canine and feline cardiology*, New York, 1988, Churchill Livingstone.)

bradycardia, emphysema, shock, and insufficient filling of the ventricles.

- S_1 varies in intensity with arrhythmias.
- Splitting of S_1 (see Fig. 1.10) is caused by asynchronous closure of the mitral and tricuspid valves. It can be split normally in large breeds of dogs or abnormally with right bundle branch block, atrial or ventricular premature beats, cardiac pacing, or stenosis of the mitral or tricuspid valve. The arterial pulse occurs just after S_1, and the precordial impact occurs simultaneously with S_1.
- Box 1.12 lists the causes of changes in S_1.
- S_2 (see Fig. 1.10) is produced by passive closure of the aortic and pulmonic valves. It is short, high pitched, and sharp. It is loudest over the aortic and pulmonic areas.
- A split S_2 (see Fig. 1.10) is caused by closure of the pulmonic valve after the aortic valve. This occurs in pulmonary hypertension (e.g., severe heartworm disease or right-to-left PDA), right bundle branch block, ventricular premature beats originating in the left ventricle, atrial septal defect, pulmonic stenosis, and mitral stenosis.
- Paradoxical splitting of S_2 is caused by delayed closure of the aortic valve. This results from left bundle branch block, premature beats originating from the right ventricle, subaortic stenosis, severe systemic hypertension, and left ventricular failure.
- S_2 may be absent in arrhythmias where there is incomplete filling of the ventricles and insufficient pressure to open the semilunar valves.
- Box 1.13 lists the causes of changes in S_2.

Key Point

It is critical to identify the first and second heart sounds in all patients and use auscultation as an effective tool in the diagnosis of heart disease. If extraneous sounds are present, then the clinician may need to move to another area of the thorax to identify these sounds properly.

ABNORMAL HEART SOUNDS

- The third heart sound (S_3) (see Fig. 1.10) is caused by rapid ventricular filling and is not heard in normal dogs or cats. It is lower pitched than the S_2 and is heard best in the mitral valve area. It occurs during diastole after S_2.
- An auscultable S_3 in dogs is indicative of dilated ventricles, which most commonly occur with dilated cardiomyopathy, decompensated mitral or tricuspid regurgitation, large ventricular or atrial septal defects, and large PDA. In cats it is associated with dilated cardiomyopathy, severe anemia, and severe hyperthyroidism.

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Box 1.12 Causes of Changes in the First Heart Sound (S_1)

- Loud S_1
  - Anemia
  - Excitement
  - Exercise
  - Fear
  - Fever
  - Hyperthyroidism
  - Mitral regurgitation
  - Positive inotropic agents
  - Pregnancy
  - Systemic hypertension
  - Tachycardia
  - Thin animals
- Soft S_1
  - Bradycardias
  - Decreased cardiac output
  - Diaphragmatic hernias
  - Emphysema
  - Hypothyroidism
  - Left bundle branch block
  - Negative inotropic agents
  - Obesity
  - Pericardial effusion
  - Pleural effusion
  - Severe aortic or mitral regurgitation
  - Severe heart failure
  - Shock
  - Thoracic masses
- Variable S_1
  - Arrhythmias
- Split S_1
  - Atrial or ventricular premature beats
  - Cardiac pacing
  - Right bundle branch block
  - Stenosis of mitral or tricuspid valve

Box 1.13 Causes of Changes in the Second Heart Sound (S_2)

- Loud S_2
  - Atrial septal defect
  - Mild valvular pulmonic stenosis
  - Patent ductus arteriosus
  - Pulmonary embolism
  - Pulmonary hypertension
  - Systemic hypertension
  - Valvular aortic stenosis
  - Ventricular septal defect
- Soft S_2
  - Dilated cardiomyopathy
  - Hypothyroidism
  - Marked aortic regurgitation
  - Shock
  - Significant pulmonic stenosis
  - Valvular aortic stenosis
- Split S_2
  - Atrial septal defect
  - Heartworms
  - Mitral stenosis
  - Pulmonary hypertension—moderate to severe
  - Pulmonic stenosis
  - Right bundle branch block
  - Right-to-left patent ductus arteriosus
  - Ventricular premature beat from left ventricle
- Paradoxical split S_2
  - Left bundle branch block
  - Left ventricular failure
  - Severe systemic hypertension
  - Significant aortic regurgitation
  - Subaortic stenosis
  - Ventricular premature beat from right ventricle
- Absent S_2
  - Arrhythmias

- Table 1.5 lists the causes of abnormal heart sounds.
- An auscultable fourth heart sound (S_4) (see Fig. 1.10) is caused by atrial contraction into an already overdistended ventricle or into a stiff ventricle in dogs and cats. It is heard best over the aortic or pulmonic areas, but sometimes it can be heard over the mitral valve area. It occurs in late diastole, after atrial contraction, and just before S_1.
- S_4 is present in dogs or cats when the atrium is dilated in response to ventricular diastolic dysfunction, such as hypertrophic cardiomyopathy or third-degree heart block. It may also be heard in dogs with ruptured chordae tendineae.
- Because the rhythm sounds like a cantering horse when an S_3, an S_4, or a combination (summation) of the two occur (in addition to the normal heart sounds [S_1 and S_2]), auscultable S_3 and S_4 sounds are called "gallop sounds." These sounds have a low frequency and can be difficult to hear. A gallop can be an early sign of heart failure, preceding clinical signs.
- Systolic clicks are short, mid- to high-frequency clicking noises that occur in systole between S_1 and S_2 (see Fig. 1.10). They are usually loudest over the mitral and tricuspid areas. A systolic click may come and go and may change its position in systole (gets closer to or further away from S_2). The intensity can also change.
- A systolic click can be hard to differentiate from a gallop, especially if the animal's heart rate is fast.
- The precise cause of the click is unknown, but it may be caused by the mitral valve buckling into the left atrium (mitral valve prolapse) in dogs with early mitral regurgitation (many of these animals have a mitral regurgitation murmur later in life). This is a benign finding and not usually associated with heart failure.
- Ejection sounds are high-frequency sounds generated in early systole caused by hypertension, dilation of the great vessels, or opening of abnormal semilunar valves such as in valvular pulmonic stenosis. These sounds are easily missed and misinterpreted as early systolic murmurs.

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Table 1.5 Causes of Abnormal Heart Sounds

SOUND	CAUSE
Gallop sounds	
S_3	Decompensated mitral or tricuspid regurgitation
	Dilated cardiomyopathy
	Large atrial septal defect
	Large patent ductus arteriosus
	Large ventricular septal defect
	Severe anemia in cats
	Severe hyperthyroidism in cats
	Significant aortic regurgitation
S_4	Anemia
	Hypertrophic cardiomyopathy
	Dogs with ruptured chordae tendineae
	Pulmonary hypertension
	Systemic hypertension
	Third-degree heart block
	Thyrotoxicosis
Systolic click	Mitral valve prolapse
Ejection sounds	Anemia
	Atrial septal defect
	Dilation of great vessels
	Exercise
	Heartworms
	Hyperthyroidism
	Pulmonary embolism
	Pulmonary hypertension
	Systemic hypertension
	Valvular aortic stenosis
	Valvular pulmonic stenosis

Key Point

The presence of a gallop is an indication of cardiac disease in small animals.

ARRHYTHMIAS HEARD ON AUSCULTATION

- Sinus arrhythmia has a cyclical pattern. The heart rate will increase during inspiration and decrease during expiration owing to changes in vagal tone. The intensity of the pulse and heart sounds may vary. It is normal in dogs, but in cats it is usually associated with heart disease or systemic disease. Sinus arrhythmia occurs at normal heart rates in dogs and cats and tends to disappear as the heart rate increases.
- Arrhythmias that increase the heart rate (tachycardias) include both atrial and ventricular arrhythmias. Monomorphic ventricular tachycardia and atrial tachycardias are usually rapid and regular. All the heart sounds are of uniform intensity. The tachycardia may be intermittent.
- Animals with atrial fibrillation have a rapid, irregular rhythm with heart sounds that vary in intensity. Pulse deficits are present. Atrial fibrillation has been described as an irregularly irregular rhythm and is rarely mistaken for other tachycardias.
- Ventricular tachycardias are usually intermittent and tend to be more regular than atrial fibrillation. However, an animal with multifocal ventricular tachycardia can sound like a dog with atrial fibrillation. Pulse deficits are frequently present with ventricular tachycardias. An electrocardiogram is necessary to distinguish among sinus, atrial, and ventricular tachycardia.
- Atrial and ventricular premature beats generate extra sounds that mimic S_3 and S_4. It is difficult to differentiate between the two types of premature beats, as well as between S_3 and S_4 during physical examination. An electrocardiogram and phonocardiogram may be necessary. Both atrial and ventricular premature beats interrupt the normal rhythm and are usually followed by a pause. Usually only S_1 is heard with a premature beat, and S_2 is absent. However, sometimes S_1 and S_2 can be heard close together. Premature beats can also cause a split S_1 or S_2.
- Arrhythmias that decrease the heart rate are called bradycardias; examples include sinus bradycardia and heart blocks. Sinus bradycardia has a very slow rhythm. The heart sounds may vary. The heart rate in dogs is usually between 50 and 70 beats/min, depending on the size of the dog. The heart rate in cats is less than 120 beats/min.
- Second- and third-degree heart blocks also result in slow heart rates. The heart sounds will vary in intensity. S_4 may be present in a third-degree block. The pulses will be slow and hyperkinetic, but there are no pulse deficits. A jugular pulse is usually present. Extra sounds may be generated by escape beats. An electrocardiogram is necessary to diagnose the type of bradycardia present.
- Unexpected pauses can occur with sinoatrial (or sinus) arrest. Sinus arrest occurs when an impulse does not leave the sinoatrial node. The pause continues until the next normal beat or an escape beat occurs. The heart sound following a pause may be louder than usual because the ventricles have had longer to fill and eject a larger amount of blood. An electrocardiogram is necessary to diagnose sinus arrest.
- Sick sinus syndrome will have long pauses where the heart is not beating at all. If the pause lasts long enough, the dog will faint. Sometimes there are also periods of tachycardia, which gives it the name "bradycardia/tachycardia syndrome." Sick sinus syndrome was first described in older female Miniature Schnauzers but has been found in multiple breeds of dogs.

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Table 1.6 Types of Heart Murmurs and Their Causes

MURMUR	CAUSE
Physiologic	Anemia
	Athletic heart
	Fever
	Hypoproteinemia
	Hypertension
	Hyperthyroidism
	Pregnancy
Innocent	No known cause
Pathologic	Aortic regurgitation
	Aorticopulmonary septal defect
	Arteriovenous fistula
	Atrial septal defect
	Mitral dysplasia
	Mitral regurgitation
	Mitral stenosis
	Patent ductus arteriosus
	Pulmonic regurgitation
	Pulmonic stenosis
	Subaortic stenosis
	Tetralogy of Fallot
	Tricuspid dysplasia
	Tricuspid regurgitation
	Tricuspid stenosis
	Ventricular septal defect

MURMURS

- Murmurs are caused by turbulent blood flow through the heart and vessels. The turbulence can be caused by the disruption of blood flow through holes in the heart (e.g., ventricular or atrial septal defect), a stenotic valve (e.g., aortic, pulmonic, mitral, or tricuspid stenosis), an insufficient valve (e.g., mitral, tricuspid, aortic, or pulmonic regurgitation), or an abnormal arterial venous connection near the heart (e.g., PDA) or can be caused by altered blood viscosity or changes in blood vessel diameter. Table 1.6 lists the types of murmurs and their causes.
- Functional murmurs are divided into physiologic and innocent murmurs. Physiologic murmurs have a known cause, such as increased cardiac output or decreased blood viscosity, and occur with anemia, hypoproteinemia, fever, increased blood pressure, pregnancy, hyperthyroidism, or an athletic heart. These are high-frequency murmurs that occur in the early to midsystolic phase, are loudest over the aortic and pulmonic areas, and rarely radiate to other areas.
- Innocent murmurs have no known cause and are not associated with any cardiac problem. These murmurs are soft systolic murmurs (no louder than grade 3) and usually occur in young animals. They can be located over any valve area but are most frequent over the mitral and aortic areas. Also these murmurs usually disappear by the time of the animal's last vaccinations (5 months of age).
- Pathologic murmurs are caused by underlying heart or vessel disease, such as stenosis of valves, outflow tract, or great vessels; valvular regurgitation; or abnormal intracardiac or extracardiac shunts. Refer to individual defects in the following chapters for a description of the murmurs associated with each defect (see Chapters 6–8 and 12).
- A murmur should be described with the following classification. First, the murmur should be identified as to its timing in the cardiac cycle (e.g., systolic, diastolic, continuous). Also the duration of the murmur (e.g., early systolic, holosystolic, pansystolic) should be noted (Figs. 1.11 and 1.12). Second, the site at which the murmur is loudest (PMI) (e.g., valve area) and where it radiates because of blood flow through the defect (e.g., other valve areas where it can be heard) should be noted. Third, the intensity or loudness of the murmur can be graded on a scale of 1 to 6: grade 1 can only be heard after listening for several minutes and sounds like a prolonged S_1; grade 2 is very soft but can be heard immediately; grade 3 is low to moderate in intensity; grade 4 is very loud, but a thrill cannot be palpated on the thorax; grade 5 is very loud, and a thrill can be palpated on the thorax; grade 6 can be heard without a stethoscope or with the stethoscope slightly off the thoracic wall. In general, the loudness of a murmur may not indicate the severity of the underlying problem. Fourth, the other areas where the murmur is heard should also be identified. These are the areas of radiation of the murmur and can help to identify the cause of the murmur. These four characteristics should be used in the description of all murmurs.
- These additional descriptions can also aid in the diagnosis of the underlying problem. The quality or shape of the murmur is subjective but can be evaluated according to graphic appearance on a phonocardiogram (see Figs. 1.11 and 1.12). Regurgitant murmurs are plateau shaped (e.g., equal loudness throughout). Ejection murmurs are usually decrescendo, crescendo, or diamond shaped. Machinery or continuous murmurs are diamond shaped and peak at S_2, continuing through all of systole and most of diastole. Blowing murmurs are decrescendo murmurs (e.g., decrease in intensity). The pitch or frequency of the murmur can also be described. Some murmurs are high, medium, low pitched, or a mix. Also they can be harsh, blowing, or musical.

OTHER SOUNDS AUSCULTATED IN THE THORAX

- Normal respiratory sounds include referred sounds from the trachea that are commonly heard over the lungs. Vesicular sounds are caused by air moving

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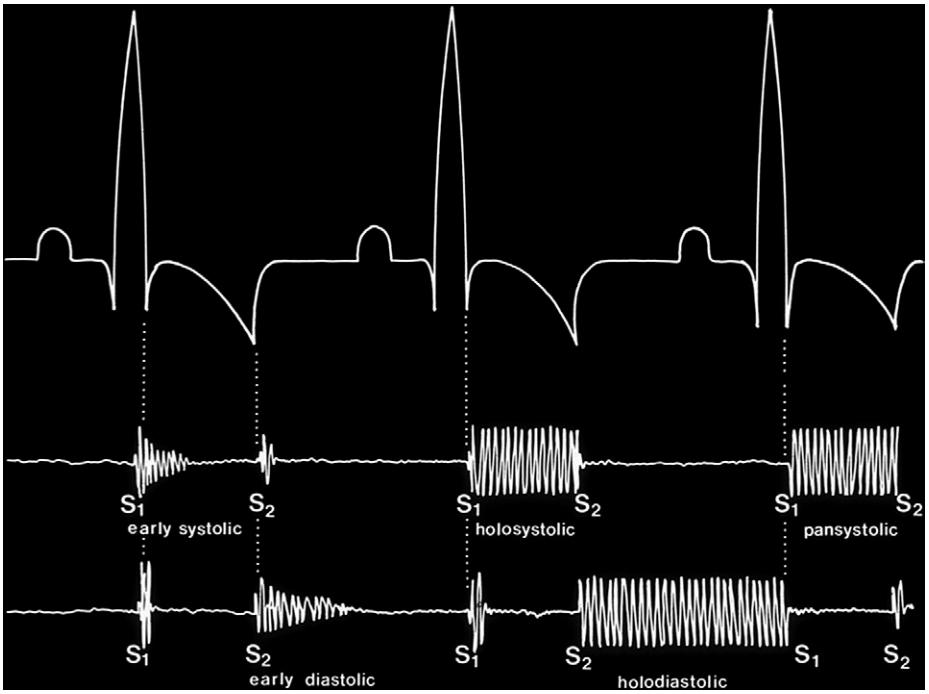


Fig. 1.11 Timing and duration of murmurs. (From Gompf RE: The clinical approach to heart disease: history and physical examination. In Fox PR, editor: *Canine and feline cardiology*, New York, 1988, Churchill Livingstone.)

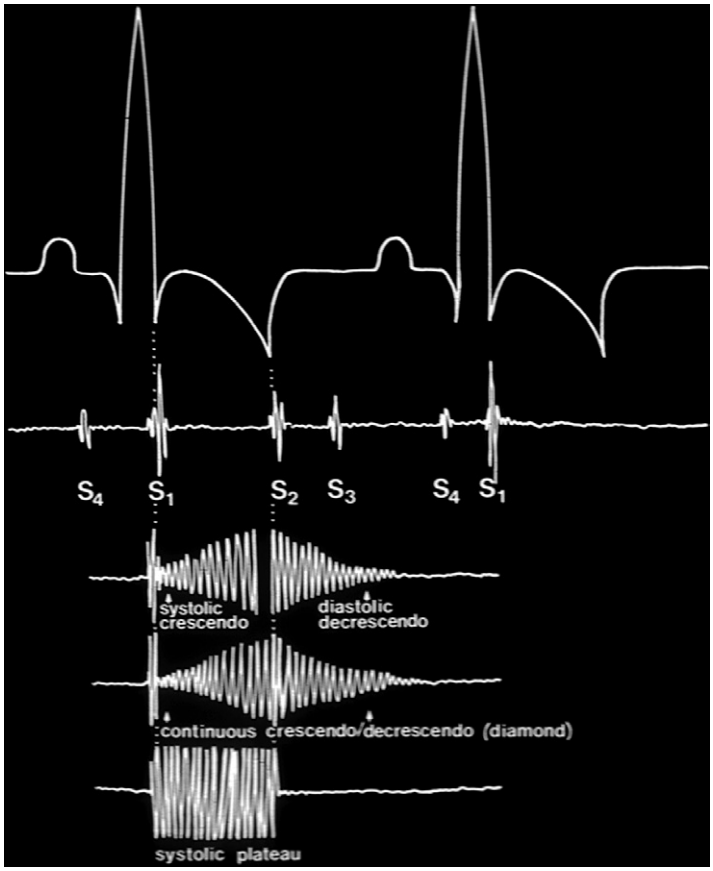


Fig. 1.12 Timing and quality of murmurs. (From Gompf RE: The clinical approach to heart disease: history and physical examination. In Fox PR, editor: *Canine and feline cardiology*, New York, 1988, Churchill Livingstone.)

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through the small bronchi and are louder on inspiration. Bronchial sounds are caused by air moving through the large bronchi and trachea and are heard best on expiration. Bronchovesicular sounds are the combination of these two sounds and are heard best over the hilar area.

- Abnormal respiratory sounds include attenuated sounds and increased, abnormal sounds.
- Attenuated bronchovesicular lung sounds are caused by thoracic masses, pleural effusion, pneumothorax, obesity, pneumonia, shallow breathing, or early consolidation of the pulmonary parenchyma.
- Rhonchi are caused by air passing through partially obstructed airways in the bronchial tubes or smallest airways. Rhonchi from the large bronchi are low pitched, sonorous, and almost continuous. They are heard best on inspiration. Rhonchi from the small bronchi are high pitched, sibilant, or squeaky and are heard best on expiration.
- Crackles are interrupted, crepitant, inspiratory sounds heard in many disease conditions and are not pathognomonic for pulmonary edema. They are caused by the opening of alveoli or airways that are collapsed or partially filled with fluid or bubbles bursting in the airways. They are further defined as fine or coarse in quality.
- Other sounds that can be auscultated include pleural friction rubs. Pleural friction rubs are grating, rubbing sounds heard during inspiration and expiration that are due to the moving of two relatively dry, roughened pleural surfaces against each other. Pericardial friction rubs are short, scratchy noises produced by pericarditis and heart movement. Pericardial knocks are diastolic sounds that occur in animals with constrictive pericarditis. Wheezes are relatively high-pitched musical sounds and are often a sign of pulmonary disease.

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Chapter 2

Radiology of the Heart

Wilfried Mai

Thoracic radiography is a key component of the cardiovascular evaluation. Careful attention to proper positioning is of primary importance to the use of radiographic guidelines for interpretation. Imperfect positioning causes major distortion of the perceived shape and size of cardiovascular structures, which can lead to significant interpretative errors. Radiographic interpretation relies heavily on possible disease considerations (i.e., differential diagnosis) derived from signalment, physical examination, and clinical pathology. Radiographic findings are not consistently specific enough to lead to a definitive diagnosis without supportive clinical evidence. The clinician must be aware of certain parameters and guidelines for interpretation to derive information from the radiographic image.

RADIOGRAPHIC TECHNIQUE

EXPOSURE TECHNIQUE

- Exposure technique will vary depending on equipment and radiographic detectors. The current standard for veterinary radiographic equipment is a 300 mA/125 to 75 kVp machine.
- Most veterinary practices nowadays are equipped with digital radiographic detectors and the use of analog radiography has become antiquated. As a result, the exposure charts have become simpler. It is extremely important to use the appropriate display algorithm (called the look-up table) for the body region examined. A thoracic look-up table provides the adequate contrast for the pulmonary parenchyma using an extended grayscale. It is very different from the high-contrast look-up tables used for abdominal radiography. As a result, whole body radiographs imaging the thoracic and abdominal cavity all at once are *highly* inappropriate, as the look-up table used will only be optimized for one of the two body regions and inadequate for the other. Thoracic radiographs and pulmonary patterns should not be interpreted on images that have been obtained using an abdominal radiographic look-up table. In addition, the large field of view used for whole body images results in geometric distortions, which complicate interpretation.
- Use of a grid is important for adequate image quality when the thickness of the thoracic cavity exceeds 10 cm.

RADIOGRAPHIC PROJECTIONS: TECHNICAL CONSIDERATIONS

Lateral Projection

- There are subtle differences in cardiac conformation and position when the right lateral radiographic projection is compared with the left lateral radiographic projection. These differences are not significant enough to warrant further discussion except to note that the same projection should be used on all serial radiographic examinations when repeated evaluation is required.
- Patient positioning and adequate radiographic exposure are critical to an accurate radiographic interpretation in the lateral projection.

Key Point

A normal heart can appear diseased and vice versa when positioning is not adequate.

Guidelines for proper exposure and positioning of a lateral thoracic radiograph (Fig. 2.1) include the following:

- Radiographic exposure should be adequate to define the spinous processes of the cranial thoracic vertebrae superimposed on the scapula.
- To ensure no rotation is present, the heads of corresponding pairs of ribs should be superimposed to each other.
- The thoracic limbs should be pulled cranially so that they are not superimposed over the cranial thorax or cranial margin of the heart.
- The radiographic exposure is taken during full inspiration, identified as an adequate lung field spacing between the caudal margin of the heart and the cupula of the diagram. Two primary disease considerations for consistent expiratory phase radiographs are the following:
  - Pickwickian syndrome (obesity hypoventilation syndrome), where the overabundance of abdominal fat prevents adequate inspiratory distraction of the diaphragm.

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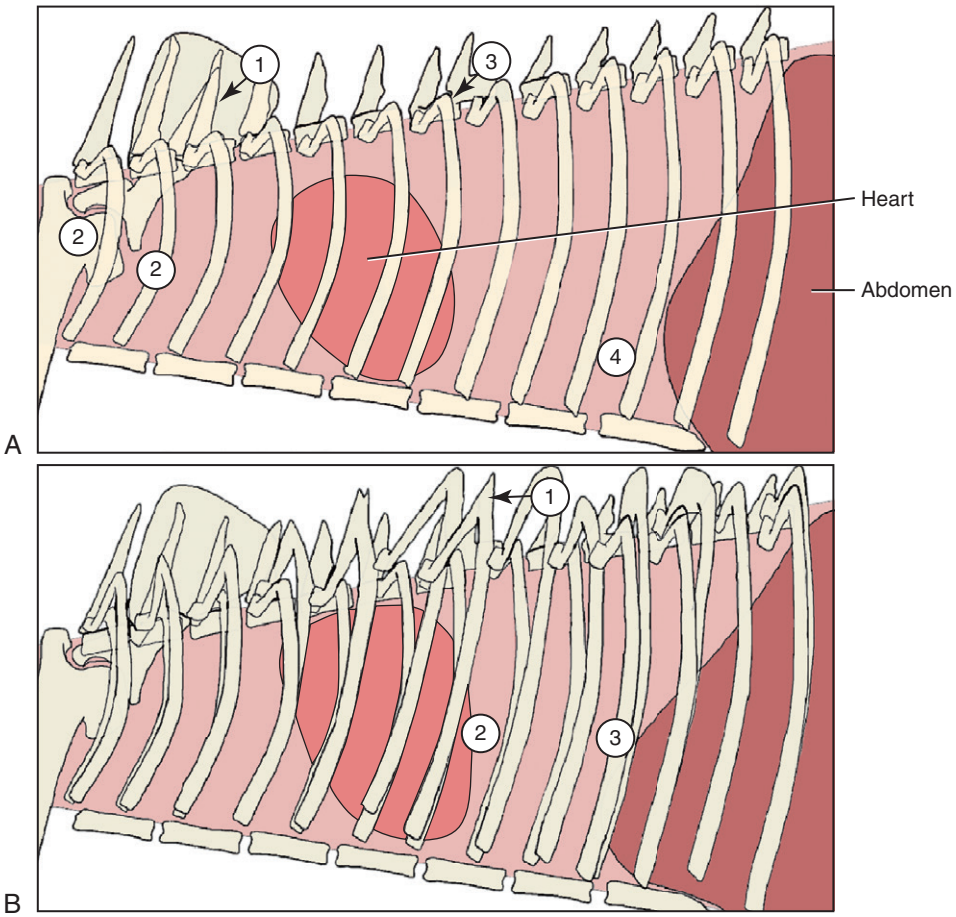


Fig. 2.1 **(A)** Guidelines for proper exposure and positioning of a lateral thoracic radiographic projection. (*1*) Exposure should allow delineation of the thoracic vertebral spinous processes superimposed over the scapula. (*2*) The thoracic limbs should be pulled cranially to not be overlapped with the cranial thorax. (*3*) The paired dorsal rib heads should be superimposed (compare with B). (*4*) The exposure should be performed during inspiration, which provides maximum separation between the caudal cardiac margin and diaphragmatic cupula. **(B)** Improperly positioned lateral thoracic radiographic projection (compare with A). (*1*) Nonsuperimposed paired left and right rib heads. (*2*) The oblique projection markedly distorts cardiac silhouette conformation and intrathoracic position. (*3*) Expiratory phase radiographic exposure with poor lung volume between caudal cardiac margin and cupula of the diaphragm.

- Upper airway disease, which most commonly causes obstruction during the inspiratory phase of respiration.

Dorsoventral/Ventrodorsal Projection

- The dorsoventral (DV) radiographic projection is preferred over the ventrodorsal (VD) projection for cardiac evaluation for two reasons:
  1. The anatomic positioning of the heart in the DV projection is less dependent on thoracic cavity conformation (deep-chested vs. barrel-chested breeds).
  2. The dorsal lung fields are better inflated when they are not dependent, resulting in a better contrast around the caudal lobar vessels, and hence an improved radiographic definition of the lobar pulmonary arteries and veins of the caudal lung fields. The DV projection also allows increased detection of caudodorsal pulmonary infiltrates, a common location for pulmonary changes secondary to cardiogenic pulmonary edema. However, an improperly positioned DV/VD projection is worthless for cardiac radiographic interpretation.

Key Point

Although the DV projection is preferred, a straight (symmetric) projection is the ultimate goal, with patient compliance determining which projection (DV or VD) is attainable.

Guidelines for proper exposure and positioning for the DV/VD projections (Fig. 2.2) include the following:

- Radiographic exposure should be sufficient to define the outline of the thoracic vertebrae superimposed over the cardiac silhouette.
- The kilovolt (peak) should be increased 10% from technique-chart values for obese patients. Examination of the thoracic body wall thickness on the VD view should assist in evaluation of obesity.

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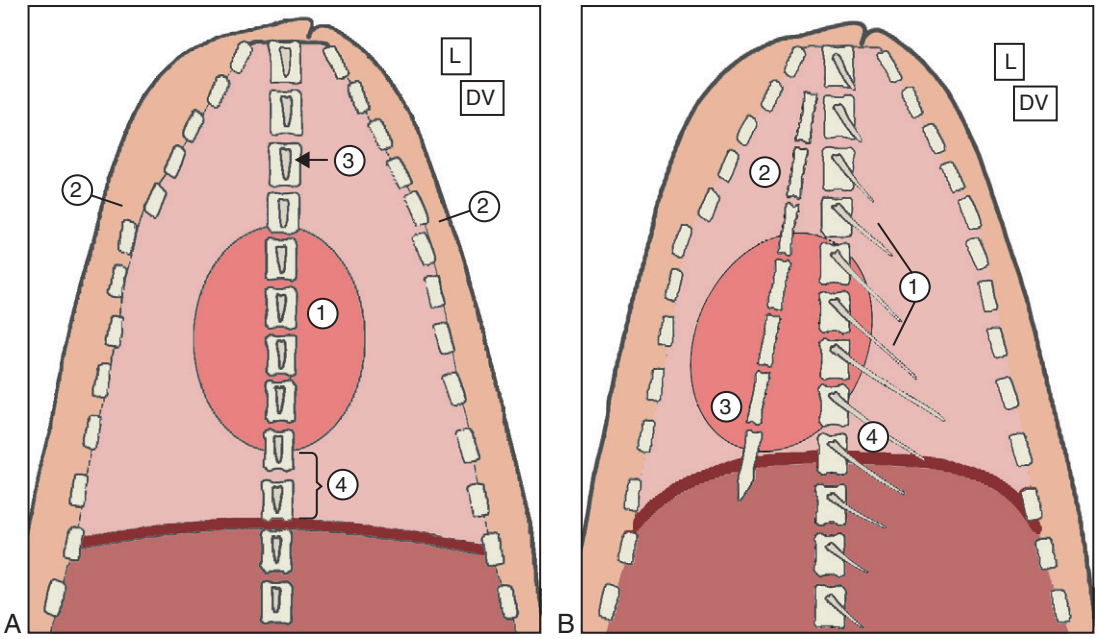


Fig. 2.2 **(A)** Guidelines for proper exposure and positioning of a dorsoventral (*DV*) thoracic radiographic projection. (*1*) The radiographic exposure should provide outline definition of thoracic vertebra superimposed over the cardiac silhouette. (*2*) Exposure should be increased (usually a 10% kVp increase with obesity as suggested by an increase in thoracic body wall thickness). (*3*) The thoracic vertebral spinous processes should be superimposed over the body portions for the entire length of the thoracic spine. (*4*) Adequate lung volume between the caudal cardiac margin and cupula indicates an inspiratory phase radiographic exposure. **(B)** Improperly positioned DV radiographic projection. Thoracic vertebral spinous processes projected over the left hemithorax (*1*) and the sternebrae projected over the right hemithorax (*2*); these projections indicate an oblique thoracic dorsoventral radiographic projection. A lack of lung volume between caudal cardiac margin (*3*) and cupula (*4*) indicates an expiratory phase radiographic exposure. *L*, Left.

- The spinous processes of the thoracic vertebrae should be centered over the vertebral bodies along the full length of the thoracic spine. The sternum and thoracic spine should be perfectly superimposed to each other and should be essentially indistinguishable.
- The radiograph is taken during full inspiration and identified by an adequate lung field spacing between the caudal cardiac margin and cupula of the diaphragm.

PROJECTION SELECTION IN CARDIAC-RELATED DISEASE

Pulmonary Edema

- The DV projection is preferred over the VD projection for radiographic detection of pulmonary edema. Due to better air inflation of the dorsal lung fields when the animal is in sternal recumbency, the DV view provides better contrast in the dorsal lung field, which is the most common location for the formation of early cardiogenic pulmonary edema. Adequate exposure is critical to ensure definition of caudal pulmonary vasculature superimposed over the cupula of the diaphragm.
- The radiographic evaluation of the caudodorsal pulmonary vasculature is the best objective parameter to differentiate left-sided congestive heart failure from other causes of caudodorsal pulmonary infiltrates. Vessels in healthy lungs are detected by their soft tissue opacity that contrasts with the normal radiolucent gas-filled lung parenchyma that surrounds them. As pulmonary parenchyma (interstitial spaces and alveoli) becomes filled with edema fluid, the normal radiographic soft tissue/gas contrast is lost, and delineation of the vessels diminishes. In other words, the vessels start to "disappear" due to silhouetting with the increased opacity of the surrounding edematous lung parenchyma (Fig. 2.3).
- When interpreting pulmonary opacity, the phase of respiration at which the radiographs were obtained is important to take into account, as pulmonary disease can be mimicked when underinflation decreases the parenchymal gas content per unit volume and thus reduces the radiographic contrast between lung parenchyma and associated vasculature. This phenomenon is especially common in older patients, who already have slightly increased pulmonary parenchymal radiographic opacity because of age-related pulmonary degenerative changes (interstitial fibrosis, bronchial mineralization, heterotopic pulmonary bone formation).

PLEURAL EFFUSION

- Pleural effusion is radiographically evident as focal areas of increased soft tissue opacity located within

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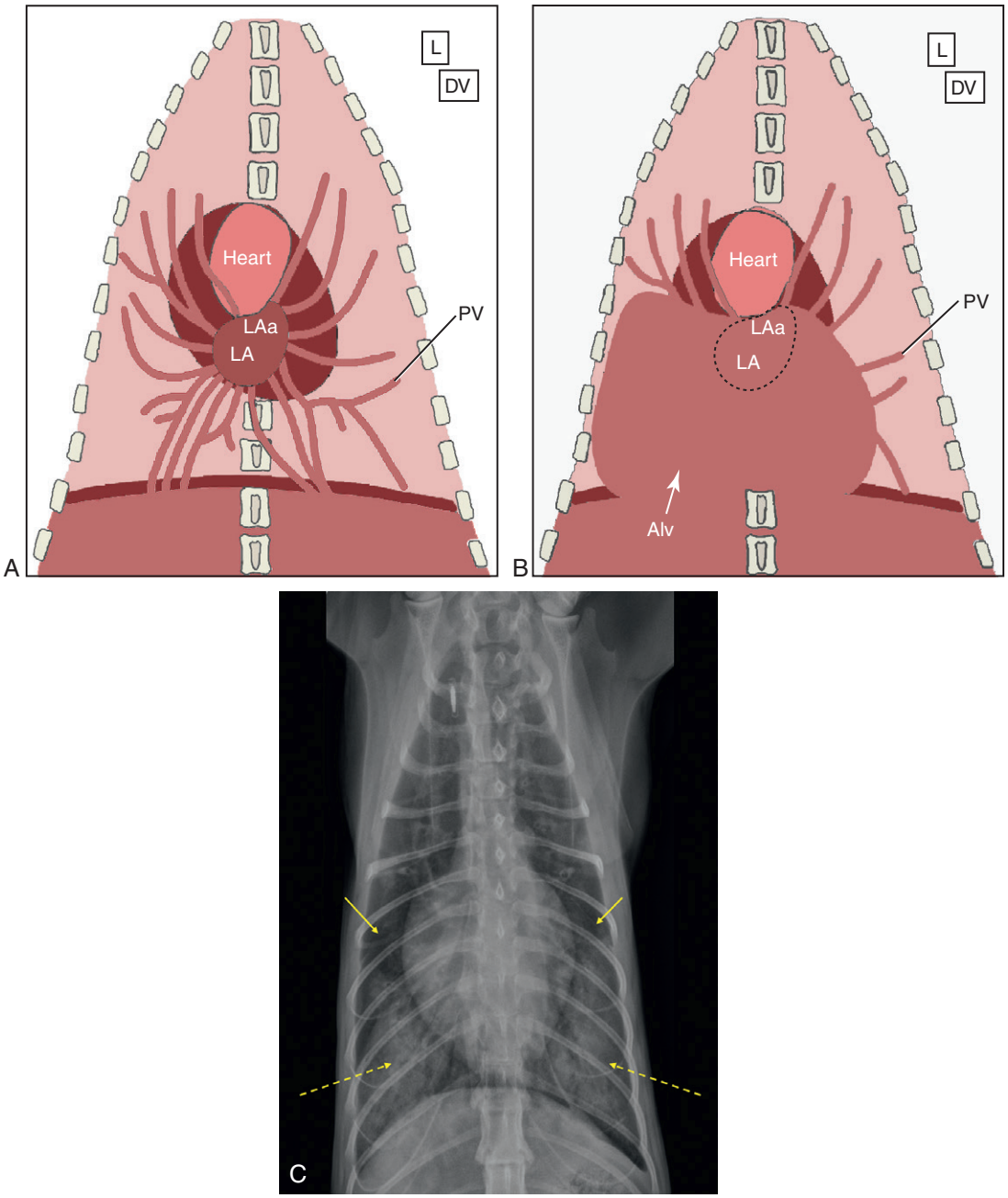


Fig. 2.3 **(A)** Normal radiographic definition and contrast of pulmonary venous vasculature (*PV*) with surrounding normal radiolucent lung parenchyma. **(B)** Radiographic obliteration of PV by alveolar consolidation (*Alv*) of hilar and caudal lung lobes, a characteristic distribution for cardiogenic pulmonary edema. *DV*, Dorsoventral; *L*, left; *LA*, left atrium; *LAa*, left atrial auricular appendage. **(C)** VD projection of the thorax in a cat with hypertrophic cardiomyopathy and left-sided congestive heart failure. There is a severe alveolar lung pattern in the caudal lung lobes (*dashed arrows*) obscuring the pulmonary vasculature; pleural fissure lines (*solid arrows*) are also seen suggestive of pleural effusion. The heart is enlarged with a Valentine shape.

the thoracic cavity. It causes separation of lung lobes from both the thoracic wall (VD/DV views) and sternum/spine (lateral views).

- On the lateral projection, pleural effusion causes an increase in the soft tissue thickness of the caudodorsal thoracophrenic angle and diaphragm, as well as between the heart and sternum. In the ventral thorax, pleural effusion causes loss of definition of the cranial and caudal margins of the heart (radiographic positive-silhouette sign). It also causes separation between the lung lobes, creating pleural fissure lines, which appear as linear soft tissue opacities at anatomic locations consistent with the anatomic interlobar fissures (Fig. 2.4).
- On the VD/DV projections, pleural effusion causes pleural fissure lines in the expected location of interlobar fissures, and soft tissue thickening in the

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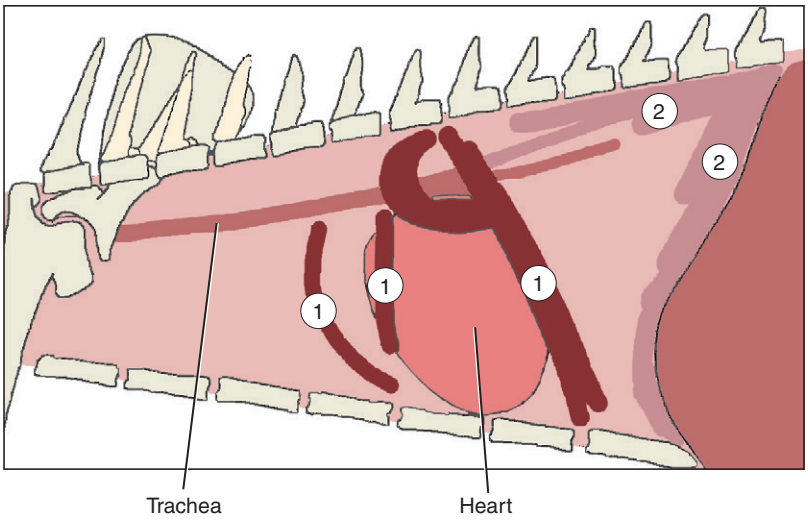


Fig. 2.4 Lateral thoracic radiographic projection of pleural effusion. Intrathoracic fluid accumulation causes separation of adjacent lung lobes by (*1*) linear interlobar opacities, radiographically defined as pleural fissures, and (*2*) separation of lung lobes from the thoracic wall.

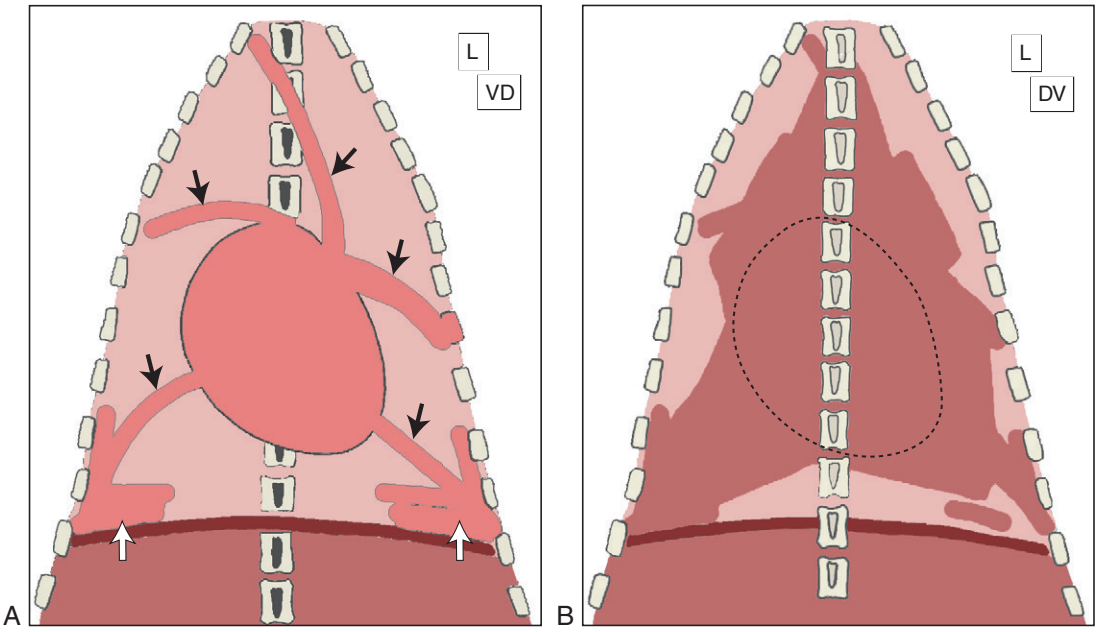


Fig. 2.5 **(A)** Ventrodorsal (*VD*) thoracic radiographic projection of pleural effusion consisting of pleural fissure lines (*closed arrows*) with blunting of the thoracophrenic angles (*open arrows*). Note that the cardiac silhouette is still well outlined. **(B)** Dorsoventral *(DV)* thoracic radiographic projection of pleural effusion. The intrathoracic fluid distribution creates a "positive-silhouette sign" where a complete loss of the cardiac silhouette has occurred. Thus the VD projection (A) is preferred for cardiac silhouette definition in the presence of pleural effusion. *L*, Left.

costophrenic angles in the right and left caudolateral lung fields.

- In cases of pleural effusion, the VD projection is much preferred over the DV view for the assessment of cardiac size and shape. If intrathoracic fluid volumes are severe enough, the heart can effectively disappear on the DV view because fluid accumulates ventrally around the heart due to gravity causing a positive-silhouette sign obscuring the cardiac margins (Fig. 2.5). However, patient positioning for the DV projection puts less physiologic demand on the patient compromised by pleural effusion and thus the patient's clinical stability and degree of respiratory compromise should always be taken into account in deciding for a DV or VD projection.
- If significant amounts of pleural effusion are suspected, increasing radiographic exposure may improve intrathoracic radiographic contrast. When possible, thoracocentesis and fluid drainage before radiography are always preferred.

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RADIOGRAPHIC ANATOMY

LATERAL THORACIC RADIOGRAPHIC PROJECTION

Cardiac Silhouette

- In the dog and the cat, the heart is slightly rotated along its base-apex axis so that the right-sided cardiac chambers are positioned more cranially, and the left-sided cardiac chambers are positioned more caudally. Thus on lateral radiographs, the cranial margin of the cardiac silhouette represents the right side of the heart, while the caudal margin represents the left side (Figs. 2.6–2.8).
- The radiographic silhouette of the heart is ovoid, with the apex more pointed in conformation than the broader base. This base-apex difference in conformation is accentuated in the cat. The heart axis is defined by drawing a line from the tracheal bifurcation (carina) to the apex; this axis typically forms an angle of approximately 45 degrees to the sternum. This angle can decrease in the cat with age and is often called a "lazy" heart. It has been postulated that this may be related to a loss of aortic connective tissue elasticity. This is most often seen in cats older than 7 years. Shallow, barrel-chested dog breeds (e.g., Dachshund, Lhasa Apso, Bulldog) tend to have more globular-shaped hearts, with increased sternal contact of the cranial margin of the heart.
- The heart chambers can be roughly defined by a line connecting the apex to the tracheal bifurcation and a second line perpendicular to the base-apex axis and positioned at the level of the ventral aspect of the caudal vena cava (see Fig. 2.8).
- The dorsal cardiac margin includes both atria, pulmonary arteries and veins, the cranial and caudal vena cavae, and the aortic arch (see Figs. 2.6–2.8). The cranial border is formed by both the right ventricle and the right atrial appendage, resulting in the radiographically defined "cranial waist" (see Figs. 2.6 and 2.8). The caudal margin is formed by the left atrium and left ventricle, with the atrioventricular junction defined as the radiographic "caudal waist."
- The base-to-apex cardiac dimension or length occupies approximately 70% of the DV distance of the thoracic cavity at its position within the thorax. For objective measurements, thoracic cavity distance between the thoracic spine and the sternum should be measured *at an axis perpendicular to the thoracic spine*.
- The cranial-caudal dimension or width, as it appears on the lateral projection, is measured at its maximum width (which is usually at the level of the ventral aspect of the insertion of the caudal vena cava) and perpendicular to the base-apex axis. This has been classically defined as between 2.5 (deep-chested conformation breeds [Setters, Afghans, Collies]) and 3.5 (barrel-chested conformation breeds [Dachshunds, Bulldogs]) intercostal spaces (ICSs) in the dog, and between 2.5 and 3.0 ICSs in the cat. The ICS measurement is made at an axis *perpendicular to the long axis of the ribs*. Thus the cardiac width distance

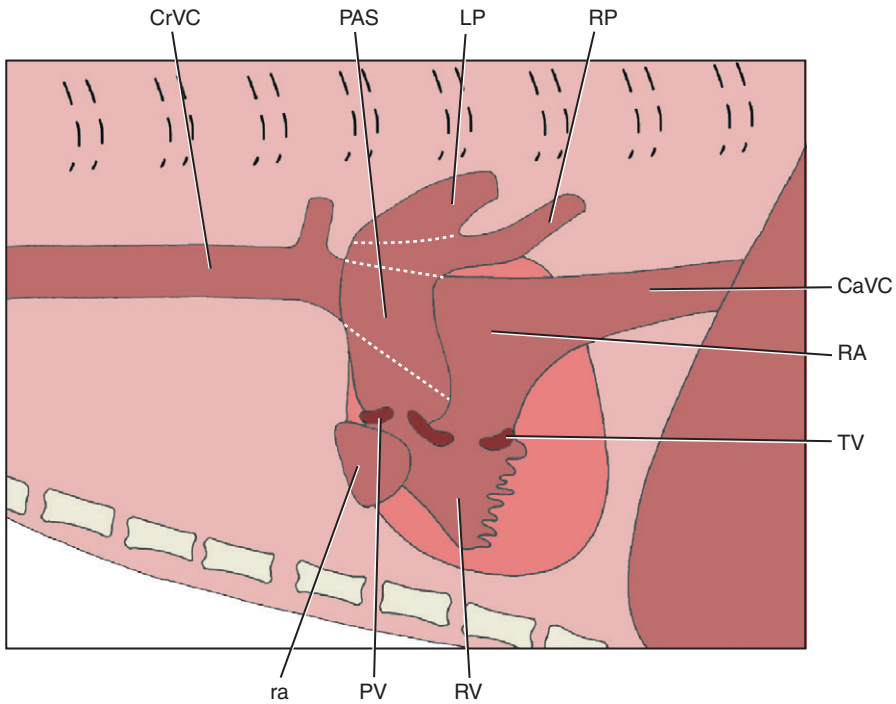


Fig. 2.6 Schematic lateral thoracic radiographic projection of the relative position and size of the right-side structures of the heart. Note the more cranial position of the right chambers of the heart. *CaVC*, Caudal vena cava; *CrVC*, cranial vena cava; *LP*, left pulmonary artery; *PAS*, main pulmonary artery segment; *PV*, pulmonic valve; *ra*, right atrial auricular appendage; *RA*, right atrium; *RP*, right pulmonary artery; *RV*, right ventricle; *TV*, tricuspid valve.

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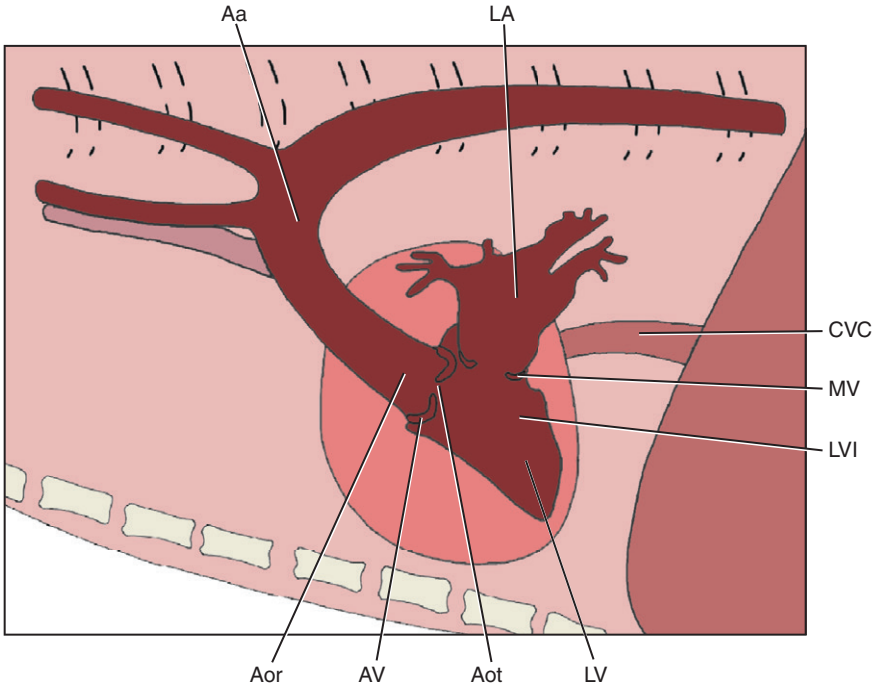


Fig. 2.7 Schematic lateral thoracic radiographic projection of the relative position and size of the left-side structures of the heart. Note the more caudal position of the left chambers of the heart. *Aa*, Aortic arch; *Aor*, aorta; *AV*, aortic valve; *Aot*, aortic outflow tract; *CVC*, caudal vena cava; *LA*, left atrium; *LV*, left ventricle; *LVI*, left ventricular inflow tract; *MV*, mitral valve.

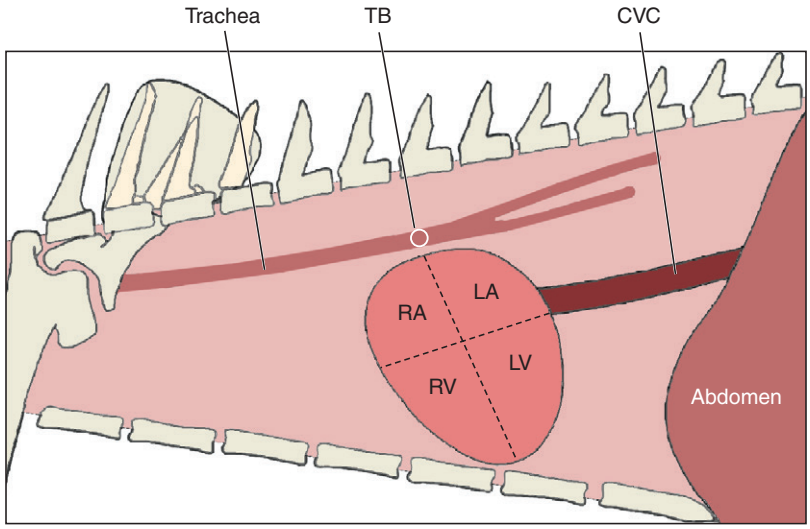
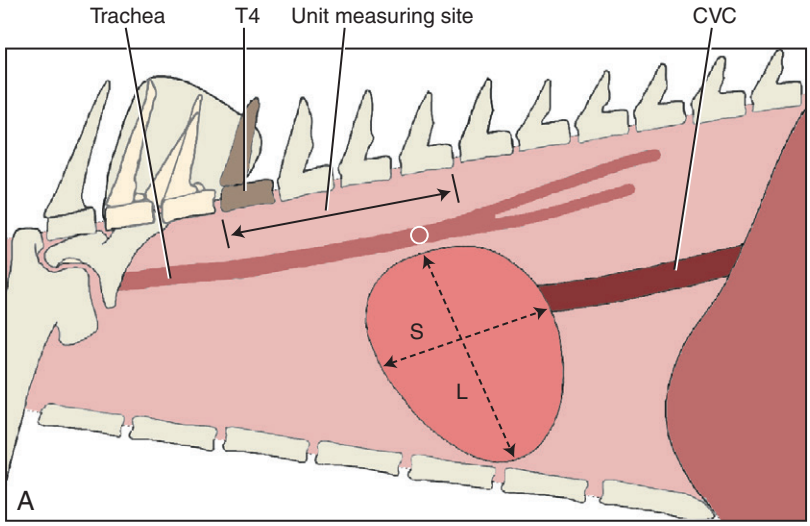


Fig. 2.8 Schematic lateral thoracic radiographic projection outlining the approximate location of the four heart chambers. *CVC*, Caudal vena cava; *LA*, left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle; *TB*, tracheal bifurcation.

determination may have to be shifted in axis angle before comparison with ICS length.

- A more objective determination of cardiac size has been formulated and uses a vertebral scale system in which cardiac dimensions are scaled against the length of specific thoracic vertebrae (Fig. 2.9A and 9B).
  - In lateral radiographs, the long axis of the heart (L) is measured with a caliper extending from the ventral aspect of the left mainstem bronchus (tracheal bifurcation hilus, carina) to the left ventricular apex. The caliper is repositioned along the vertebral column, beginning at the cranial edge of the fourth thoracic vertebra. The length of the heart is recorded as the number of vertebrae caudal to that point and estimated to the nearest tenth of a vertebra.
  - The short-axis dimension of the heart (S) is measured along a line perpendicular to the long axis positioned at the level of the ventral border of the caudal vena cava and is measured in the same manner beginning at the fourth thoracic vertebra.

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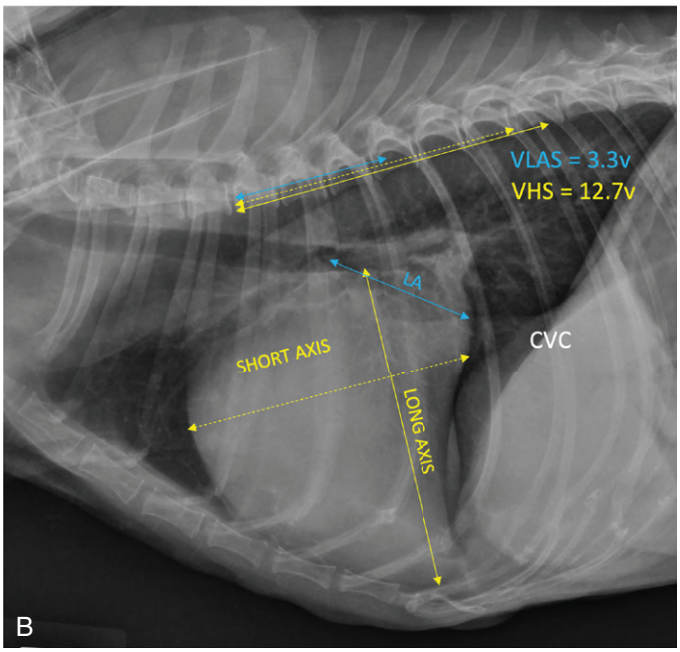


Fig. 2.9 **(A)** Schematic representation parameters for the vertebral scale system of cardiac size. The vertebral heart sum (*VHS*) is the sum of the long-axis cardiac dimension (*L*) and the maximal perpendicular short-axis dimension (*S*). S and L are measured in vertebral units beginning at T4. **(B)** Lateral radiograph of the thorax in a dog with mitral endocardiosis and insufficiency. The yellow markings illustrate the calculation of the vertebral heart score (*VHS*) and the blue markings illustrate the calculation of the vertebral left atrial size (*VLAS*). *CVC*, Caudal vena cava; *LA,* left atrium; *v,* vertebral unit.

If obvious left atrial enlargement is present, the short-axis measurement is made at the ventral juncture of left atrial and caudal vena caval silhouettes.

- The lengths in vertebrae (v) of the long and short axes are then added to obtain a vertebral heart sum or score (VHS), which provides a single number representing heart size proportionate to the size of the dog.
- The average VHS on lateral radiographs in the dog is 9.7 v (range, 8.5–10.5 v), and 7.3 v (±0.5 v) in the cat. Caution must be exercised in some breeds that have excessively disproportionate skeletal-body weight conformations. An example is the English Bulldog, which has relatively small thoracic vertebrae and commonly has hemivertebrae as well; thus a normal heart may be interpreted as large with the VHS method.
- The normal VHS is variable across different canine breeds and many breed-specific ranges have been established in the literature.
- Although the VHS concept is more objective and has low inter-observer variability, clinical judgment is still necessary to avoid over-diagnosing or under-diagnosing heart disease.

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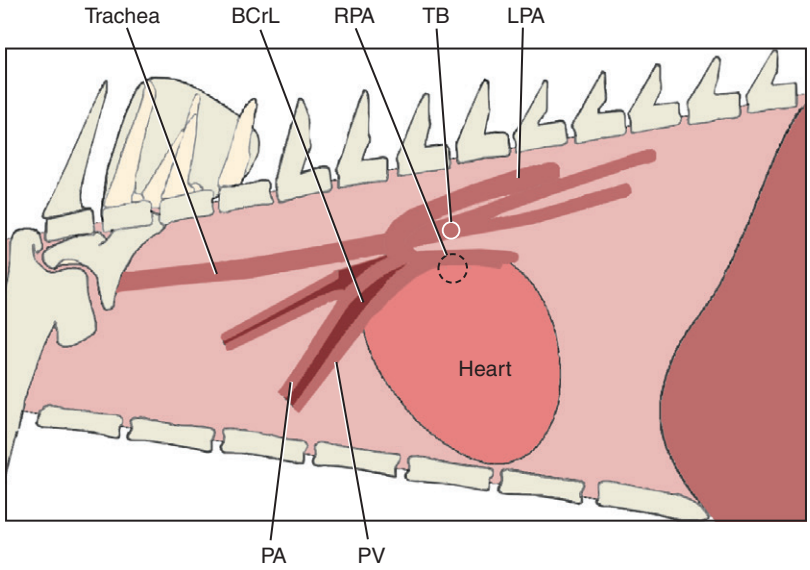


Fig. 2.10 Pulmonary vascular anatomy in the lateral thoracic projection. Cranial lung lobe branch of the pulmonary artery (*PA*), cranial lung lobe branch of the pulmonary vein (*PV*), end-on view of the right main pulmonary artery (*RPA*) as it traverses the thorax from left to right, and left main pulmonary artery (*LPA*). *BCrL*, Bronchus to a cranial lung lobe; *TB*, tracheal bifurcation (carina).

- The sensitivity of the VHS to detect cardiac disease depends on the cardiac condition at hand. It performs usually better in cardiac diseases that cause eccentric cardiomegaly (e.g., valvular endocardiosis/insufficiency) than disease that cause concentric cardiomegaly (e.g., pulmonic or aortic stenosis).
- The lateral projection VHS tends to be higher on right lateral compared to left lateral radiographs; therefore the same recumbency should be used to follow-up changes of VHS over time in the same patient.

- Recently a newer index of radiographic left atrium size, the vertebral left atrial size (VLAS), was included in the American College of Veterinary Internal Medicine consensus guidelines for the diagnosis and treatment of myxomatous mitral valve disease.
  - This index is measured on lateral radiographs, drawing a line from the center of the most ventral aspect of the carina to the most caudal aspect of the left atrium where it intersects with the dorsal border of caudal vena cava. That measurement is then transposed onto the spine beginning at the cranial edge of T4 and extending caudally just ventral and parallel to the vertebral canal; the left atrial size is then expressed in vertebral body units to the nearest 0.1 vertebra.
  - Reference interval of the VLAS in dogs was established to be 1.4 to 2.2, though there may be some inter-breed variability.
  - A cut-off of 2.3 was proposed as a predictor of left atrial dilation in dogs.
- Even more recently, a heart-to-single-vertebra ratio (HSVR) was introduced. For that ratio, the sum of long and short axis of the heart is measured like the classic VHS. A ratio of this sum to the length of T4, T5, T6, T7, or T8 (including their respective caudal intervertebral disc space) is then obtained to obtain an HSVR. This ratio had strong correlation with full VHS measurement, and good-to-excellent inter- and intra-observer variation. This simpler ratio was proposed as an alternative to the VHS in dogs that have thoracic vertebral abnormalities (e.g., congenital hemivertebrae), in which the consecutive thoracic vertebral bodies are more challenging to use for computation of a true VHS.

Pulmonary Vessels

- The main pulmonary artery (pulmonary trunk) cannot be seen on the lateral projection because of a positive-silhouette sign with the craniodorsal base of the heart. The left pulmonary artery can sometimes be seen extending dorsal and caudal to the tracheal bifurcation (carina). The right pulmonary artery is frequently seen end-on as it leaves the main pulmonary artery immediately ventral to the carina (Fig. 2.10). This end-on appearance may be confused with a mass lesion on normal radiographs and is accentuated in cases of pulmonary hypertension such as heartworm disease. The pulmonary veins are best identified as they enter the left atrium caudal to the heart base.
- With the use of the larger, more proximal segments of the mainstem bronchi as a reference, the pulmonary arteries are dorsal to the bronchus, and the pulmonary veins are ventral to the bronchus (see Fig. 2.10).
- The vessels to the cranial lung lobes are usually seen as two pairs of vessels, each with their respective

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bronchi. The more cranial pair of vessels generally corresponds to the side on which the lateral projection was made. Thus in the right lateral projection, the right cranial lobar vessels are more cranial than vessels of the left cranial lung lobe. The pulmonary arteries and veins should be equal in size. There usually is more overlap between the right and left cranial lobar vessels on the right lateral view, and more separation on the left lateral view; hence, the left lateral view is preferred to evaluate specifically the right cranial lobar vessels, which will be more conspicuous due to better inflation of the right (nondependent) lung on that view. The width of the vessels where they cross the fourth rib should not exceed the width of the narrowest portion of that rib at its juncture with the rib head (the dorsal aspect of the rib near the thoracic spine). The dorsal section of the rib is used as a reference to adjust for radiographic magnification because of thoracic conformation.

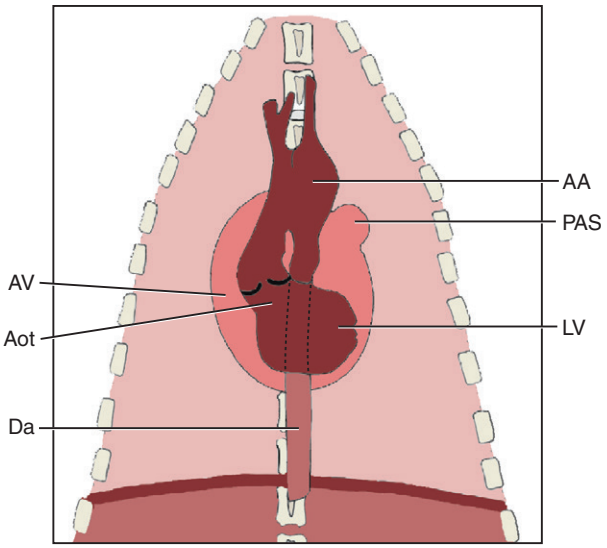


Fig. 2.11 Schematic anatomy of the chambers and vasculature of the left ventricular outflow tract of the heart in the dorsoventral radiographic projection. *AA*, Aortic arch; *Aot*, aortic outflow tract; *AV*, aortic valve; *Da*, descending aorta; *LV*, left ventricle; *PAS*, pulmonary artery segment.

DORSOVENTRAL AND VENTRODORSAL PROJECTIONS

Cardiac Silhouette

- On DV/VD radiographs, the canine heart appears as an elliptical opacity with its base-apex axis orientation approximately 30 degrees to the left of the midline. The width of the heart across its widest point is usually 60% to 65% of the thoracic width at that same location. In the cat, the cardiac axis is closer to midline, and its width does not usually exceed 50% of the width of the thoracic cavity during full inspiration.
- The cardiac silhouette may be artificially widened in the obese patient because of an excessive amount of pericardial fat. In these cases, the cardiac silhouette margin appears to be less well defined because the radiographic contrast at heart/pericardial fat and pericardial fat/lung interfaces is less than at a heart (soft tissue)/lung interface.
- In long, narrow-chested breeds, the heart stands more vertical in the thorax and thus produces a smaller and more circular cardiac silhouette conformation on DV/VD projections, which should not be mistaken for pericardial effusion. Conversely, dog breeds with a short, barrel-chested conformation have a radiographic silhouette that appears wider than that of breeds with intermediate thoracic conformation.
- The margins of the heart that create the cardiac silhouette contain a number of structures that often overlap. A clock face analogy can be used to simplify the location of these structures. The aortic arch extends from the 11 to 1 o'clock position (Fig. 2.11). The main pulmonary artery is located from the 1 to 2 o'clock position, with its radiographic designation as the pulmonary artery segment (PAS) (Figs. 2.12 and 2.13). In the cat, the body of the left atrium proper forms the 2 to 3 o'clock position of the cardiac silhouette. In the dog, the left atrium is superimposed over the caudal portion of the cardiac silhouette in the DV projection, between the caudal lobar bronchi (see Fig. 2.12). With severe cases of left atrial enlargement in the dog, the left auricular appendage contributes to

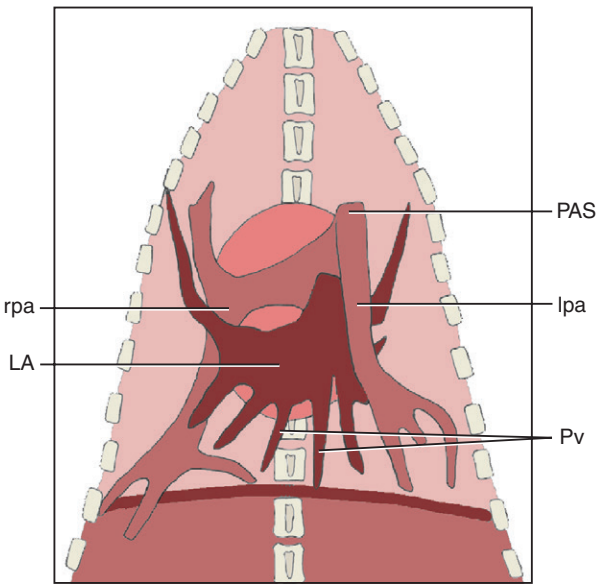


Fig. 2.12 Schematic anatomy of the pulmonary vasculature in the dorsoventral projection. *LA*, Left atrium; *lpa*, left pulmonary artery; *PAS*, main pulmonary artery (radiographic description-pulmonary artery segment); *Pv*, pulmonary veins; *rpa*, right pulmonary artery.

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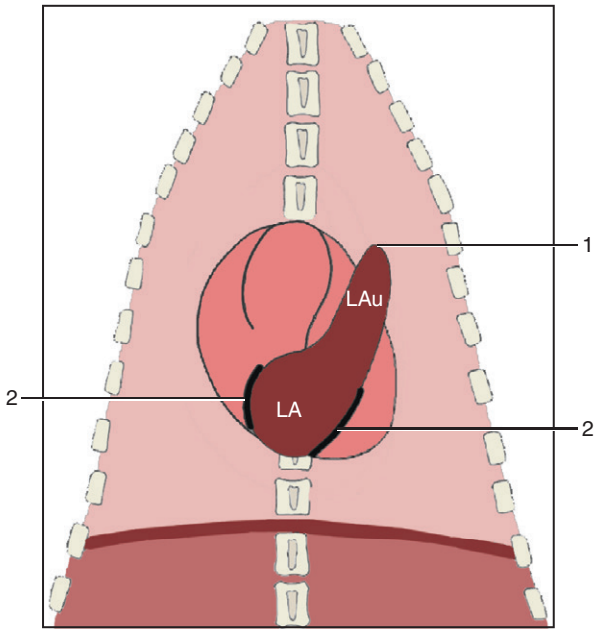


Fig. 2.13 Dorsoventral thoracic radiographic projection of a dog with severe left atrial (*LA*) enlargement. The left atrial auricular appendage (*LAu*) contributes to the cardiac silhouette at the 2–3 o'clock position (*1*). The body of the left atrium superimposed over the caudal cardiac silhouette produces a radiolucent "Mach" line, a radiographic edge effect caused by an acute change in soft tissue thickness (*2*).

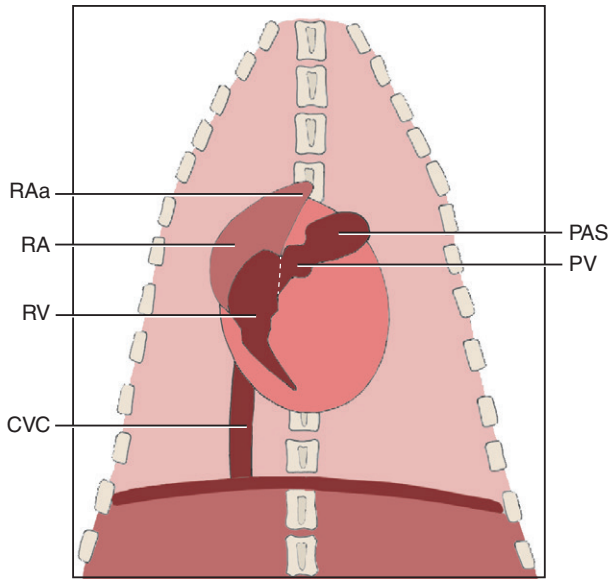


Fig. 2.14 Schematic anatomy of the chambers and outflow tract of the right side of the heart in the dorsoventral radiographic projection. *CVC*, Caudal vena cava; *PAS*, main pulmonary artery segment; *PV*, pulmonic valve; *RA*, right atrium; *RAa*, right atrial auricular appendage; *RV*, right ventricle;

the definition and enlargement of the cardiac silhouette at the 2 to 3 o'clock position (see Fig. 2.13). The left ventricle forms the left heart margin from the 2 to 5 o'clock position (see Fig. 2.11). The right ventricle is located from the 7 to 11 o'clock position (the right ventricle does not extend to the apex of the heart) (Fig. 2.14). The right atrium is located at the 9 to 11 o'clock position (see Fig. 2.14).

- Pericardial fat in the dog can asymmetrically contribute to cardiac silhouette enlargement at the 4 to 5 o'clock and 8 to 11 o'clock positions. In cats, pericardial fat on DV/VD views can cause a squared protrusion along the right cranial cardiac margin in the area of the right atrium.

Pulmonary Vessels

- The pulmonary arteries originate from the main pulmonary artery (PAS) with the right branch coursing transversely, superimposed over the cranial portion of the heart silhouette, extending beyond the right heart margin at approximately the 8 o'clock position (see Fig. 2.12). The left pulmonary artery branch courses caudally, superimposed over the caudal left ventricular portion of the heart, and extends beyond the left heart margin at approximately the 4 o'clock position. The pulmonary veins are best seen as they enter the left atrium along the caudal margin of the cardiac silhouette (see Fig. 2.12). Compared with the pulmonary arteries, they are clustered in a more axial position. Thus the pulmonary arteries extend to both the cranial and caudal lung fields in a more abaxial position relative to the pulmonary veins. As a result, on a DV or VD projection, when evaluating the cranial and caudal lobar bronchi, the lobar artery is located lateral to the bronchus and the lobar vein is located medial to the bronchus.
- The caudal lobar vessels are easier to identify and trace radiographically than the vessels to the cranial lung lobes or right middle lung lobe. The size of these vessels is compared to the width of the ninth ribs where they intersect and normally the vessels should be about equal to the rib's width at that level (i.e., the summation opacity of the vessel and rib should roughly form a square).
- The aortic arch is within the cranial mediastinum at the cranial heart margin and is normally not visible. The descending aorta is superimposed over the heart and extends caudally, dorsally, and medially. The left lateral margin of the aorta can be seen to the left of the vertebral column on both DV and VD views (see Fig. 2.11). The caudal vena cava courses cranially from the diaphragm to the right of midline and into the right caudal margin of the heart (see Fig. 2.14). This is one of the most useful landmarks for determination of proper orientation of the DV radiograph on a viewing station.

RADIOGRAPHIC INTERPRETATION

BASIC PRINCIPLES

A systematic evaluation of the entire thoracic cavity involves adherence to and inclusion of the

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following steps with each radiographic interpretation. Abnormalities supportive of disease should be substantiated on multiple radiographic views where applicable.

- Evaluate the radiographs for technical quality, positioning, and proper exposure. *If the study is substandard, then stop right here and repeat the radiographic study*.
- Determine the phase of respiration; inspiratory phase is preferred to ensure adequate lung inflation and avoid misdiagnosis of increased pulmonary opacity.
- Review the entire thoracic cavity: spine, sternum, diaphragm, thoracic wall, ribs, cranial and caudal mediastinum, conformation, and position of the diaphragm.
- Review the portion of the cranial abdomen included in the projection. A cursory evaluation of abdominal contrast, detail, and hepatic size (with gastric axis) can be performed, keeping in mind that abdominal contrast on digital thoracic radiographs obtained using a thoracic algorithm (look-up table) may look artificially lowered.
- Evaluate the position, course, and diameter of the trachea and mainstem bifurcations.
- Evaluate the position of the cardiac apex and caudal mediastinum.
- Evaluate the size, shape, and course of the main pulmonary artery, and peripheral pulmonary arteries and veins.
- Evaluate the lung fields for hyperinflation or underinflation and for distribution and pattern of increased or decreased opacity.
- Evaluate the cardiac margin (cranial, caudal, right, left "clock position" segmentation) for enlargement, abnormal position, or conformation.

INTERPRETATION PITFALLS: RADIOGRAPHIC MIMICS OF CARDIAC DISEASE

NONCARDIAC CAUSES OF CHANGES IN CARDIAC POSITION

- Asymmetrical changes in pulmonary or pleural space volume (e.g., pulmonary collapse or atelectasis, pulmonary mass, asymmetrical pleural effusion or pneumothorax, previous lobectomy) can cause a mediastinal shift and alter the position and axis of the heart in the thoracic cavity particularly on VD or DV projections.
- If radiographs are taken on animals after a prolonged period of lateral recumbency, or during/immediately after heavy sedation or general anesthesia, hypostatic congestion and atelectasis of the dependent lung fields can produce a mediastinal shift and unilateral increased lung opacity, altering cardiac position and/or mimicking pulmonary disease.
- Mediastinal mass lesions can affect the cardiac position and axis and obscure the cranial cardiac margins when in contact with the heart by producing a radiographic positive-silhouette sign.
- Pneumothorax commonly produces elevation of the cardiac apex from the sternum on the lateral radiographs. This is supported by other radiographic signs of pneumothorax:
  - Premature termination of lung vasculature into the periphery of the thoracic cavity
  - Lung lobe margin detection outlined by nonparenchymal free intrathoracic gas.
- Sternal conformational abnormalities due to congenital defects (e.g., *pectus excavatum*) or previous trauma can alter cardiac position and axis.
- In dogs with deep-chested conformation, the cardiac apex can be elevated far enough from the sternum to mimic pneumothorax. Scrutinizing carefully the area between heart and sternum looking for fine bronchovascular markings should help differentiation from a true pneumothorax.

NONCARDIAC CAUSES OF CHANGES IN CARDIAC SIZE

- Younger animals appear to have larger hearts relative to their thoracic size than do mature patients. In kittens the vertebral heart scale (median 9.5 vertebrae, range 8.0–10.9) is higher than that reported for adult cats; however, in puppies, the VHS was determined to be like that in adult dogs (9.7 ± 0.5 vertebrae).
- The heart appears smaller on inspiration than on expiration. During expiration, increased sternal contact of the right-sided heart margin and dorsal elevation of the trachea occurs, falsely suggesting right-sided heart enlargement.
- Prolonged moderate-to-severe anemia in cats and dogs has been shown to cause compensatory left-sided cardiomegaly and an increased vertebral heart scale measurement.
- Hypovolemic patients often have small hearts (microcardia); causes included acute fluid loss, such as from severe gastrointestinal disease or hemorrhage, as well as endocrine disorders such as hypoadrenocorticism. Concurrent pulmonary hyperinflation to compensate for hypoxemia may exacerbate the impression of microcardia.

EVALUATION OF HEART CHAMBER ENLARGEMENT

RIGHT ATRIAL ENLARGEMENT

Radiographic Signs

- Lateral projection (Fig. 2.15):
  - Accentuation of the cranial waist. Preferential enlargement of the more dorsal margin of the cranial margin of the cardiac silhouette defines selective enlargement of the right atrial auricular appendage.

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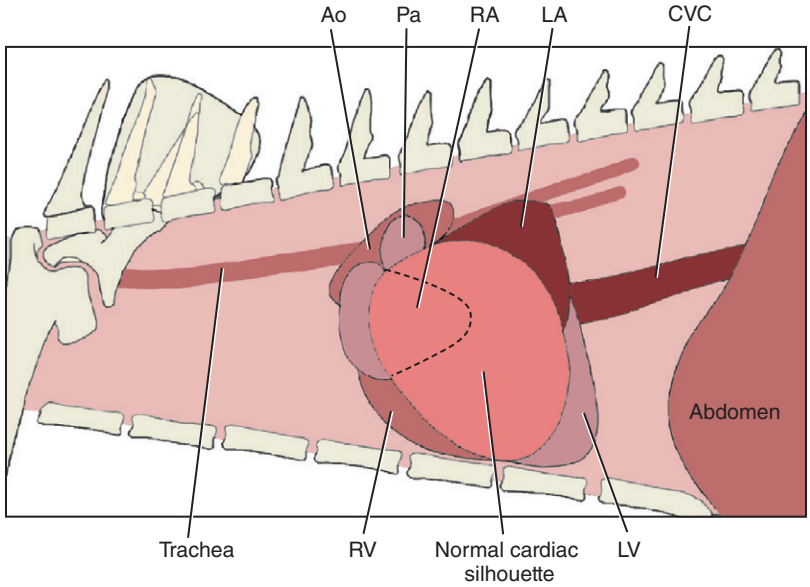


Fig. 2.15 Cardiac silhouette changes associated with vessel and chamber enlargement in the lateral thoracic radiographic projection. *Ao*, Aortic arch; *CVC*, caudal vena cava; *LA*, left atrium; *LV*, left ventricle; *Pa*, main pulmonary artery; *RA*, right atrium; *RV*, right ventricle. *Dotted line*, area of right atrial superimposition over the right ventricle.

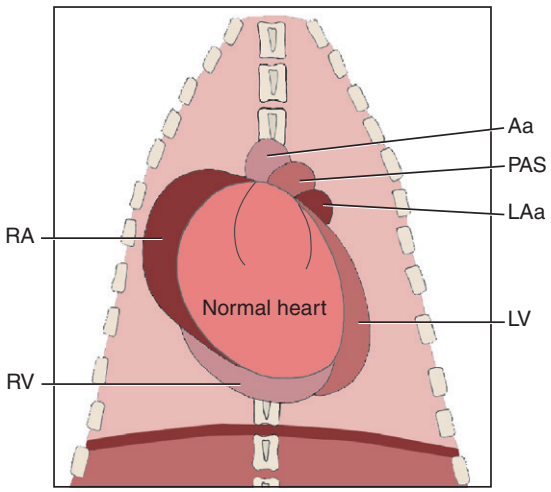


Fig. 2.16 Cardiac silhouette changes associated with vessel and chamber enlargement in the dorsoventral radiographic projection. *Aa*, Aortic arch; *LAa*, left atrial auricular appendage; *LV*, left ventricle; *PAS*, main pulmonary artery segment; *RA*, right atrium; *RV*, right ventricle.

  - Relative increased soft tissue opacity of the cranial aspect of the cardiac silhouette due to increased soft tissue thickness of the right atrium superimposed over the right ventricle.
- DV projection (Fig. 2.16):
  - Enlargement of the cardiac margin at the 9 to 11 o'clock position.
  - Enlargement can be dramatic in severe cases (especially in the cat) and can be easily mistaken for a pulmonary hilar mass lesion.

Causes of Right Atrial Enlargement

- Tricuspid insufficiency (e.g., from endocardiosis or congenital dysplasia)
- Cardiomyopathy
- Right atrial neoplasia (e.g., hemangiosarcoma)
- Aneurysmal dilation of the right auricular appendage (incidental)

Differential Diagnosis

- Cranial mediastinal mass
- Heart base neoplasia (most common in brachycephalic breeds)
- Tracheobronchial lymphadenopathy
- Superimposition of an enlarged aortic arch or main pulmonary artery
- Right cranial or middle lobar pulmonary alveolar consolidation or mass lesion

RIGHT VENTRICULAR ENLARGEMENT

Radiographic Signs

- Lateral projection (see Fig. 2.15)
  - Disproportionate enlargement of the cranial portion of the cardiac silhouette when empirically divided into its right and left chambers (Fig. 2.17)
  - Increased sternal contact of cranial cardiac margin (Fig. 2.18)
  - Elevation of the cardiac apex from the sternum in cases of concentric hypertrophy of the right ventricle (e.g., pulmonary hypertension, heartworm disease) (Fig. 2.19)
  - Overall rounding of the entire cardiac silhouette and increased cardiac width
  - Dorsal elevation of the caudal vena cava
- DV projection (see Figs. 2.16, 2.18, and 2.19)
  - Cardiac silhouette enlargement at the 6 to 11 o'clock position
  - Given the preferential enlargement and rounding of the right cardiac margin, the left margin

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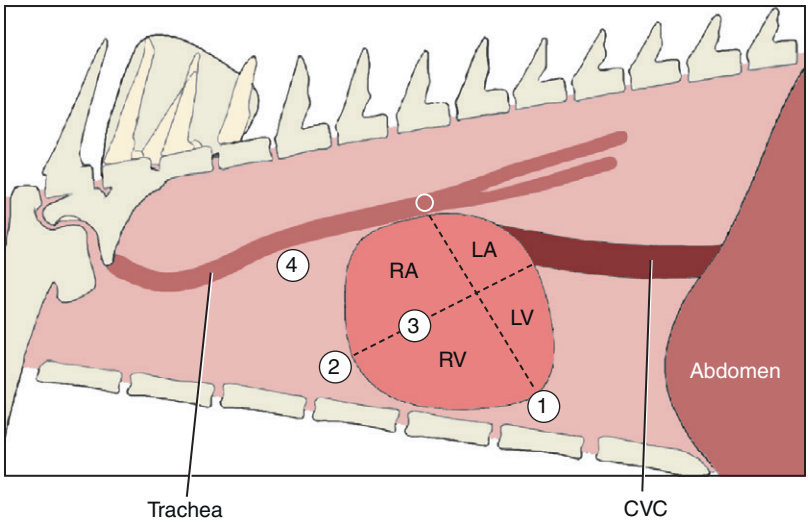


Fig. 2.17 Schematic representation of radiographic signs associated with right-sided heart enlargement in the lateral projection. (*1*) Dorsal lifting of apex from sternum. (*2*) Increased sternal contact of cranial cardiac margin. (*3*) Disproportionate enlargement of the cranial portion of the cardiac silhouette when empirically divided into its right and left chambers. (*4*) Elevation of the trachea as it courses dorsally over the right atrium. *CVC*, Caudal vena cava; *LA*, left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

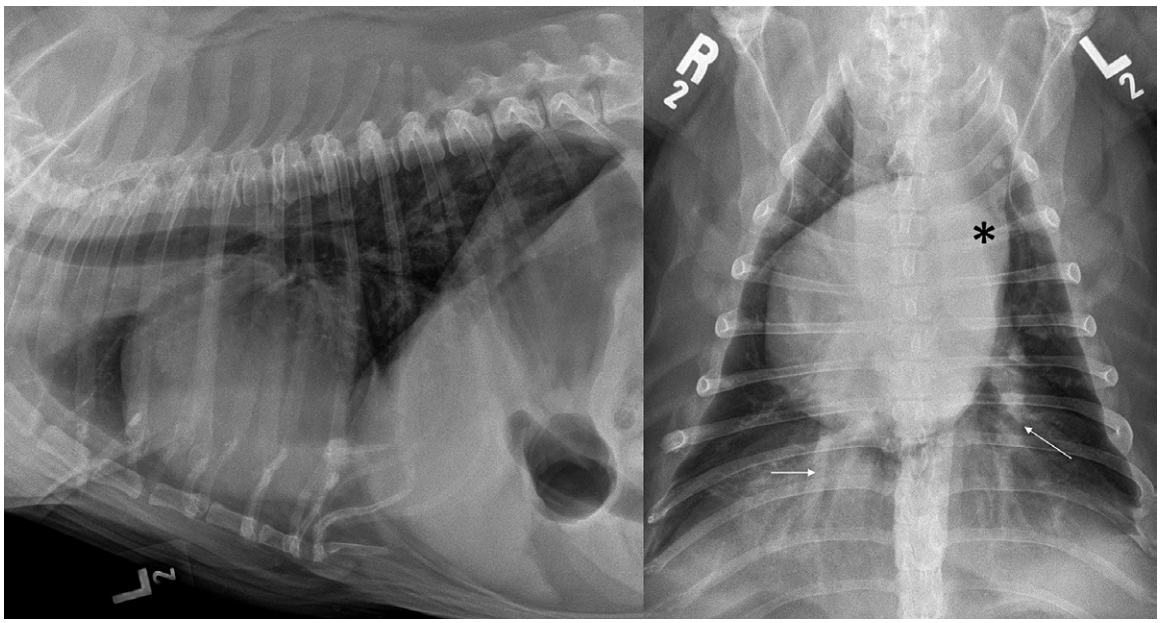


Fig. 2.18 Radiographs of the thorax in a dog with pulmonary hypertension. There is marked right-sided cardiomegaly, with increased cardiosternal contact on the lateral view and a reversed D appearance of the heart on the dorsoventral (DV) view. Marked enlargement of the main pulmonary artery is seen on the DV view as a bulge at 1–2 o'clock (*asterisk*). The caudal lobar pulmonary arteries (*arrows*) are significantly larger than their corresponding lobar pulmonary veins.

in comparison assumes a more straightened conformation, resulting in a "reverse-D" shape of the cardiac silhouette
- Shift of cardiac apex to the left

Causes of Right Ventricular Enlargement

- Tricuspid endocardiosis and insufficiency
- Cardiomyopathy
- Cor pulmonale/pulmonary hypertension (see Fig. 2.18)
- Dirofilariasis (see Fig. 2.19)
- Congenital heart disease: pulmonic stenosis, ventricular septal defects, tetralogy of Fallot, tricuspid valve dysplasia

LEFT ATRIAL ENLARGEMENT

RADIOGRAPHIC SIGNS

- Lateral projection (see Fig. 2.15; Fig. 2.20 and Fig. 2.21)
  - Dorsal elevation of the caudal portion of the trachea and carina

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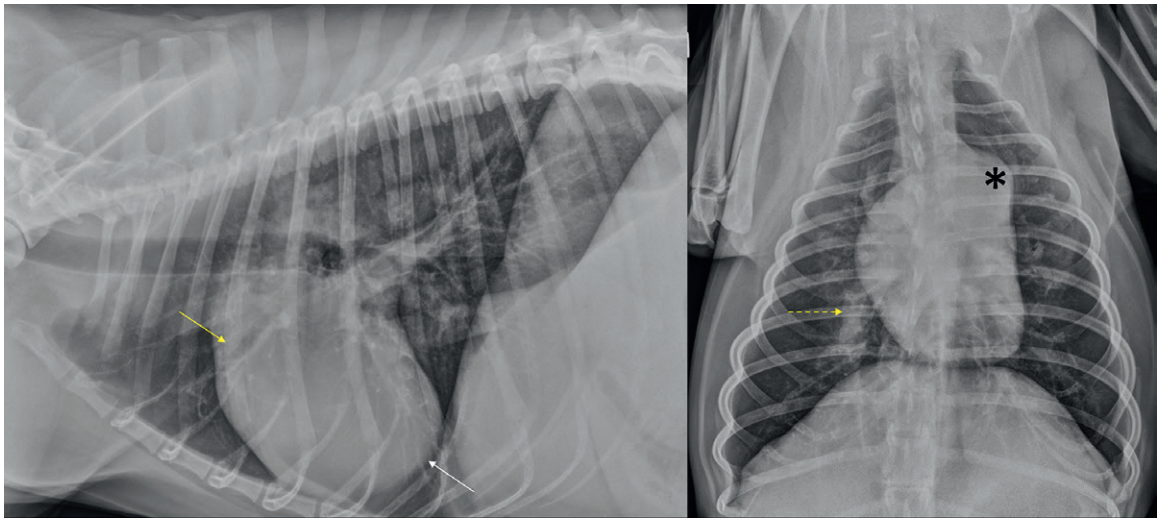


Fig. 2.19 Radiographs of the thorax in a dog with dirofilariasis. The right side of the heart is predominant with a reversed D appearance on the dorsoventral (DV) view. The apex of the heart is dorsally displaced on the lateral view, which may indicate concentric hypertrophy of the right ventricle. The main pulmonary artery is enlarged, causing a bulge at 1–2 o'clock on the DV view (*asterisk*). On the lateral view, the cranial lobar pulmonary artery (*solid arrow*) is enlarged relative to the corresponding vein and on the DV view the right caudal lobar pulmonary artery (*dashed arrow*) is also enlarged.

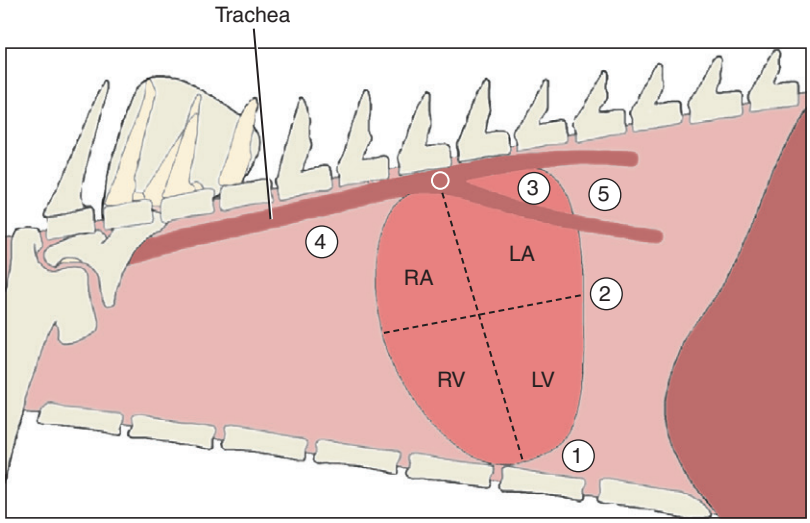


Fig. 2.20 Schematic representation of radiographic signs associated with left-sided heart enlargement in the lateral projection. (*1*) Rounding and widening of the cardiac apex conformation. (*2*) Straightening and increased vertical axis of the caudal cardiac margin. (*3*) Left atrial enlargement with characteristic right-angular caudodorsal margin conformation. (*4*) Dorsal elevation of the intrathoracic portion of the trachea, carina, and mainstem bronchi. The angle between the thoracic spine axis and the trachea is diminished to the point of becoming parallel. (*5*) Separation of normally superimposed caudal mainstem bronchi. Left side is more dorsal in position than the right side. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

- Disproportionate dorsal elevation of the mainstem bronchi (the two will no longer be superimposed; the left bronchus will appear more dorsal than the right bronchus); in some dogs, narrowing of one or both principal bronchi may also be seen as a result of preexisting chondromalacia exacerbated by the mechanical compression by the subjacent dilated left atrium
- Enlargement and straightening of the caudodorsal portion of the cardiac silhouette with almost a right-angle margin conformation (see Figs. 2.20 and 2.21); straightening of the caudal margin of the heart and loss of the caudal waist (determined by the atrioventricular junction)
- DV projection (see Figs. 2.16 and 2.21)
  - In the dog:
    - Enlargement of the atrial auricular appendage produces a noticeable focal "bulge" at the 2 to 3 o'clock position (see Figs. 2.13 and 2.16)
    - A double opacity sign over the caudal aspect of the cardiac silhouette between the caudal bronchi, caused by dilation of the atrial body; a radiographic edge effect ("Mach" line) may be seen around the enlarged left atrium caused

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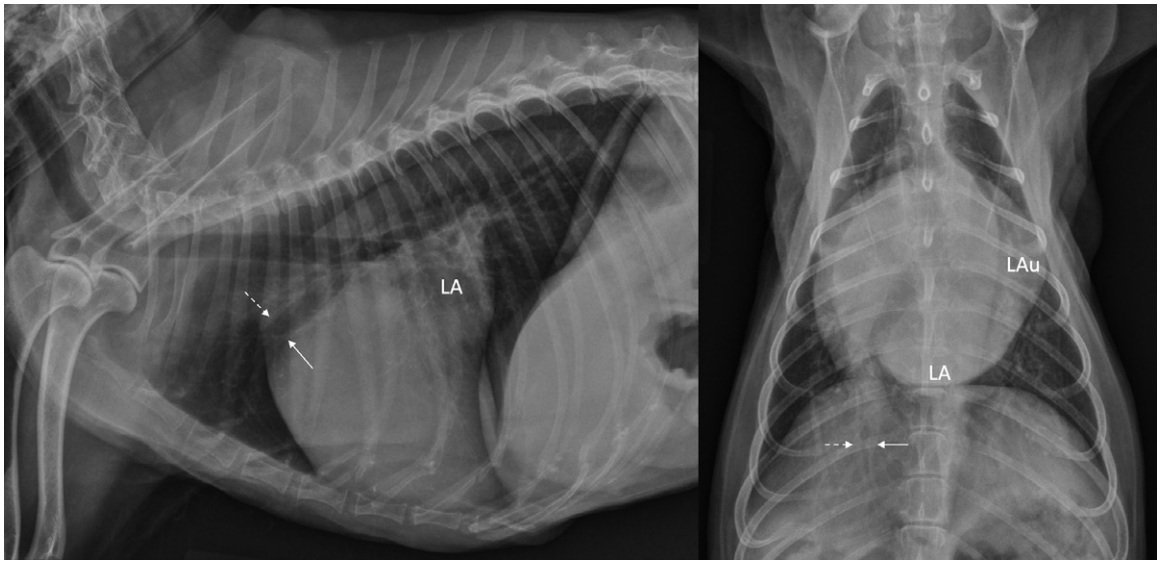


Fig. 2.21 Compensated mitral insufficiency in a dog with mitral endocardiosis; the left atrium (*LA*) is markedly enlarged, causing a focal bulge at 12–2 o'clock on the lateral projection and a double opacity sign superimposed to the heart and spine on the dorsoventral (DV) view. The left auricle (*LAu*) is dilated, causing a bulge at 2–3 o'clock on the DV projection. The pulmonary arteries (dashed arrows) and veins (solid arrows) are not congested, and the pulmonary parenchyma is normal, indicating that the condition is still well compensated.

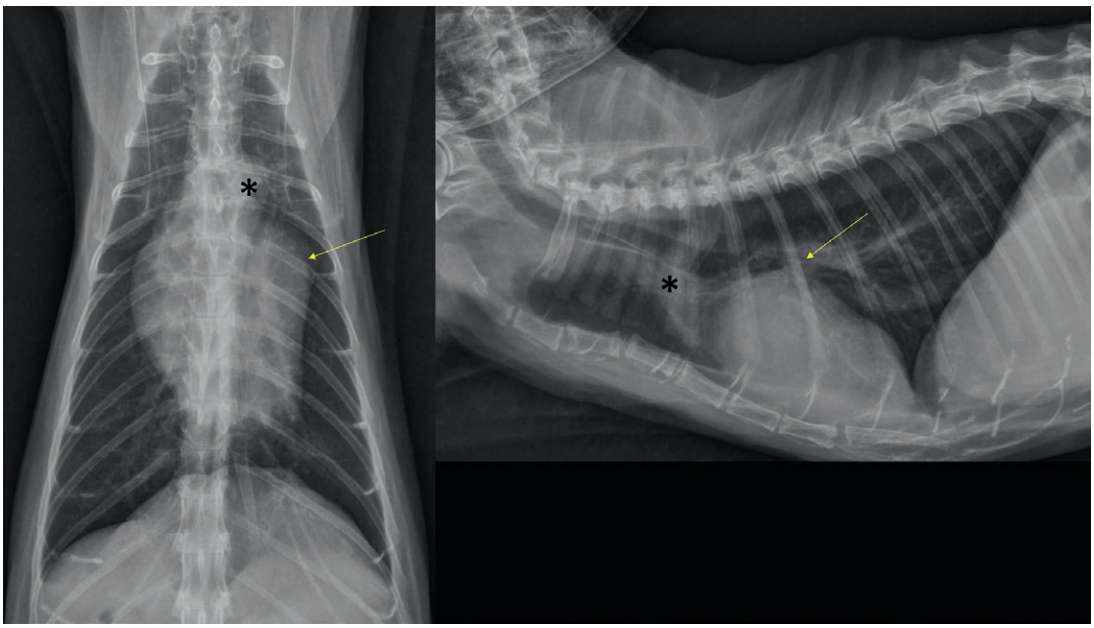


Fig. 2.22 Radiographs of the thorax in a cat with compensated hypertrophic cardiomyopathy. The left atrium (*arrows*) is enlarged, forming a bulge at 2–3 o'clock on the ventrodorsal (*VD*) view. The heart assumes a Valentine shape on the VD view. The aortic arch is cranially redundant which can be an age-related variation and can be accentuated with systemic hypertension.

by an acute change in soft tissue thickness (see Fig. 2.13)
    - Subjective splaying of the caudal mainstem bronchi resulting from abaxial displacement by the enlarged left atrium
- In the cat (Fig. 2.22):
    - The left atrium sits more along the left lateral margin of the heart and, when enlarged, causes a bulge of the cardiac margin at the 2 to 3 o'clock position rather than a double opacity sign seen in dogs
    - In cats with marked enlargement of the left atrium, the cardiac silhouette may assume a "Valentine shape" often misinterpreted as biatrial dilation

Causes of Left Atrial Enlargement

- Mitral endocardiosis and insufficiency (see Fig. 2.21)
- Cardiomyopathy (see Fig. 2.22)
- Congenital heart disease; mitral valve dysplasia, patent ductus arteriosus (PDA), ventricular septal defects, atrial septal defects

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Differential Diagnosis
- Hilar lymphadenopathy
- Pulmonary mass adjacent to hilus

LEFT VENTRICULAR ENLARGEMENT

Radiographic Signs
- Lateral projection (see Fig. 2.15)
  - Loss of the caudal waist
  - Caudal cardiac margin straighter and more vertical than normal
  - Dorsal elevation of the intrathoracic portion of the trachea, carina, and mainstem bronchi; the angle between the thoracic spine axis and trachea is diminished to the point of becoming parallel
  - Disproportionate enlargement of the caudal portion of the cardiac silhouette when empirically divided into its right and left cardiac chambers (see Fig. 2.20)
- DV projection (see Fig. 2.16)
  - Rounding and enlargement of the left ventricular margin
  - Rounding and broadening of the cardiac apex conformation
  - Shift of the cardiac apex to the right

Causes of Left Ventricular Enlargement
- Mitral endocardiosis and insufficiency
- Cardiomyopathy
- Congenital heart disease: PDA, aortic stenosis, ventricular septal defects
- High-output cardiac disease: fluid overload, chronic anemia, peripheral arteriovenous fistula, obesity, chronic renal disease, hyperthyroidism

ENLARGEMENT OF THE AORTIC ARCH AND AORTA

Radiographic Signs
- Lateral projection (see Fig. 2.15)
  - Widening of the dorsal aspect of the cardiac silhouette
  - Enlargement of the craniodorsal cardiac margin
  - In cases of enlargement of the cranial descending aorta, fusiform aneurysmal dilation of the aorta is seen dorsal to the heart over the carina region
- DV projection (see Fig. 2.16)
  - Widening and increased cranial extensions of the cardiac margin at the 11 to 1 o'clock position
  - In cases of enlargement of the cranial descending aorta, fusiform aneurysmal dilation of the aorta is seen to the left of the spine in the 1 to 3 o'clock position

Causes of Aortic Enlargement
- Aortic arch:
  - PDA: enlargement more abaxial (1 o'clock position)
  - Aortic stenosis with poststenotic enlargement of the aortic arch: enlargement more axial and cranial (11 o'clock position)
  - Systemic hypertension: formation of an "aortic knob": due to lateral bowing of the aortic arch to cranial descending aorta forming a curved opacity bulging to the left in the cranial cardiac region on DV/VD radiographs (Fig. 2.23and Fig. 2.24)
- Cranial descending aorta: aortic coarctation (very rare)

Differential Diagnosis
- Normal variation in some dogs
- Can be a normal variant in older cats with "lazy" heart conformation
- Cranial mediastinal mass
- Thymic enlargement (e.g., thymoma) or normal thymus ("sail sign") in puppies
- Cranial mediastinal fat in obese dogs particularly brachycephalic

ENLARGEMENT OF THE PULMONARY ARTERY SEGMENT

Radiographic Signs
- Lateral projection (see Fig. 2.15)
  - Protrusion of the craniodorsal cardiac border
- DV projection (see Figs. 2.16, 2.18, and 2.19)
  - Lateral bulge of the cardiac margin at the 1 to 2 o'clock position

Causes of Pulmonary Artery Segment Enlargement
- Dirofilariasis
- Pulmonary thrombosis and thromboembolism
- Cor pulmonale
- Congenital disease: pulmonic stenosis (poststenotic dilation), PDA, septal defects both ventricular and atrial, with left-to-right shunting

Differential Diagnosis
- Previous dirofilariasis infection and treatment
- Rotational (oblique) positional artifact (usually on VD projection) most commonly seen with deep-chested conformation dogs

EVALUATION OF THE PULMONARY CIRCULATION

UNDERCIRCULATION: DECREASED SIZE OF PULMONARY ARTERIES AND VEINS

Radiographic Signs
- Lung field more radiolucent than normal because of lack of pulmonary vascular volume (Fig. 2.25)
- Hyperinflation caused by hypoxemia or ventilation/perfusion mismatch
- Pulmonary arteries smaller than normal; may be smaller than corresponding pulmonary veins

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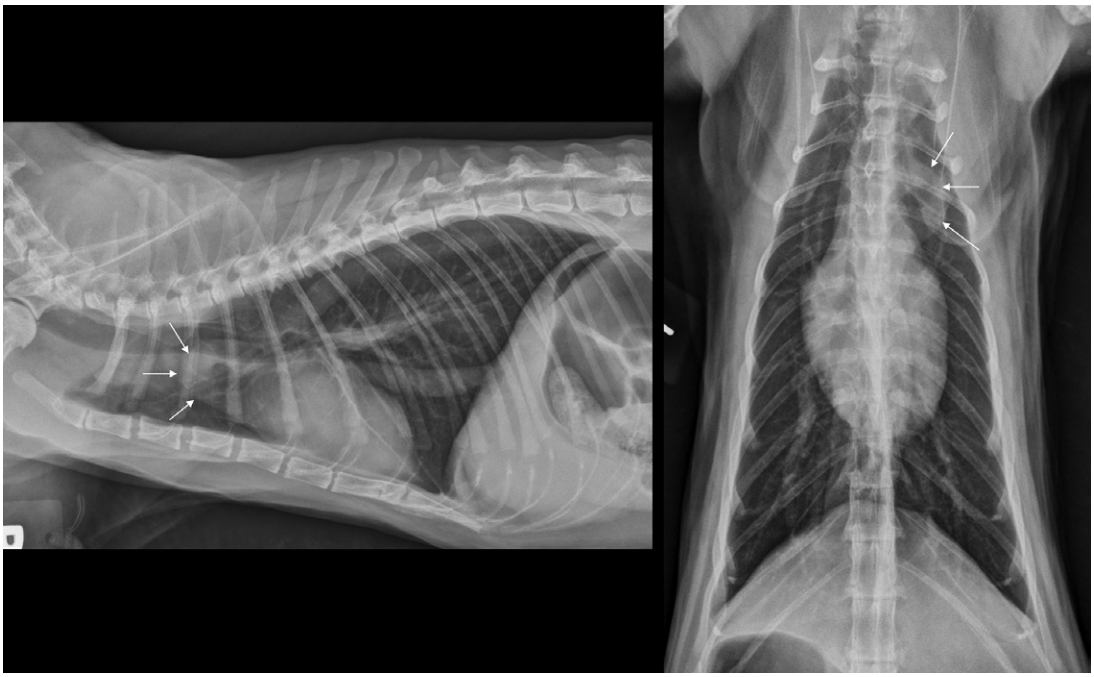


Fig. 2.23 Radiographs of the thorax in an old cat with severe systemic hypertension. The aortic arch is very prominent and redundant, forming a curved opacity between the spine and thoracic wall on the ventrodorsal view (*arrows*). The descending thoracic aorta on the lateral view has an undulating path. The cardiac silhouette has an increased sternal contact, which is a common age-related variation in geriatric cats.

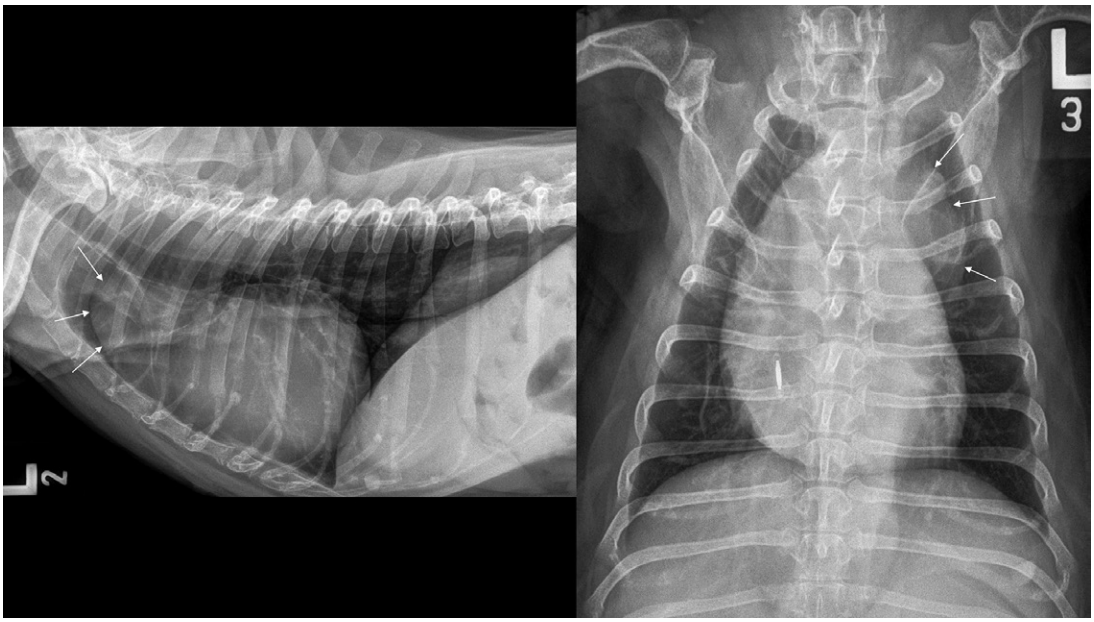


Fig. 2.24 Radiographs of the thorax in a dog with severe systemic hypertension. The aortic arch is very prominent and redundant, forming a curved opacity between the spine and thoracic wall on the ventrodorsal view (*arrows*).

Causes of Pulmonary Undercirculation

- Congenital disease: pulmonic stenosis, tetralogy of Fallot, reverse PDA (right-to-left shunting)

Differential Diagnosis

- Emphysema, chronic obstructive pulmonary disease
- Hyperinflation (e.g., feline asthma)
- Pneumothorax
- Overexposure
- Pulmonary thromboembolism
- Hypovolemia, shock (the heart and caudal vena cava will also be smaller than normal) (see Fig. 2.25)
- Hypoadrenocorticism (Addison disease); the heart may also be smaller than normal

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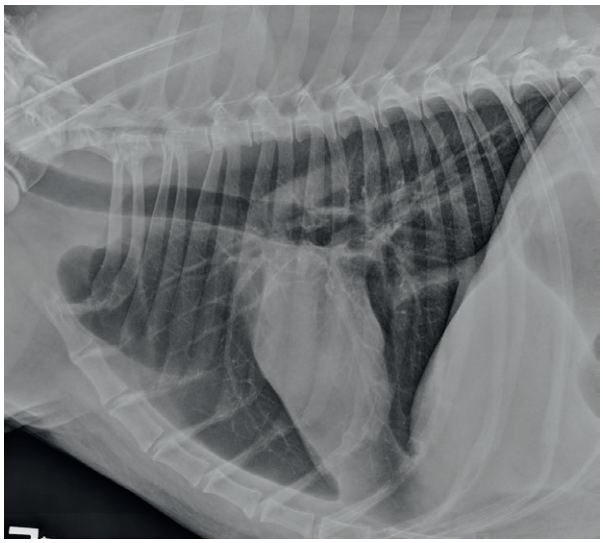


Fig. 2.25 Microcardia and pulmonary undercirculation in a dog in septic shock due to peritonitis. The cardiac silhouette is very small and the pulmonary vessels and caudal vena cava are small as well.

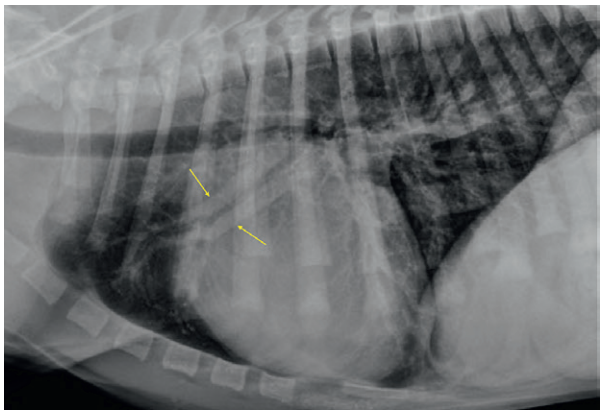


Fig. 2.26 Lateral radiograph of the thorax in a dog with a patent ductus arteriosus. There is enlargement of both lobar pulmonary arteries and veins (*arrows*) consistent with left-to-right shunt.

OVERCIRCULATION: ENLARGED PULMONARY ARTERIES AND VEINS

Radiographic Signs

- Both the pulmonary arteries and the veins are enlarged (pulmonary vascular pattern) (Fig. 2.26)
- Pulmonary thoracic opacity is increased because of larger vascular volume, causing a diffuse interstitial pattern

Causes of Pulmonary Overcirculation

- PDA (Fig. 2.27)
- Left-to-right cardiac shunts (ventricular and atrial septal defects)
- Congestive left-sided heart failure and concurrent pulmonary hypertension
- Fluid overload

Differential Diagnosis

- Underexposure
- Expiratory phase of respiration

PREDOMINANT ENLARGEMENT OF PULMONARY ARTERIES

Radiographic Signs

- Selective enlargement of pulmonary lobar arteries relative to the corresponding veins
- Arteries larger than the anatomic landmarks (rib width) they can be compared to (see the section Radiographic Anatomy)
- Can be generalized or segmental depending on underlying etiology

Causes of Pulmonary Arterial Enlargement

- Pulmonary hypertension (generalized; see Fig. 2.18)
- Dirofilariasis (generalized or segmental; see Fig. 2.19)
- Pulmonary thromboembolism (segmental)

PREDOMINANT ENLARGEMENT OF PULMONARY VEINS

Radiographic Signs

- Selective enlargement of pulmonary lobar veins relative to the corresponding arteries
- Veins larger than the anatomic landmarks (rib width) they can be compared to (see the section Radiographic Anatomy)

Causes of Pulmonary Venous Enlargement

- Left-sided congestive heart failure (Fig. 2.28)

RADIOGRAPHIC DIAGNOSIS OF HEART FAILURE

The radiographic diagnosis of heart failure is dependent on recognition of imbalances in the blood and fluid distribution within the body. This circulatory imbalance is the result of diminished cardiac output into the pulmonary or systemic vascular systems, or reduced acceptance of blood by the failing ventricle (hypertrophy), or both. Depending on which side of the heart is most severely affected, blood is shifted from the systemic to the pulmonary circulation (left-sided heart failure) or from the pulmonary to the systemic circulation (right-sided heart failure).

RIGHT-SIDED HEART FAILURE

Pathophysiology

- In right-sided heart failure, an inadequate right ventricular output into the pulmonary arteries exists concurrently with a reduced acceptance of blood from the systemic veins (cranial and caudal vena cavae). The blood volume and pressure in the splanchnic and systemic veins are elevated. The venous congestion causes hepatomegaly.

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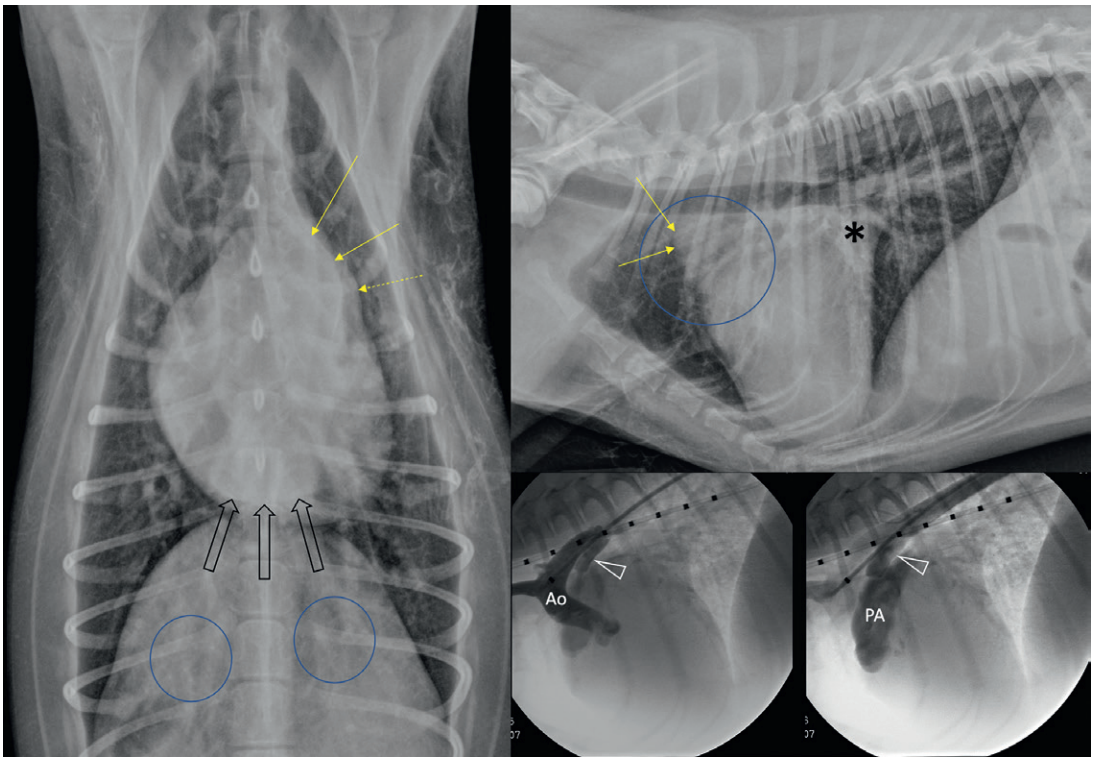


Fig. 2.27 Patent ductus arteriosus (PDA) in a dog. On the radiographs, the aorta is enlarged (*solid arrows*); on the dorsoventral (DV) view, the main pulmonary artery is enlarged causing a bulge at 1–2 o'clock (*dashed arrow*). There is enlargement of the lobar pulmonary arteries and veins (*circles*) consistent with left-to-right shunting and overcirculation. The left atrium is enlarged, causing a bulge at 12–2 o'clock on the lateral view (*asterisk*) and a double opacity sign on the DV view (*opened arrows*). On the selective angiocardiography (bottom-right corner), opacification of the aorta (*Ao*) then PDA (*arrowhead*) and then pulmonary artery (*PA*) is visible.

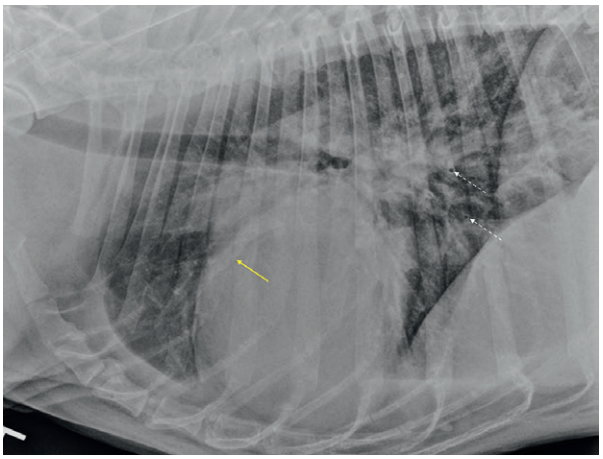


Fig. 2.28 Left-sided congestive heart failure in a dog. Note the enlarged cranial lobar vein (*solid arrow*) compared to the corresponding artery. There is a severe perihilar and caudodorsal interstitial and alveolar pulmonary pattern consistent with pulmonary edema; a peribronchial pattern (*dashed arrows*) is also present. The left heart is enlarged, as indicated by the upright appearance of the caudal cardiac margin and the bulge in the area of the left atrium with loss of the caudal waist.

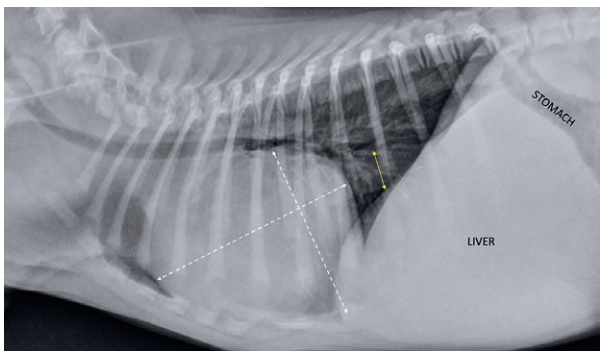


Fig. 2.29 Severe right-sided cardiomegaly in a young dog with congenital tricuspid valve dysplasia. Note the predominance of the right heart when empirically dividing the heart into right and left heart chambers. There is severe increased cardiosternal contact and loss of the cranial cardiac waist, indicative of right ventricular and atrial enlargement. The caudal vena cava (*double-headed arrow*) is congested; the liver is enlarged, causing caudodorsal displacement of the gastric axis.

- With further progression of right-sided heart failure, a progression of systemic hypertension leads to increased amounts of fluid, solutes, and protein escaping from the capillary beds of the major organs. The lymphatic circulation is overtaxed, and fluid exudes into the serosal cavities, producing ascites, pleural, and even pericardial effusions.

Radiographic Signs

- Right-sided cardiomegaly (see Figs. 2.15–2.17 and Fig. 2.29). Patients with concentric cardiac hypertrophy (e.g., pulmonic stenosis), early cardiomyopathy, or acute arrhythmias often may not have

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dramatic radiographic cardiomegaly. Thus subtle cardiac silhouette changes in both the DV and the lateral projections may be considered significant with supportive clinical evidence of cardiac disease.
- Enlargement of the caudal vena cava.
- Hepatomegaly: rounded liver margin, which extends caudal to costal arch; displacement of stomach caudally (lateral view) and to the left (VD view) (see Fig. 2.29).
- Ascites: abdominal distention; diffuse loss of intraabdominal detail.
- Pleural effusion:
  - Generalized increase in thoracic opacity.
  - Visualization of interlobar pleural fissures (see Figs. 2.4 and 2.5A).
  - Obliteration of cardiac silhouette definition (best demonstrated on the DV projection) (see Fig. 2.5B).
  - Separation of pulmonary visceral pleural margin away from thoracic wall (see Figs. 2.4 and 2.5).

Causes of Pleural Effusion Secondary to Right-Sided Heart Failure
- Decompensated tricuspid insufficiency (e.g., from endocardiosis or congenital dysplasia)
- Decompensated pulmonic stenosis, tetralogy of Fallot
- Dirofilariasis (caval syndrome)
- Pericardial effusion with tamponade
- Restrictive pericarditis

Differential Diagnosis
- Pleuritis
- Chylothorax
- Hemothorax
- Pyothorax
- Hypoproteinemia
- Neoplasia (pleural, mediastinal, cardiac, pulmonary, primary, or metastatic)

LEFT-SIDED HEART FAILURE

Pathophysiology
- In left-sided heart failure, inadequate left ventricular output into the aorta occurs, and a diminished acceptance of blood from the pulmonary veins entering the left atrium results. This causes pulmonary venous congestion and leakage of fluid into the pulmonary interstitium, with progression to flooding of the alveoli.
- Clinically, this evolves as a progression of physiologic events: pulmonary venous congestion, interstitial pulmonary edema, alveolar edema, and lung consolidation.

Radiographic Signs
- Left-sided cardiomegaly (see Figs. 2.20 and 2.28). Patients with concentric cardiac hypertrophy (e.g., aortic stenosis, hypertrophic cardiomyopathy), dilated cardiomyopathy (large- and giant-breed dogs), or acute arrhythmias often may not have dramatic radiographic cardiomegaly. Thus when noncardiac changes (e.g., pulmonary venous congestion, pulmonary edema) are suggestive of left-sided heart failure, subtle cardiac silhouette radiographic changes may be significant.
- Pulmonary venous congestion:
  - Engorgement and distention of the pulmonary veins, especially in the hilar area as they enter the left atrium. On the DV view, these are identified as the more axial of the caudal vasculature (see Fig. 2.12).
  - The diameter of the pulmonary veins is greater than that of their corresponding pulmonary arteries (best seen with cranial lobar vessels on lateral projections or with caudal lobar vessels on DV projections, though the latter are often obscured by cardiogenic edema which has typically a caudodorsal distribution) (see Figs. 2.10 and 2.28).
- Interstitial edema:
  - Diffuse increased radiopacity of the lung fields owing to a hazy interstitial opacity is apparent.
  - The margins of the pulmonary veins and arteries are indistinct, owing to perivascular edema. As the lung parenchyma surrounding the pulmonary vasculature fills with fluid, the normal pulmonary radiographic contrast between gas (air-filled lung) and soft tissue (blood-filled vessels) is lost. Thus the pulmonary vasculature becomes indistinct and begins to disappear in the surrounding fluid-filled lung parenchyma.
  - In some patients, fluid accumulates around major bronchi, producing prominent peribronchial markings forming donut-like opacities.
- Alveolar edema:
  - Radiographic signs:
    - Fluid enters the alveolar air spaces and peripheral bronchioles, causing a coalescent fluffy alveolar infiltrate. Air bronchograms (black tubes in a white radiopaque background) and air alveolograms (small, interspersed lucencies representing groups of air-filled alveoli surrounded by fluid-filled lung parenchyma) are present. In the cat, cardiogenic alveolar edema can appear as well-margined, "cloud-like" areas of increased pulmonary radiopacity.
    - The margins of the pulmonary vessels are usually completely obscured (see Figs. 2.3 and 2.28). The alveolar infiltrate is of greatest opacity in the perihilar area, fading peripherally. In the dog, alveolar edema can be asymmetrical, with the right lung fields more severely affected than the left (best seen on the DV projection).
  - Differential diagnosis for cardiogenic pulmonary edema includes:
    - Neurogenic: electrocution, head trauma, postseizure, encephalitis, brain neoplasm

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- Hyperdynamic (excessive negative intrathoracic pressures): choking, strangulation, upper airway obstruction
- Fluid overload: overhydration
- Toxicity
- Systemic shock
- Acute respiratory distress syndrome
- Drowning

- Pleural effusion:
  - In the dog, this can occur only in progressive or severe forms of left-sided heart failure; this usually indicates early concurrent left- and right-sided heart failure.
  - In the cat, pleural effusion is common with left-sided heart failure alone; this can be differentiated from right-sided heart failure by the absence of accompanying hepatomegaly and ascites (see Fig. 2.3C).

RADIOGRAPHIC DIAGNOSIS OF PERICARDIAL EFFUSION

- Generalized enlargement of cardiac silhouette in a "basketball" conformation, with elimination of all normal cardiac margin contours on all views
- Increased sternal contact of the cranial margin and convex bulging of the caudal margin without the angular conformation and straightening characteristic for left atrial and ventricular enlargements (Fig. 2.30)
- Dorsal elevation and enlargement of the caudal vena cava
- Dorsal elevation of the trachea (similar to left-sided enlargement)
- Hepatomegaly, peritoneal and pleural effusion secondary to cardiac tamponade (see Figs. 2.4 and 2.5)

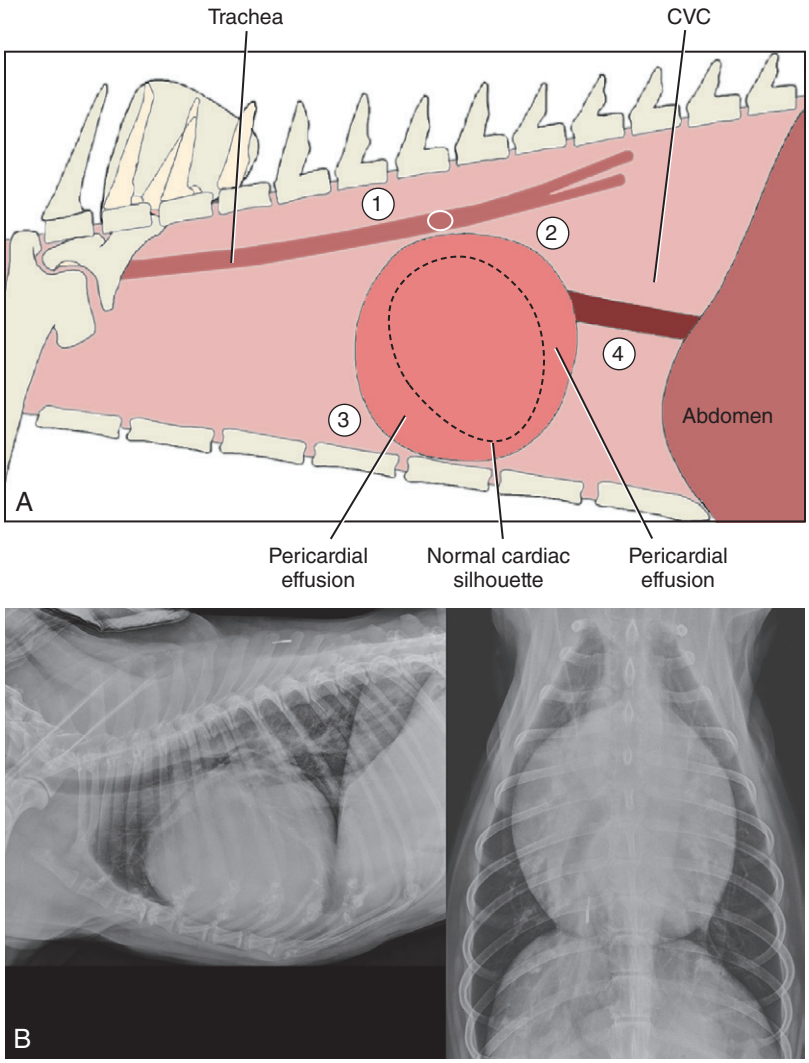


Fig. 2.30 **(A)** Schematic representation of radiographic signs associated with pericardial effusion. (*1*) Dorsal elevation of the intrathoracic portion of the trachea, carina, and mainstem bronchi. The angle between the thoracic spine axis and the trachea is diminished to the point of becoming parallel. (*2*) Convex enlargement of the caudodorsal cardiac margin without a "right-angle" conformation characteristic for left atrial enlargement. (*3*) Increased sternal contact of cranial margin. (*4*) Dorsal elevation and enlargement of the caudal vena cava (*CVC*). The cardiac silhouette takes on a smoothly contoured circular conformation with obliteration of normal cardiac contour. **(B)** Lateral and DV radiographs of a dog with pericardial effusion.

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Table 2.1 Summary of Radiographic Signs of Congenital and Acquired Cardiac Disease

LESION	RA	RV	LA	LV	AORTA	MPAS	PAB	PV	CVC	FAILURE/ SIDE	FAILURE/ TYPE
Congenital Defects											
Patent ductus arteriosus (LtoR)	N	In	In	In	In	In	In	In	N/In	Left	Volume
Pulmonic stenosis	In	In	N/De	N/De	N	In	N/De	N/De	In	Right	Pressure
Aortic stenosis	N	N/In	N/In	In	In	N	N	N/In	N	Left	Pressure
Ventricular septal defect	N	In	In	In	N	N/In	In	In	N/In	Left	Volume
Tetralogy of Fallot	N/In	In	N/De	N/De	N	De/N/In	De	De	N	Right	Pressure
Atrial septal defect	In	In	N/In	N	N	N/In	N/In	N/In	N/In	Left	Volume
Acquired Heart Disease											
Mitral insufficiency	N	N/In	In	In	N	N	N	In	N	Left	Fluid
Tricuspid insufficiency	In	In	N	N	N	N	N	N	In	Right	Fluid
Aortic insufficiency	N	N	In	In	N/In	N	N	In	N	Left	Fluid
Hypertrophic cardiomyopathy	In	In	In	In	N	N	N/In	N/In	N/In	Left > Right	Myocardial
Dilated cardiomyopathy	In	In	In	In	N	N	N/In	N/In	N/In	Right > Left	Myocardial
Pericardial	In	In	In	In	N	N	N/De	N/De	In	Right	Tamponade

CVC, Caudal vena cava; *De*, smaller or decreased; *In*, enlarged or increased; *LA*, left atrium; *LV*, left ventricle; *MPAs*, main pulmonary artery segment; *N*, normal; *PAb*, pulmonary artery branches; *PV*, pulmonary vein; *RA*, right atrium; *RV*, right ventricle.

SUMMARY OF RADIOGRAPHIC SIGNS

The clinician must be armed with both potential radiographic parameters and a clinically derived differential diagnostic list for cardiac disease before the radiographic image can begin to provide useful information. Table 2.1 summarizes the radiographic signs associated with congenital and acquired heart diseases. Awareness of noncardiac and artifactual conditions that can present with the same radiographic signs is also paramount to a correct diagnosis.

DIGITAL RADIOGRAPHY AND TELERADIOLOGY

- Digital radiography (DR) has become the standard of care in veterinary diagnostic imaging and film-screen technology has become obsolete. There are many advantages to DR beyond the excellent image quality (Fig. 2.31), which include the following:
  - No lost films
  - No film degradation over time
  - The ability to view images on any networked computer at a clinic or at home
  - The ability to easily send images to specialists for consultation
  - There are financial savings over time, including the following:
    - No cost for radiology disposables (film, chemicals).
    - No expense for processor maintenance, film jackets, and storage space.
    - Significant reduction in the number of retakes because of a much wider exposure latitude of DR systems resulting in little to no need to retake images caused by under- or overexposure.
- There are several types of digital acquisition systems:
  - Flat panel technology (also known as digital radiography [DR] or direct digital radiography

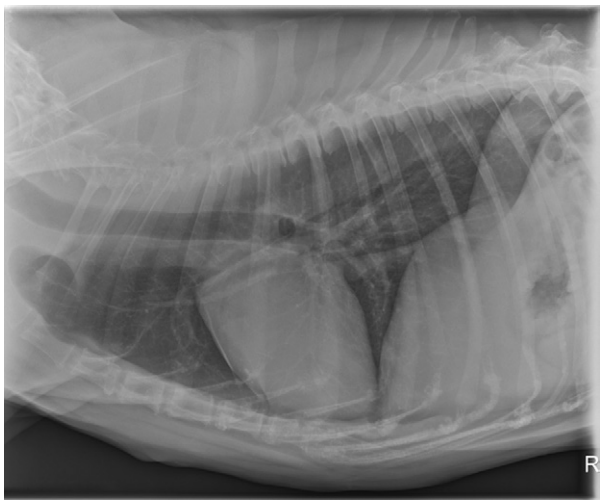


Fig. 2.31 Right lateral view of the thorax taken with a flat panel detector system (digital radiography). Note that all structures (e.g., bone, lung, pulmonary vessels, spine) are visible in the same image. There are no areas of overexposure or underexposure.

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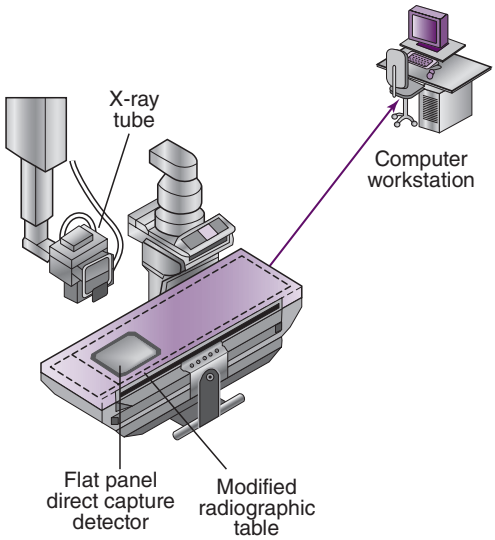


Fig. 2.32 A flat panel detector (digital radiography plate) is mounted out of sight under the x-ray tabletop in the location of the Bucky tray. The plate converts x-ray photon energy to an electrical pulse, which is then interfaced with an acquisition station computer. (From Fauber TL: *Radiographic imaging & exposure*, ed 4, St. Louis, 2013, Mosby.)

[DDR] [Fig. 2.32]) is the most expensive form of DR; however, this technology results in the highest quality image. These systems consist of a DR plate that is physically mounted in the area of the Bucky tray under the x-ray tabletop. The plate is then electronically interfaced to both the x-ray machine and a dedicated computer (acquisition station). DR systems are extremely forgiving as far as technique (kilovolt [peak] and milliampere settings) is concerned (Fig. 2.33). This in turn simplifies a typical technique chart to essentially three or four settings (small, medium, large, and extra-large) whether you are imaging bone, thorax, or abdomen. Another advantage of DR systems includes extremely quick image time (3–8 seconds before the image is seen on a computer monitor), which allows the user to either save or delete the image immediately if it is not satisfactory (e.g., rotated, crooked).

- Computed radiography (CR) systems use imaging plates enclosed in what resembles a traditional analog x-ray cassette. The major difference is that the intensifying screen and film within the cassette is replaced by a flexible phosphor plate that can store a latent image. These storage phosphor plates retain a portion of the x-ray energy from the exposure as a latent image, which is extracted ("read out") by a CR laser reader. In general, the image quality from a CR system is very high (similar to that of flat panel technology); however, CR is typically less forgiving as to technique (compared with flat panel), which may necessitate a more complicated technique chart.

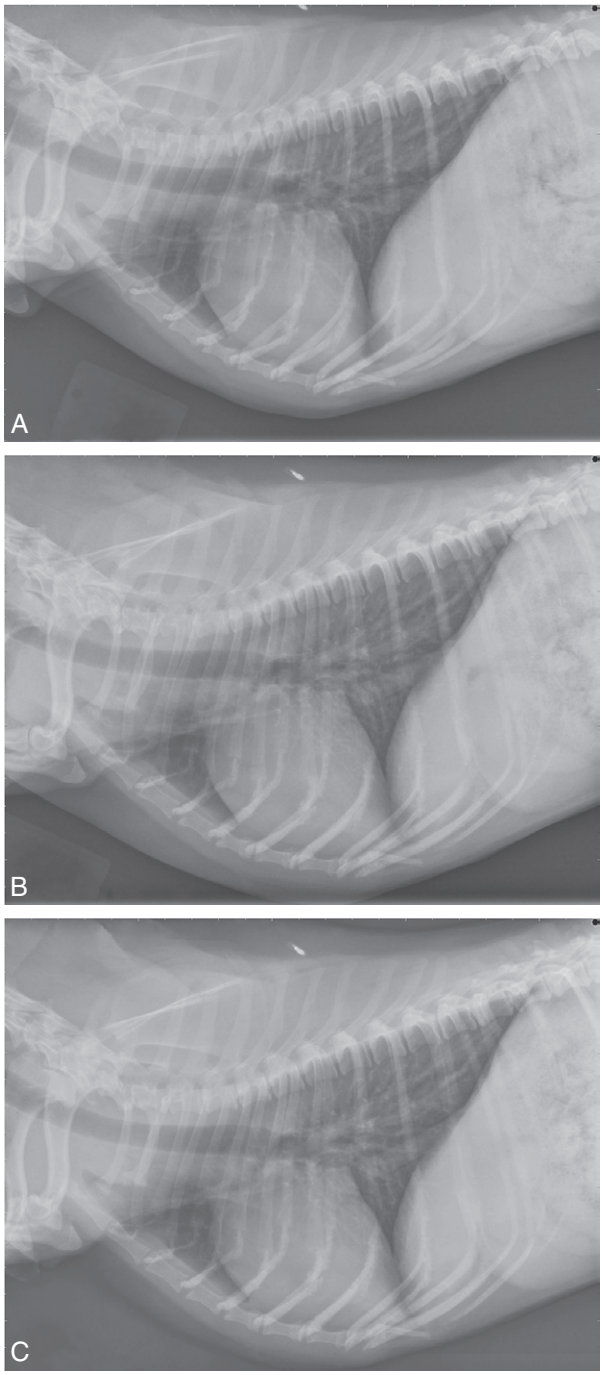


Fig. 2.33 Technique independence. These three exposures were made with different milliampere-second (mAs) settings and identical kilovolt potential (KvP) of 90. **(A)** 1.8 mAs, **(B)** 2.5 mAs, and **(C)** 5.0 mAs. Note that all three exposures appear similar and are diagnostic. The computer software corrects for underexposure or overexposure automatically. This capability decreases the number of retakes and increases productivity. On the other hand, if image (A) is magnified, it will appear much grainier than the other images.

The image time for most CR systems ranges from about 55 to 90 seconds, due to the necessity of laser reader processing. However, CR systems are less expensive than DR systems.

- Charge-coupled device (CCD) systems consist of a phosphor storage plate mounted under the

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x-ray tabletop that is in turn interfaced with a small light-sensitive chip (CCD chip) similar to that found in digital cameras and video cameras. These CCD chips are commonly about 2 cm in size and may have thousands of individual light-sensitive elements on them. Because of the small size of the chips, the aerial image (e.g., 14 × 17 in.) must be minified down to the size of the CCD chip. This is usually accomplished with a series of mirrors and lenses, which unfortunately results in a significant loss (90%) of the photon data. This loss of data can often make the resultant image appear "grainy" on the computer monitor, which is accentuated if the image is electronically magnified. On the other hand, CCD systems have fast image time (similar to flat panel systems) and are less expensive than DR systems. Because of the nature of these systems, they are usually sold as a complete system that includes the x-ray machine and table, and cannot be retrofitted into an existing X-ray installation.

- Dedicated x-ray film scanners and digital cameras are not forms of DR. Both methods only reproduce the traditional hard copy radiograph and, in general, do a poor job of image reproduction. Even expensive multi-megapixel digital cameras now available do a poor job of converting an analog x-ray image into a digital format without the loss of significant grayscale data (dynamic range). Because of this, the use of film scanners and digital cameras is unacceptable as a means of sending images for consultation (teleradiology).

Key Point

- Flat panel digital radiography is extremely fast, is technique independent, has the highest image quality, but is the most expensive method.
- Computed radiography is slower, is somewhere between conventional film-screen and flat panel technology as to reliance on x-ray technique, has high image quality, and is moderately expensive.
- Charged-coupled device systems are fast, are similar to CR systems regarding technical factors, have the poorest image quality, and are the least expensive.
- Film scanners and digital cameras are not forms of DR and are unacceptable as a means of sending images for consultation.

- Teleradiology (telemedicine) offers the practitioner quick access to board-certified specialists for case consultation. Once the radiographic images are in a digital format, they can be sent to any specialist for review via the Internet.
- Usually telemedicine companies or individual specialists provide the necessary software that allows the veterinarian to upload digital images to the company's or specialist's server.
- Submission of images through standard e-mail can be simple, but it is absolutely unacceptable. Because of the very large image size of digital radiographs (a 14 × 17 radiograph of the thorax can be 14 megabytes of information), these images must be compressed or saved in a "lossy" format (such as JPG) before they are e-mailed, thus making the transmitted image of poor quality.
- DICOM transfer is the *only* acceptable method to share digital radiographs for the purpose of interpretation by a specialist. It is a proven and worldwide recognized method of transmitting high-quality, lossless, digital radiographs (and other medical images such as ultrasounds) from one place to another. DICOM images are embedded with specific information regarding patient data, as well as the type of system that the images were acquired on, and this information cannot be altered. Also, DICOM allows transmission of images without the need for proprietary software that is vendor specific. For example, if you have a GE Healthcare brand ultrasound machine, the images can be read by any radiologist with a DICOM viewer (which can be found for free), and they do not need to have specific GE Healthcare software to view the images. DICOM allows the practicing veterinarian to send lossless, high-quality images with embedded patient data directly to any radiologist of their choosing.

ACKNOWLEDGMENT

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Chapter 3

Electrocardiography

Larry Patrick Tilley and Francis W.K. Smith, Jr.

Electrocardiography in clinical practice is the recording at the body surface of electrical fields generated by the heart. Specific waveforms represent stages of myocardial depolarization and repolarization. The electrocardiogram (ECG) is a basic and valuable diagnostic test in veterinary medicine and is relatively easy to acquire. It is the initial test of choice in the diagnosis of cardiac arrhythmias and may also yield information regarding chamber dilation and hypertrophy.

INDICATIONS AND ROLE OF THE ELECTROCARDIOGRAM IN CLINICAL PRACTICE

DOCUMENTATION OF CARDIAC ARRHYTHMIAS

- An ECG should be recorded when an arrhythmia is detected during physical examination. This may include bradycardia, tachycardia, or irregularity in rhythm that is not secondary to respiratory sinus arrhythmia.
- Animals with a history of syncope or episodic weakness may have cardiac arrhythmias, and an ECG is indicated in these cases. Arrhythmias in such cases may be transient—a normal ECG result does not rule out transient arrhythmias. In some cases, long-term electrocardiographic monitoring (Holter monitor or cardiac event recorder) is warranted.
- Arrhythmias often accompany significant heart disease and may significantly affect the clinical status of the patient. An ECG should be recorded in animals with significant heart disease.
- The ECG is also used to monitor efficacy of antiarrhythmic therapy and to determine whether arrhythmias may have developed secondary to cardiac medications (e.g., digoxin).
- Significant arrhythmias may also occur in animals with systemic disease, including those diseases associated with electrolyte abnormalities (hyperkalemia, hyponatremia, hypercalcemia, and hypocalcemia), neoplasia (particularly splenic neoplasia), gastric dilatation-volvulus, and sepsis.

ASSESSMENT OF CHAMBER ENLARGEMENT PATTERNS

- Changes in waveforms may provide indirect evidence of cardiac chamber enlargement. The ECG result may be normal, however, in cases with chamber enlargement. Right ventricular hypertrophy most consistently results in waveform changes.
- As heart disease progresses, waveform changes may indicate progressive chamber enlargement.
- Thoracic radiography and, ideally, echocardiography should be performed for definitive assessment of chamber enlargement.

MISCELLANEOUS INDICATIONS FOR ELECTROCARDIOGRAPHY

- The ECG may provide evidence of pericardial effusion (electrical alternans, low-amplitude complexes).
- Electrocardiographic abnormalities are often present with hypothyroidism and hyperthyroidism.
- A pronounced sinus arrhythmia may be present in animals with elevated vagal tone (often seen with diseases affecting the respiratory tract, CNS, and gastrointestinal tract).

PRINCIPLES OF ELECTROCARDIOGRAPHY

PLACEMENT OF SURFACE ELECTRODES IN A DESIGNATED FASHION TO OBTAIN STANDARD ELECTROCARDIOGRAPHIC LEADS

- A lead consists of the electrical activity measured between a positive electrode and a negative electrode.
- Electrical impulses with a net direction toward the positive electrode will generate a positive waveform or deflection. Electrical impulses with a net direction away from the positive electrode will generate a negative waveform or deflection. Electrical impulses with a net direction perpendicular to the positive electrode will not generate a waveform or deflection (isoelectric).
- Standard electrocardiographic lead systems are used to create several angles of assessment. A single lead would provide information on only one dimension of the current (e.g., left compared with right). Two leads would allow two-dimensional information (e.g., left compared with right and cranial compared with caudal). As many as 12 leads may be acquired simultaneously.

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STANDARD LEAD SYSTEMS

- The standard leads are I, II, III, aVR, aVL, and aVF (Fig. 3.1 and Box 3.1). Placement of electrodes to generate each lead is illustrated in Fig. 3.2.
- Leads I, II, and III are bipolar limb leads. These are termed *bipolar* because the ECG is recorded from two specific electrodes.
- Leads aVR, aVL, and aVF are augmented unipolar leads. For these leads to be generated, two electrodes are electrically connected (as a negative electrode) and compared with the single electrode (positive).
- Precordial chest leads are obtained with an exploring unipolar positive electrode at specific locations on the chest. These leads may provide additional information or supportive evidence of cardiac chamber enlargement. They are also useful in the evaluation of the P wave when limb leads are equivocal.
- The base-apex lead is often used in equine electrocardiography and may also be used in small-animal practice for rhythm assessment. A positive electrode is placed on the left side of the chest, over the heart, and the negative electrode is placed in the area of the right shoulder or neck.

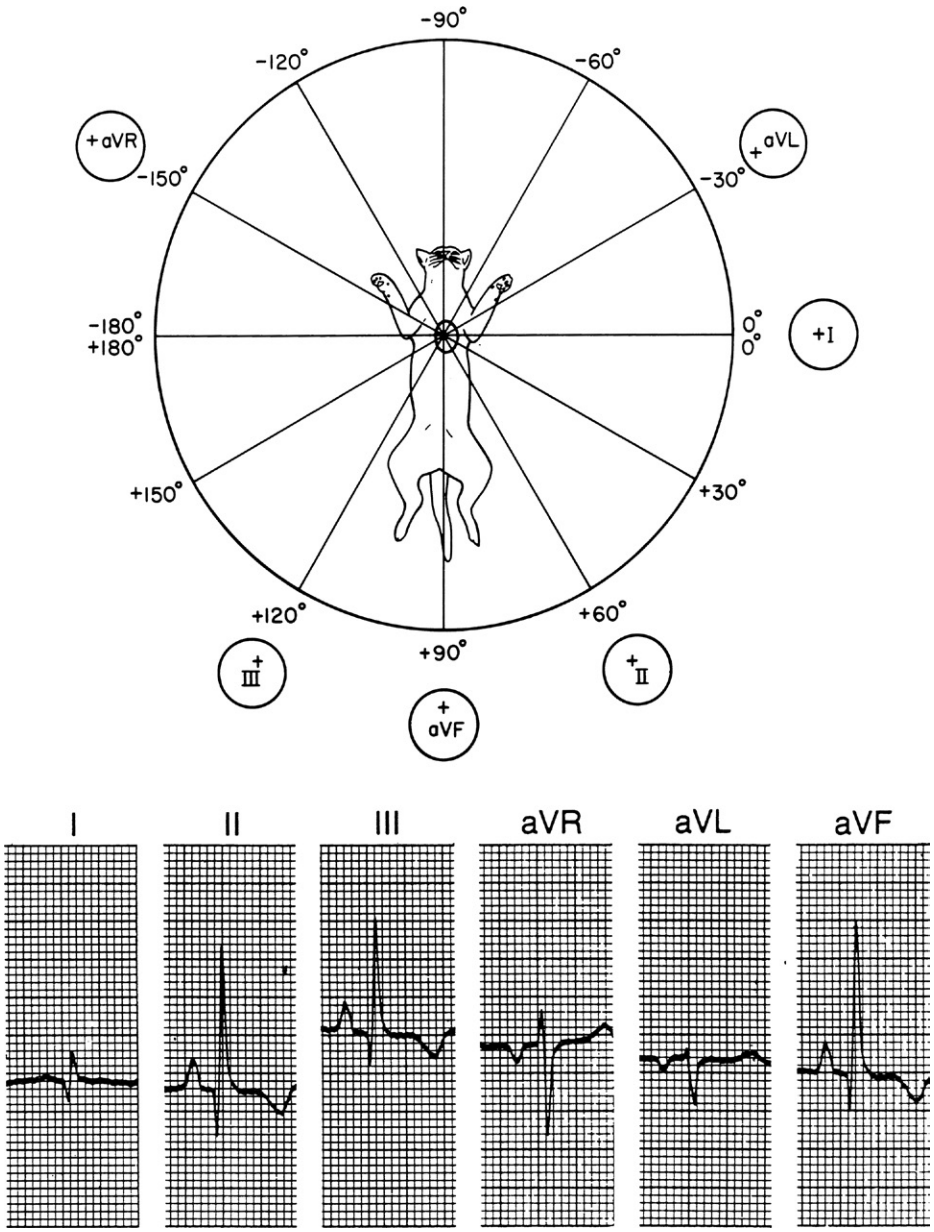


Fig. 3.1 The limb leads (I, II, III, aVR, aVL, aVF) surround the heart in the frontal plane as shown in the top part of the figure (feline). The circled limb lead names indicate the direction of electrical activity if the QRS is positive in that lead. The mean electrical axis in this canine electrocardiogram (*bottom part of the figure*) is +90. Lead I is isoelectric. The lead perpendicular to lead I is aVF (see axis chart on top). Lead aVF is positive, making the axis +90. If lead aVF had been negative, the axis would have been −90. (From Tilley LP: *Essentials of canine and feline electrocardiography: interpretation and treatment*, ed 3, Malvern, 1992, Lea & Febiger.)

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Box 3.1 Lead Systems Used in Canine and Feline Electrocardiography

BIPOLAR LIMB LEADS

I: Right thoracic limb (–) compared with left thoracic limb (+)

II: Right thoracic limb (–) compared with left pelvic limb (+)

III: Left thoracic limb (–) compared with left pelvic limb (+)

AUGMENTED UNIPOLAR LIMB LEADS

aVR: Right thoracic limb (+) compared with average voltage of left thoracic limb and left pelvic limb (–)

aVL: Left thoracic limb (+) compared with average voltage of right thoracic limb and left pelvic limb (–)

aVF: Left pelvic limb (+) compared with average voltage of right thoracic limb and left thoracic limb (–)

UNIPOLAR PRECORDIAL CHEST LEADS AND EXPLORING ELECTRODE

CV_5RL (rV_2): Right fifth intercostal space near the sternum

CV_6LL (V_2): Left sixth intercostal space near the sternum

CV_6LU (V_4): Left sixth intercostal space near the costochondral junction

V_{10}: Over the dorsal process of the seventh thoracic vertebra

BASE-APEX BIPOLAR LEAD

Record in lead I position on ECG machine with leads placed as follows: LA electrode over the left sixth intercostal space at the costosternal junction; RA electrode over the spine of the right scapula near the vertebra.

- The esophageal ECG electrode lead for surgical monitoring is useful because recorded complexes are increased in size, providing increased accuracy for diagnosing an arrhythmia during surgery (Fig. 3.3).
- Handheld, wireless ECG and ECG real-time computer display represents new technology for recording ECGs (Figs. 3.4 and 3.5).

RECORDING THE ELECTROCARDIOGRAM

- The ECG should be recorded in an area as quiet and as free of distraction as possible. Noises from clinical activity and other animals may significantly affect rate and rhythm. Any use of electrically operated equipment, such as clippers, may cause interference and should be minimized during the ECG. In some cases, fluorescent lighting may result in electrical interference.
- The patient should ideally be placed in right lateral recumbency.
  - Electrocardiographic reference values were obtained from animals in right lateral recumbency.
  - Limbs should be held perpendicular to the body. Each pair of limbs should be held parallel, and limbs should not be allowed to contact one another.
  - The animal should be held as still as possible during the ECG. When possible, panting should be prevented.
  - When dyspnea or other factors prevent standard positioning, the ECG may be recorded while the animal is standing or, less ideally, sitting.
- Electrode placement
  - Alligator clips or adhesive electrodes may be used. For reduction of discomfort, the teeth of alligator clips should be blunted, and the spring should be relaxed.
  - Limb electrodes are placed either distal or proximal to the elbow (caudal surface) and over the stifle. Electrodes placed proximal to the elbow may increase respiratory artifact.
  - Each electrode should be wetted with 70% isopropyl alcohol to ensure electrical contact.
- Recording the ECG
  - Approximately three to four complete complexes should be recorded from each lead at 50 mm/s.
  - A lead II rhythm strip should then be recorded at 25 or 50 mm/s.

Key Point

The 1-mV standardization marker should be recorded at the onset of the ECG and any time the sensitivity is changed.

CARDIAC CONDUCTION AND GENESIS OF WAVEFORMS

- The function of the cardiac conduction system is to coordinate the contraction and relaxation of the four cardiac chambers (Figs. 3.6 and 3.7).
- For each cardiac cycle, the initial impulse originates in the sinoatrial (SA) node located in the wall of the right atrium near the entrance of the cranial vena cava. This impulse is rapidly propagated through the atrial myocardium, with a resulting depolarization of the atria. Depolarization of the atria results in the P wave and atrial contraction. The initial SA nodal impulse is small and does not produce an electrocardiographic change on the body's surface.
- Immediately after atrial depolarization, the impulse travels through the atrioventricular (AV) node, located near the base of the right atrium. Conduction is slow here, which allows atrial contraction to be completed before ventricular depolarization occurs. As the impulse travels through the AV node, there is no electrocardiographic activity on the body's surface—rather, the PR interval is generated.
- On leaving the AV node, conduction velocity increases significantly, and the impulse is rapidly spread through the bundle of His, bundle branches, and Purkinje system. This results in rapid and nearly simultaneous depolarization of the

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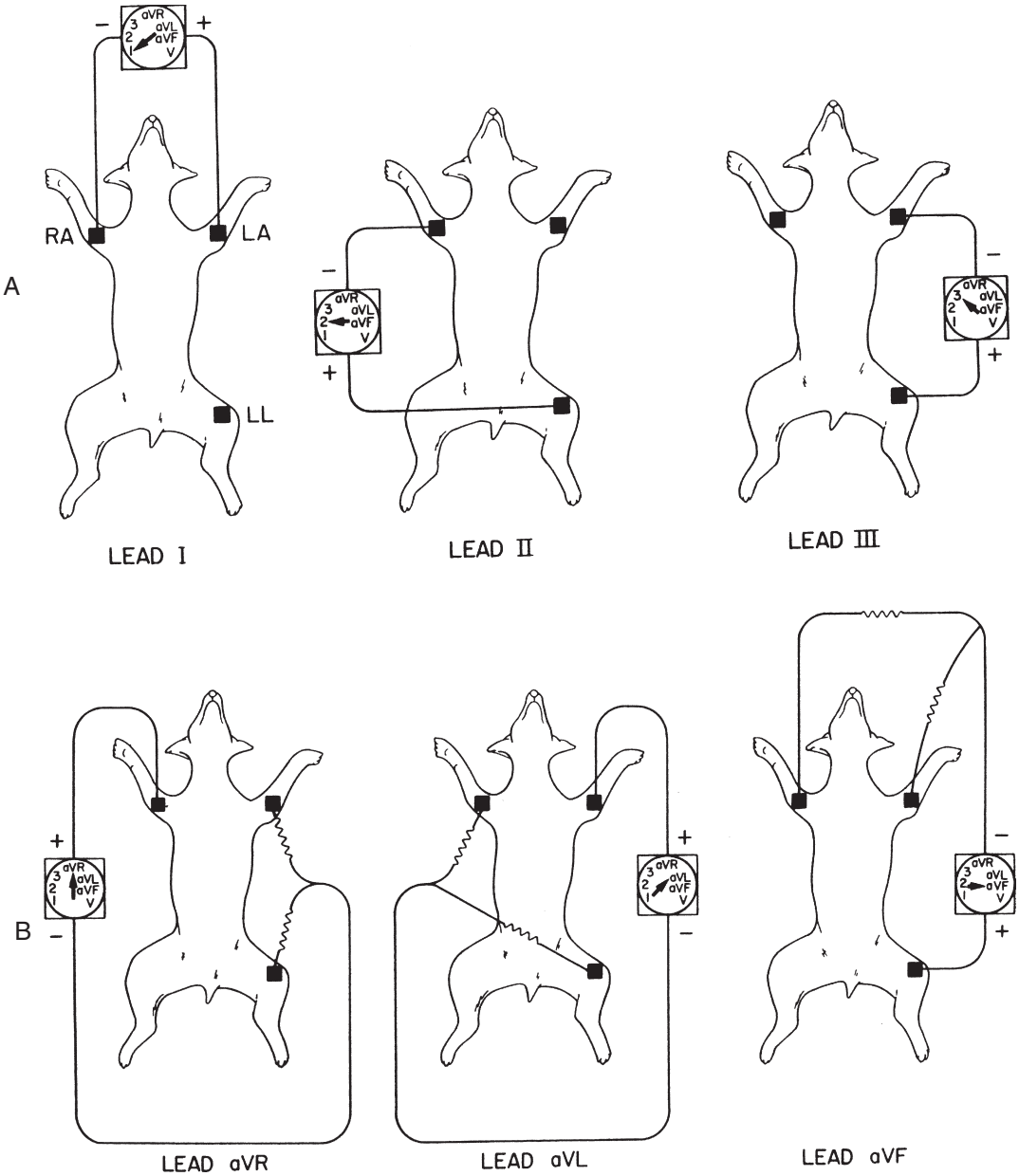


Fig. 3.2 **(A)** Three bipolar standard leads. By means of a switch incorporated in the instrument, the galvanometer can be connected across any pair of several electrodes. Each pair of electrodes is called a lead. The leads illustrated here are identified as I, II, and III. **(B)** Augmented unipolar limb leads aVR, aVL, and aVF. (From Tilley LP: *Essentials of canine and feline electrocardiography: interpretation and treatment*, ed 3, Malvern, 1992, Lea & Febiger.)

ventricles. Depolarization of the ventricles results in the prominent QRS complex and causes ventricular contraction.

- The Q wave represents initial depolarization of the interventricular septum and is defined as the first *negative* deflection following the P wave and occurring before the R wave. A Q wave may not be identified in all animals.
- The R wave represents depolarization of the ventricular myocardium from the endocardial surface to the epicardial surface. The R wave is the first *positive* deflection following the P wave and is usually the most prominent waveform.
- The S wave represents depolarization of the basal sections of the ventricular posterior wall and interventricular septum. The S wave is defined as the first negative deflection following the R wave in the QRS complex.
- Ventricular repolarization quickly follows ventricular depolarization and results in the T wave. The delay in repolarization results in the ST segment on the surface ECG.

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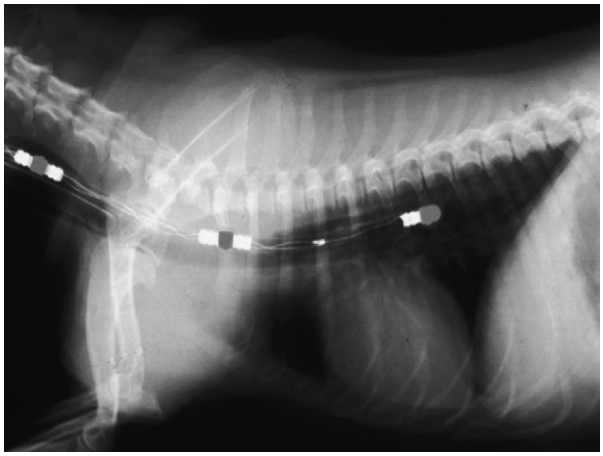


Fig. 3.3 Esophageal ECG electrode and temperature probe positioned for surgical monitoring. This technique is useful as complexes recorded are increased in size, providing increased accuracy for diagnosing an arrhythmia during surgery.



Fig. 3.4 Handheld, wireless electrocardiogram (ECG) and ECG real-time computer display. (Courtesy Vmed Technology, Redmond, Washington, www.vmedtech.com.)

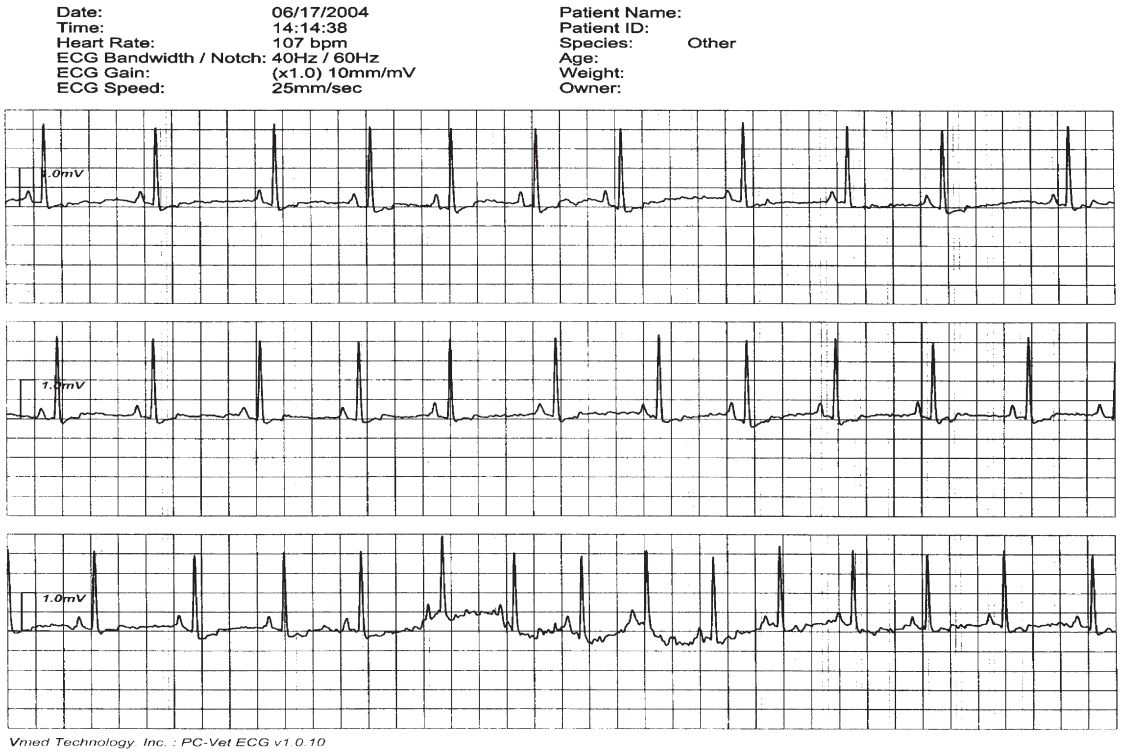


Fig. 3.5 Wireless electrocardiogram (ECG) printout report from laptop computer software system in Fig. 3.4. (Courtesy Vmed Technology, Redmond, Washington, www.vmedtech.com.)

EVALUATION OF THE ELECTROCARDIOGRAM

- The ECG should be evaluated from left to right.
- Areas of artifact should be identified and avoided in the evaluation.
- Calculate the heart rate (HR).
  - Determine the number of R waves (or R-R intervals) within 3 seconds and multiply by 20 to obtain beats per minute (beats/min) (for an ECG recorded at 50 mm/s, vertical timing marks above the gridlines occur every 1.5 seconds).
  - If the rhythm is regular, the HR may be derived by determining the number of small boxes in one R-R interval and dividing 3000 by that number (for paper speed of 25 mm/s, use 1500). The method is also useful for determining the rate of paroxysmal ventricular tachycardia and other arrhythmias lasting less than 3 seconds.

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- Obtain measurements for the waveforms and intervals (Fig. 3.8).
  - P-wave height and width
  - Duration of PR interval
  - Duration of QRS complex and height of R wave
  - Duration of QT segment
- Determine the approximate mean electrical axis (MEA).
  - The MEA refers to the direction of the net ventricular depolarization and refers solely to the QRS complex. If there is significant right ventricular hypertrophy, then the MEA will shift to the right. Because the left ventricle is normally the dominant ventricle, the normal MEA is to the left. A degree system is used: if the MEA is directly to the left, then it is said to be 0 degrees; if the MEA is directly downward, then it is 90 degrees; and if it is directly to the right, then it is 180 degrees. The MEA of the normal dog is 40 to 100 degrees. For the cat, the MEA is more variable, ranging from 0 to 160 degrees.
  - The MEA may be determined with the six standard leads and the Bailey axis system (see Fig. 3.1). If there is a lead with isoelectric QRS complexes, then the MEA equates to the lead on the Bailey axis perpendicular to the isoelectric lead.
  - The MEA may also be determined by plotting the net amplitude of a lead I QRS complex (horizontal

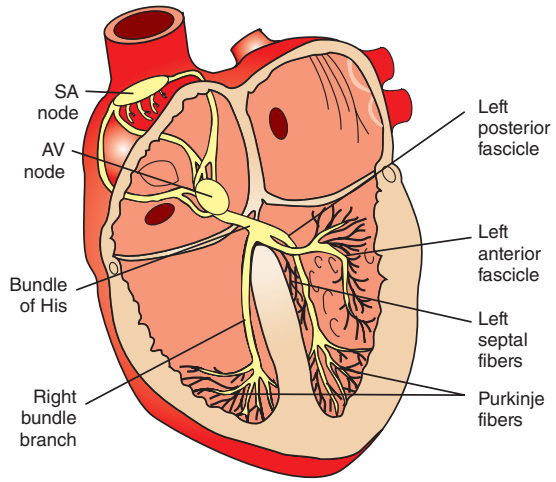


Fig. 3.6 Anatomy of the cardiac conduction system. *AV*, Atrioventricular; *SA*, sinoatrial. (Modified from Tilley LP: *Essentials of canine and feline electrocardiography: interpretation and treatment*, ed 2, Philadelphia, 1985, Lea & Febiger.)

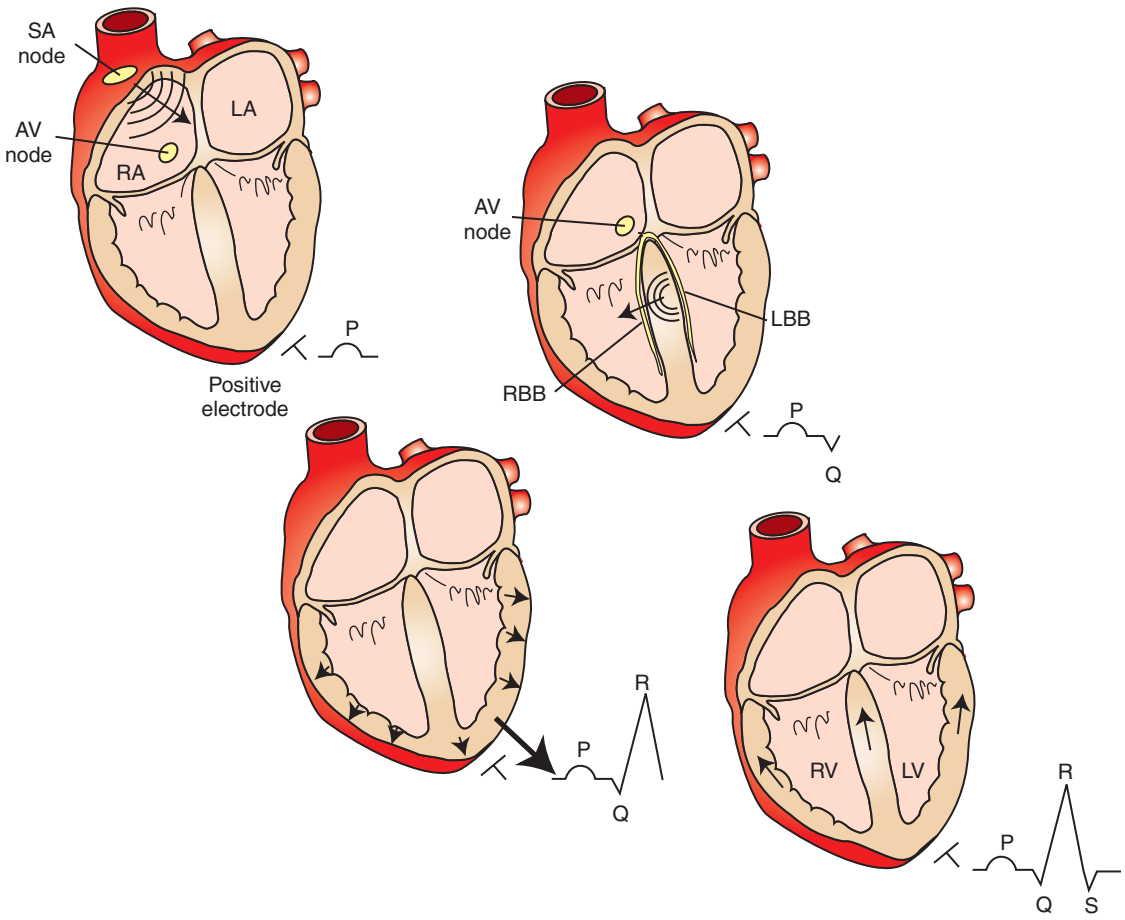


Fig. 3.7 Sequence of electrical impulse conduction and cardiac chamber activation as it relates to the ECG. *AV*, Atrioventricular; *LA*, left atrium; *LBB*, left bundle branch; *LV*, left ventricle; *RA*, right atrium; *RBB*, right bundle branch; *RV*, right ventricle; *SA*, sinoatrial. (Modified from Tilley LP: *Essentials of canine and feline electrocardiography: interpretation and treatment*, ed 3, Philadelphia, 1992, Lea & Febiger.)

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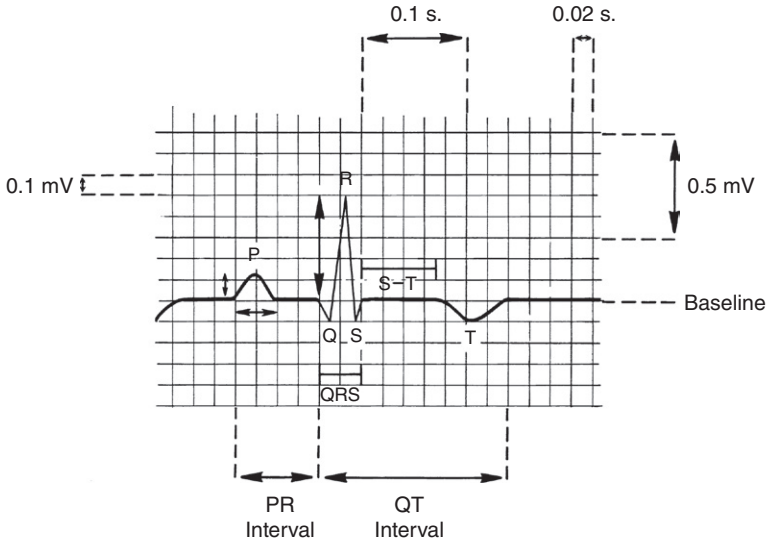


Fig. 3.8 Close-up of a normal feline lead II P-QRS-T complex with labels and intervals. Measurements for amplitude (millivolts) are indicated by positive (+) and negative (–) movement; time intervals (hundredths of a second) are indicated from left to right. Paper speed, 50 mm/s; sensitivity, 1 cm = 1 mV. (From Tilley LP: *Essentials of canine and feline electrocardiography: interpretation and treatment*, ed 2, Philadelphia, 1985, Lea & Febiger.)

axis) and the net amplitude of a lead aVF QRS (vertical axis). The intersection will provide the vector equal to the MEA (see Fig. 3.1).
  - The MEA may be approximated by inspecting leads I and aVF.
  - If the net direction of the lead I QRS is positive, then the MEA is to the left. If the net deflection of the lead I QRS is negative, then the MEA is to the right.
  - If the net direction of the lead aVF QRS is positive, then the MEA is downward or caudal. If the net deflection of the lead aVF QRS is negative, then the MEA is upward or cranial.
  - The approximate angle can be estimated by examining the relative amplitudes of leads I and aVF.
- Determine the rhythm.
  - Compare patient values with reference values (Table 3.1).

EVALUATION OF WAVEFORMS

P WAVE

- Atrial enlargement patterns (Fig. 3.9)
  - The P wave is generated by atrial depolarization. Atrial enlargement may result in an increase in width or height of the P waves recorded in lead II.
  - Enlargement of the right atrium may result in an increased P-wave height. This is referred to as P pulmonale. The height of the P wave should not exceed 0.4 mV (dog) or 0.2 mV (cat). Chronic pulmonary disease may result in P pulmonale in the absence of heart disease.
  - Enlargement of the left atrium may result in an increased P-wave width or duration. This is referred to as P mitrale. The duration of the P wave should not exceed 0.04 second (dog or cat). Left atrial enlargement may also result in notching of the P wave.
- Presence or absence of P waves
  - There is no minimum height or duration for the P wave. In some cases, P waves may be indistinct. In this situation, carefully evaluate all leads for P wave activity. If P waves cannot be discerned in any of the limb leads, evaluation of chest leads is recommended.
  - P waves may be absent in several arrhythmias, including atrial fibrillation and atrial standstill. P waves may be superimposed on other waveforms in ventricular tachycardia and supraventricular tachycardia (SVT).
  - Variation of P wave height is a normal finding in the dog and a manifestation of alterations in vagal tone.

PR Interval

- The PR interval reflects the slowed conduction through the AV node. The normal PR interval is 0.06 to 0.13 second for dogs and 0.05 to 0.09 second for cats.
- A significantly shortened PR interval may occur when an accessory pathway allows conduction to bypass the AV node.
- Prolongation of the PR interval represents first-degree AV block.
- Variation of the PR interval may occur with alterations in vagal tone or secondary to the presence of ectopic beats, causing dissociation of atrial and ventricular activity.

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Table 3.1 Normal Canine and Feline Electrocardiogram Values[a]

	CANINE	FELINE
HR	Puppy: 70,220 beats/min	120,240 beats/min
	Toy breeds: 70,180 beats/min	
	Standard: 70,160 beats/min	
	Giant breeds: 60,140 beats/min	
Rhythm	Sinus rhythm	Sinus rhythm
	Sinus arrhythmia	
	Wandering pacemaker	
P wave		
Height	Maximum: 0.4 mV	Maximum: 0.2 mV
Width	Maximum: 0.04 s (Giant breeds, 0.05 s)	Maximum: 0.04 s
PR interval	0.06–0.13 s	0.05–0.09 s
QRS		
Height	Large breeds: 3.0 mV maximum[b]	Maximum: 0.9 mV
	Small breeds: 2.5 mV maximum	
Width	Large breeds: 0.06 s maximum	Maximum: 0.04 s
	Small breeds: 0.05 s maximum	
ST segment		
Depression	No more than 0.2 mV	None
Elevation	No more than 0.15 mV	None
QT interval	0.15–0.25 s at normal HR	0.12–0.18 s at normal HR
T waves	May be positive, negative, or biphasic	Usually positive and <0.3 mV
	Amplitude range ± 0.05–1.0 mV in any lead	
	Not more than ¼ of R-wave amplitude	
Electrical axis	+40 to +100	0 ± 160
Chest leads		
CV_5RL (rV_2)	T positive; R < 3.0 mV	
CV_6LL (V_2)	S < 0.8 mV; R < 3.0 mV	R < 1.0 mV
CV_6LU (V_4)	S < 0.7 mV; R < 3.0 mV	R < 1.0 mV
V_{10}	QRS negative; T negative except in Chihuahua	T negative; R wave/Q wave <1

HR, Heart rate; *S*, second.
[a]Measurements are made in lead II unless otherwise stated.
[b]Not valid for thin, deep-chested dogs younger than 2 years.

QRS Complex

- The QRS complex is generated by ventricular depolarization (left ventricle, interventricular septum, and right ventricle). Ventricular enlargement may result in changes in the QRS complex.
- Left ventricular enlargement pattern
  - Increased amplitude of the R wave
    - Dog
      - Amplitude of R wave greater than 3.0 mV (2.5 mV in small-breed dogs) in lead II, lead aVF, and the precordial chest leads CV_6LU, CV_6LL, and CV_5RL
      - Amplitude of R wave greater than 1.5 mV in lead I
      - Sum of R-wave amplitudes in leads I and aVF greater than 4.0 mV
    - Cat
      - Amplitude of R wave greater than 0.9 mV in lead II
      - Amplitude of R wave greater than 1.0 mV in CV_6LU and CV_6LL
      - R wave/Q wave greater than 1.0 in lead V_{10}
  - QRS duration greater than 0.06 second (large dogs), 0.05 second (small dogs), or 0.04 second (cats)
  - ST slurring or coving
  - Shift in the MEA to the left (less than 40 degrees for dog, less than 0 degrees for cat)
- Right ventricular enlargement pattern (Fig. 3.10)
  - Increased amplitude of S waves
    - Dog
      - Presence of S waves in leads I, II, III, and aVF
      - S wave in lead I greater than 0.05 mV
      - S wave in lead II greater than 0.35 mV

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- S wave in lead CV_6LL greater than 0.8 mV
- S wave in lead CV_6LU greater than 0.7 mV
- Cat
  - Presence of S waves in leads I, II, III, and aVF
  - Prominent S waves in CV_6LU and CV_6LL
  - T-wave positive in lead V_{10} (except Chihuahua)
  - W-shaped QRS complex in V_{10} (dog)

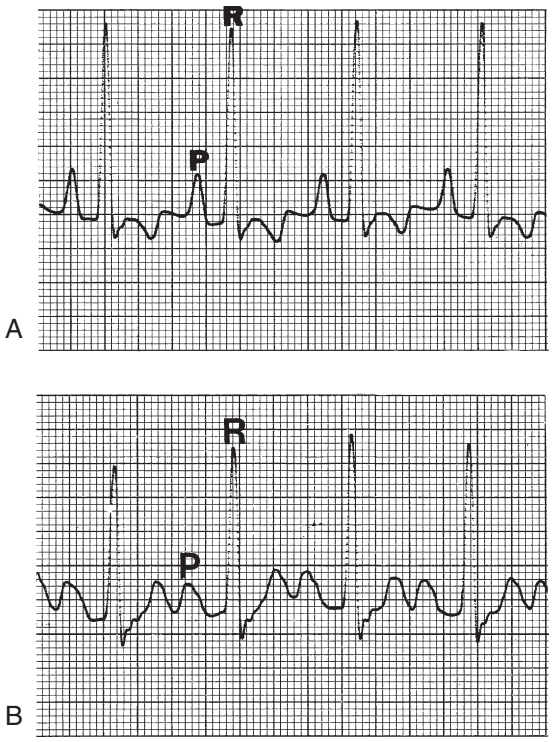


Fig. 3.9 **(A)** Biatrial enlargement in a small-breed dog with a collapsed trachea and compensated mitral regurgitation. The P wave is 0.5 mV tall (P pulmonale) and 0.05 s wide (P mitrale). **(B)** Left atrial enlargement in a geriatric, small-breed dog with chronic acquired valvular disease (mitral insufficiency). P waves are wide (0.075 s), notched, and equivocally tall. (From Fox PR, Sisson D, Moise NS, eds: *Textbook of canine and feline cardiology*, Philadelphia, 1999, Saunders.)

  - R/S ratio less than 0.87 in CV_6LU
  - Shift in the MEA to the right (more than 100 degrees for the dog or more than 160 degrees for the cat)
- Widening of the QRS complexes may occur with left ventricular enlargement, right or left bundle branch block (LBBB), and complexes of ventricular origin (ventricular premature complexes [VPCs] or ventricular escape complexes).
- Electrical alternans (Fig. 3.11A)
  - In electrical alternans, there is a pattern of regular variation in the amplitude of normal electrocardiographic complexes (excluding ventricular ectopy). This is usually manifested by an alteration in R-wave height, although variation in other waveforms may be seen. The amplitude may change significantly with every complex and alternate in height.
  - Electrical alternans is most often associated with pericardial effusion. A severe pleural effusion may cause electrical alternans.
  - SVT may result in an electrical alternans pattern.
- Low-amplitude QRS complexes
  - The minimum height for the normal R wave in the dog is 0.05 to 1.0 mV.
  - Low-amplitude R waves may occur when transmission of the cardiac electrical impulse to the skin is hindered. This may occur with pericardial effusion, pleural effusion, obesity, or subcutaneous edema. Pneumothorax and pulmonary edema may also decrease R-wave height.
  - Hypothyroidism may result in low-amplitude R waves (usually with accompanying slow rate).

ST Segment

- ST segment elevation (Fig. 3.11B)
  - Elevation of the ST segment greater than 0.15 mV in leads II, III, or aVF is abnormal in the dog. Any ST segment elevation in the cat is abnormal.
  - ST segment elevation may be caused by myocardial hypoxia, transmural myocardial infarction,

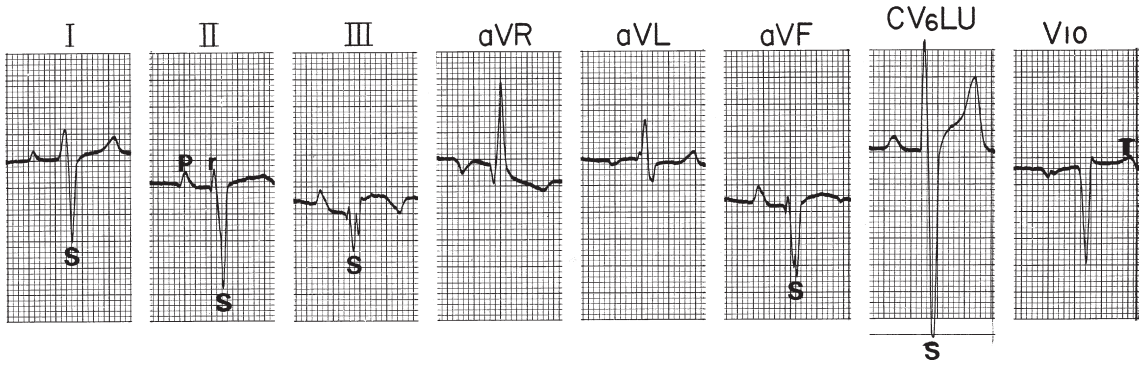


Fig. 3.10 Severe right ventricular enlargement in a dog with pulmonic stenosis. There is a right-axis deviation of approximately 110 degrees. Note the deep S waves in leads I, II, III, aVF, and CV_6LU. The T wave is positive in V_{10}. (From Tilley LP: *Essentials of canine and feline electrocardiography: interpretation and treatment*, ed 3, Philadelphia, 1992, Lea & Febiger.)

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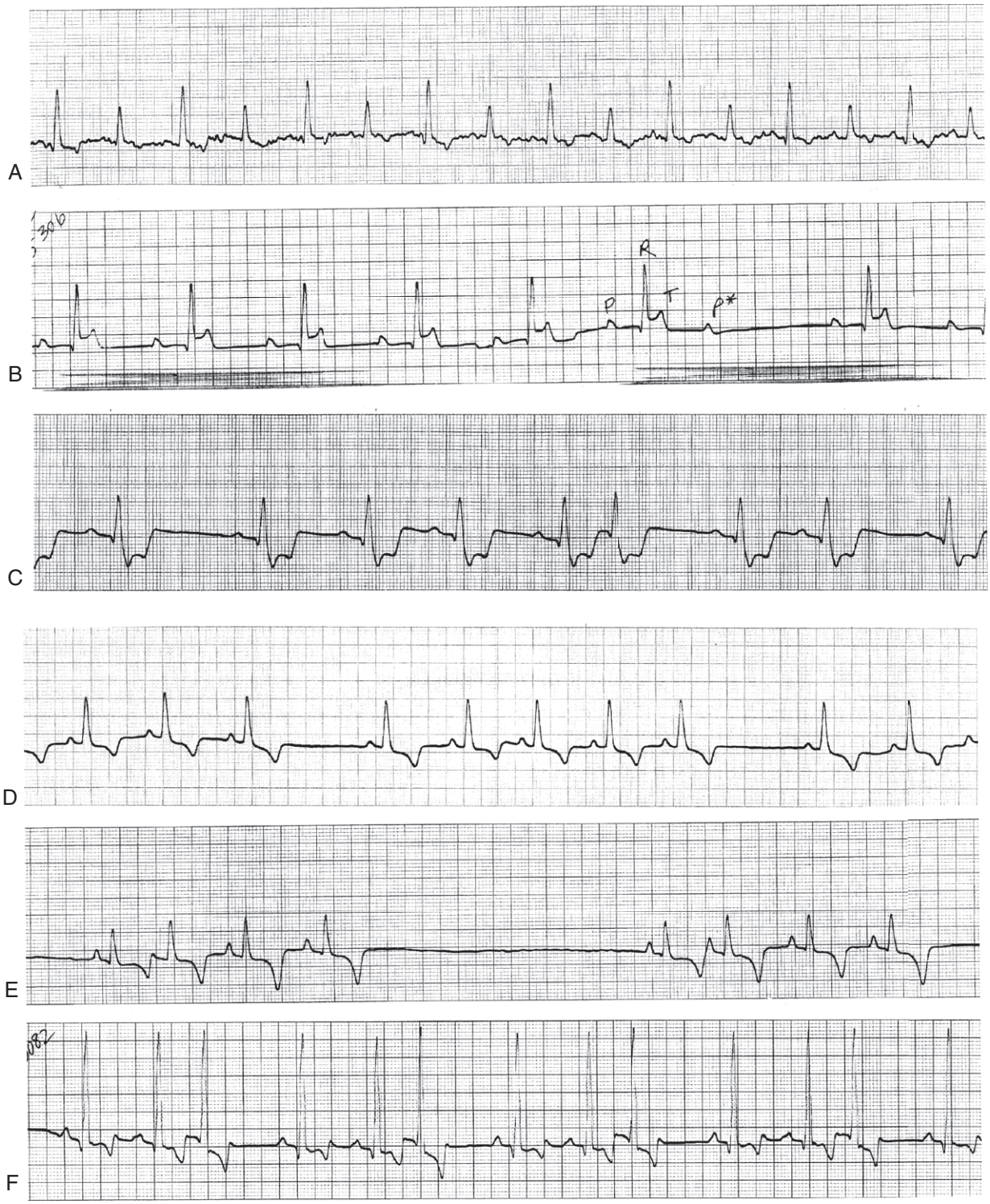


Fig. 3.11 **(A)** Electrocardiogram (ECG)–electrical alternans. **(B)** ECG–ST segment elevation. **(C)** ECG–ST segment depression. **(D)** ECG–normal sinus arrhythmia. **(E)** ECG–sinus arrest. **(F)** ECG–atrial premature complexes.

or pericardial effusion. In cats, digoxin toxicity may cause ST segment elevation.

- ST segment depression (Fig. 3.11C)
  - Depression of the ST segment greater than 0.2 mV in leads II, III, or aVF is abnormal in the dog. Any ST segment depression in the cat is abnormal.
  - ST segment depression may be caused by myocardial hypoxia, hyperkalemia, hypokalemia, subendocardial myocardial infarction, or digoxin toxicity.
  - Pseudodepression due to prominent T_a waves (atrial repolarization) caused by atrial disease or tachycardia also causes ST segment depression.

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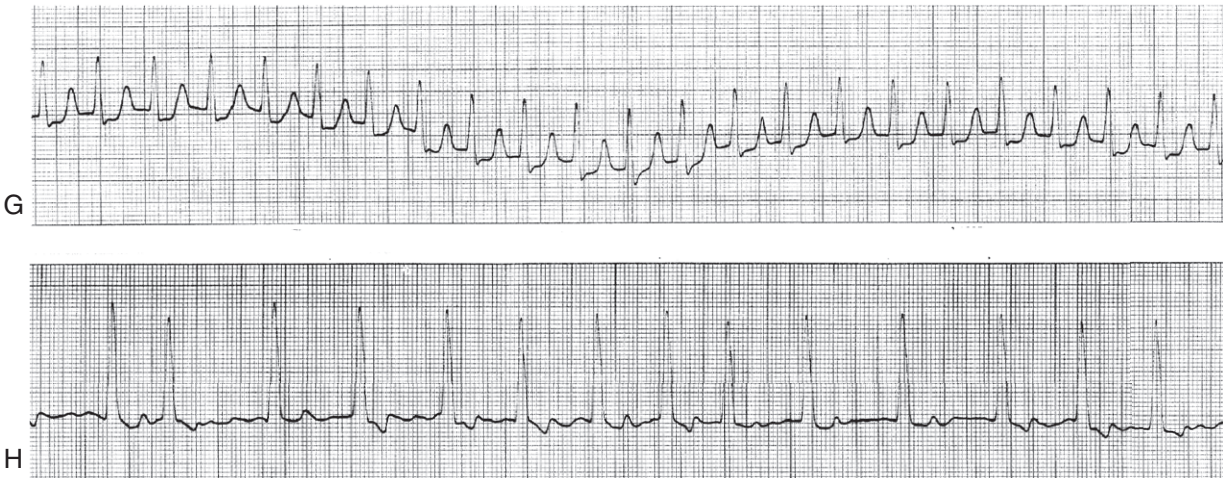


Fig. 3.11, cont'd **(G)** ECG–atrial tachycardia. **(H)** ECG–atrial fibrillation.

- Miscellaneous ST segment changes
  - ST segment changes may occur as a result of bundle branch blocks, myocardial hypertrophy, or VPCs. The changes in the ST segment are in the opposite direction from the main QRS deflection.

Key Point

Baseline artifact may mimic changes with the ST segment.

QT Interval

- The normal QT interval is 0.15 to 0.25 second (dog) and 0.12 to 0.18 second (cat). The QT interval tends to increase with slow HRs and decrease with rapid rates. In general, the QT interval should be less than half of the preceding R-R interval.
- The length of the QT interval is chiefly determined by an interplay of autonomic influences. HR and QT interval are governed separately by different sympathetic neurons that may or may not be activated together. Corrections of the QT interval (QT_c) for HR appear to be applicable under circumstances such as exercise.
- Within the past two decades, the overall ventricular repolarization and its relationship to the QT_c interval has led to studies in which the QT_c interval is used as a prognostic marker of ventricular tachyarrhythmias and death. Class IA or class III antiarrhythmic drugs, such as quinidine and sotalol, are known to prolong myocardial repolarization. This may either provide a protective effect against arrhythmias or lead to an increased occurrence of QT_c interval-related arrhythmias, including torsades de pointes ventricular tachycardia.
- Torsades de pointes (turning about a point) is a rare arrhythmia in the dog. It is a form of polymorphic ventricular tachycardia or flutter in which the amplitude of the complexes increases and decreases in size so that it appears to twist around the baseline. Humans with torsades de pointes are at risk for sudden death.

POSSIBLE FORMULAS FOR CORRECTED QT INTERVALS

- Many formulas and methods exist for correcting the QT interval for the effects of HR, including logarithmic, hyperbolic, and exponential functions, but they also have limitations. These limitations result from both physiologic and computational problems. The Bazett formula, for example, predicts an ever-increasing increment in the QT interval as the HR slows and an ever-decreasing increment as the rate rises, both of which are physiologically improbable. In addition, these formulas do not account for the effects of autonomic tone on the QT interval independent of the effects on rate. They also do not account for the relatively slow adaptation of repolarization to changes in rate. The Fridericia formula (QT divided by the cube root of the R-R interval) is a modification of the Bazett formula. This modification is important because the Bazett formula will overcorrect for rates higher than 60 beats/min. The Van de Water formula (based on a study in dogs) involves regression analysis yielding the following equation: QT_c = Van de Water formula = $QT - 87(60/HR - 1)$. Recent publications have recommended the use of the log-log formula for correcting the QT interval for HR (linear regression with $\log_e HR$ as the covariate: $QT = a + b [\log_e HR]$; a and b are constants).
- Prolongation of the QT interval may occur with interventricular conduction disturbances that are associated with prolongation of the QRS complexes, bradycardia, ethylene glycol toxicity, strenuous activity, or CNS disturbances. QT prolongation has

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been reported with many drugs and electrolyte imbalances. These include hypokalemia, hypocalcemia, quinidine, procainamide, bretylium, tricyclic antidepressants, and many anesthetics.
- Shortening of the QT interval may occur with hypercalcemia, hyperkalemia, or digoxin therapy.

T Wave

- The T wave is quite variable in the dog and cat. In most leads, the T wave may be positive, negative, or biphasic. The height of the T wave should not exceed one-fourth the height of the R wave, one-fourth the height of the Q wave (if Q wave is greater than R wave), or 0.5 to 1.0 mV in any lead.
- The T wave should be positive in CV_5RL in dogs aged 2 months and should be negative in V_{10}, except in the Chihuahua.
- Prominent T waves may occur with myocardial hypoxia, with interventricular conduction with disturbances, with ventricular enlargement, and in some animals with heart disease and bradycardia.
- Prominent and peaked T waves are associated with hyperkalemia.
- Small, biphasic T waves may occur with hypokalemia.
- Nonspecific T-wave changes may occur with metabolic disturbances (hypoglycemia, anemia, shock, fever), drug toxicity (digoxin, quinidine, procainamide), and neurologic disease.
- T-wave alternans has been reported as a result of hypocalcemia, increased circulating catecholamines, and sudden increases in sympathetic tone.

ARRHYTHMIAS AND CONDUCTION DISTURBANCES

- An arrhythmia (or dysrhythmia) refers to an irregularity in the cardiac rhythm. In general, an arrhythmia denotes an abnormality of the cardiac rhythm, although in the dog the term *normal sinus arrhythmia* is used to describe the normal variation in HR associated with respiration (Box 3.2).
  - Arrhythmias may be classified according to their **origin**.
    - Supraventricular arrhythmias arise from the atria or AV node.
    - Ventricular arrhythmias arise from the ventricles.
  - Arrhythmias may be classified according to their **rates**.
    - Arrhythmias with slow rates are referred to as bradyarrhythmias.
    - Arrhythmias with rapid rates are referred to as tachyarrhythmias.
  - Arrhythmias may be classified according to their **regularity**.
    - Fibrillation is an irregular, chaotic rhythm.
    - Tachycardia is a regular (nonirregular) rhythm.
    - Examples: Atrial fibrillation is an irregular, chaotic arrhythmia originating from the atria; ventricular tachycardia is a regular arrhythmia originating from the ventricles.
- There are several pathophysiologic causes of arrhythmias.
  - Abnormal automaticity of normal pacemaker cells
  - Shift of the pacemaker from the SA node to other areas of the heart
  - Conduction blocks that terminate or slow normal conduction through the heart
  - Abnormal pathways of impulse conduction through the heart
  - Spontaneous impulse formation in any area of the heart
- Systemic approach to arrhythmia recognition and evaluation
  - Any lead may be used for arrhythmia evaluation; lead II is generally used because waveforms are

Box 3.2 Classification of Cardiac Arrhythmias

- Normal sinus impulse formation
  - Normal sinus rhythm
  - Sinus arrhythmia
  - Wandering sinus pacemaker
- Disturbances of sinus impulse formation
  - Sinus arrest
  - Sinus bradycardia
  - Sinus tachycardia
- Disturbances of supraventricular impulse formation
  - Atrial premature complexes
  - Atrial tachycardia
  - Atrial flutter
  - Atrial fibrillation
  - Atrioventricular junctional rhythm
- Disturbances of ventricular impulse formation
  - Ventricular premature complexes
  - Ventricular tachycardia
  - Ventricular asystole
  - Ventricular fibrillation
- Disturbances of impulse conduction
  - Sinoatrial block
  - Persistent atrial standstill ("silent" atrium)
  - Atrial standstill (hyperkalemia)
  - Ventricular preexcitation
  - First-degree AV block
  - Second-degree AV block
  - Complete AV block (third degree)
  - Bundle branch blocks
- Disturbances of both impulse formation and impulse conduction
  - Sick sinus syndrome
  - Ventricular preexcitation and the Wolff-Parkinson-White (WPW) syndrome
  - Atrial premature complexes with aberrant ventricular conduction
  - Escape rhythms
  - Junctional escape rhythms
  - Ventricular escape rhythms (idioventricular rhythm)

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usually best defined in this lead. A significant duration of artifact-free lead II should be carefully evaluated.

- Determine if P waves are present. Examine all leads: P waves may be small or isoelectric in several leads. You must distinguish baseline motion (artifact) from P waves. P waves are generally consistent in size and distance from associated QRS complex. The presence of P waves indicates a sinus (originating from SA node) rhythm.
- Determine if an atrial (P wave) and ventricular (QRS complex) association exists. There should be a P wave for every QRS complex. If there are a greater number of P waves or QRS complexes, then an arrhythmia exists.
  - P waves should slightly precede QRS complexes.
  - If P waves follow the QRS complex, then AV dissociation exists, and the rhythm is ventricular or junctional rather than sinus.
  - There should be a QRS complex following each P wave. If not, then a conduction disturbance is present.
- Determine the regularity of the rhythm.
  - Normal sinus rhythm—insignificant (less than 10%) variation in the P-P or R-R intervals.
  - Normal sinus arrhythmia—significant (more than 10%) variation in the P-P or R-R intervals.
    - A pattern of irregularity is usually noted; the HR increases during inspiration and decreases during expiration in a cyclical pattern.
    - This may be referred to as a respiratory sinus arrhythmia.
  - Check for pauses in the rhythm—periods of inactivity greater than two R-R intervals.
  - Check for P waves occurring prematurely (occurring earlier than expected)—distinguish from normal sinus arrhythmia, where the P-wave rate increases during inspiration. Premature P waves will noticeably break the rhythm. Premature P waves indicate the presence of atrial premature complexes (APCs).
  - Check for QRS complexes occurring prematurely. These may occur without preceding P wave, may be superimposed on the P wave, or may follow the P wave with shortened PR interval. Premature QRS complexes indicate the presence of VPCs.

Key Point

Remember, a ventricular *premature* complex is *premature*. A ventricular complex occurring after a pause is *not* premature–it is a ventricular escape complex.

- Evaluation of PR interval
  - PR interval may vary slightly with changes in vagal tone—this would also result in a sinus arrhythmia.
  - Progressive prolongation of the PR interval signals first-degree AV block.
  - P waves without associated QRS complexes are usually indicative of second-degree AV block.
  - Shortened PR interval may be seen with increased sympathetic tone and preexcitation syndromes (presence of an accessory pathway bypassing AV node).
- Evaluation of QRS complexes
  - Height of R wave and duration of QRS complex
  - Presence of prominent S waves
  - Shape of QRS complexes should not vary significantly. Variation may be caused by the following:
    - VPCs or ventricular escape complexes
    - Fusion beats—simultaneous occurrence of VPC and normal QRS
    - Electrical alternans
    - Intermittent bundle branch block
    - Artifact
  - Premature QRS complexes suggest VPCs; QRS complexes without P waves and occurring after a pause suggest ventricular escape complexes.
- Note shape of any premature complexes: APCs have a QRS of normal shape; VPCs have a QRS that is significantly different from the normal QRS.

Key Point

QRS complexes should not vary in their shape in a normal animal.

NORMAL RHYTHMS

NORMAL SINUS RHYTHM

- Normal rhythm in the dog and cat.
- P wave for every QRS complex, regular rhythm.
- Rate between 60 and 180 beats/min for the dog, depending on the size of the dog and the degree of anxiousness.
- Rate between 140 and 240 beats/min for the cat, depending on the degree of anxiousness.
- Rate and rhythm dependent on SA nodal discharge. This is highly influenced by changes in autonomic tone. An elevated sympathetic tone will increase the rate of SA nodal discharge; an elevated parasympathetic (vagal) tone will decrease the rate of SA nodal discharge.

NORMAL SINUS ARRHYTHMIA

- Sinus arrhythmia is usually normal in the dog and generally abnormal in the cat.
- A pattern of irregularity is present—the HR increases during inspiration and decreases during expiration in a cyclical pattern (Fig. 3.11D).

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- Irregularity is secondary to fluctuations in vagal tone associated with the respiratory cycle.
- When respiratory effort is increased (brachycephalic breeds, animals with respiratory disease), fluctuations in vagal tone may be dramatic, producing a pronounced sinus arrhythmia.
- Normal sinus arrhythmia is a rhythm of high vagal tone and low sympathetic tone; this situation is rare in dogs with congestive heart failure. The presence of a sinus arrhythmia in a dog with severe cough is more supportive of primary respiratory disease than of congestive heart failure.

WANDERING SINUS PACEMAKER

- The origin of SA nodal discharge may change as a consequence of alterations in vagal tone. This is noted on the ECG as a cyclical change in the height of the P wave. At times, the P wave may become isoelectric and not detectable.
- Often associated with normal sinus arrhythmia or pronounced sinus arrhythmia.

Key Point

Sinus arrhythmia is *not* expected in the cat but is normal and is the result of variations in vagal tone in dogs.

DISTURBANCES OF SINUS IMPULSE FORMATION

SINUS ARREST

- When the SA node fails to discharge as expected, a pause in the rhythm will occur. The duration of the pause is at least twice the preceding R-R interval. When severe, pause duration may be 5 to 12 seconds (Fig. 3.11E).
- For survival, the pause is ended by a ventricular escape complex, junctional escape complex, or normal complex.
- Causes include fibrosis of the SA nodal tissue, greatly increased vagal stimulation, drug influences (digoxin, beta-blockers), and, rarely, neoplasia.
- If severe and frequent, intermittent weakness or syncope may occur.

SINUS BRADYCARDIA

- A sinus rhythm with an abnormally slow rate.
- May be physiologic: during sleep, the HR of many dogs drops to 30 to 40 beats/min. Calm animals or athletic animals at rest may have a physiologic sinus bradycardia.
- Elevated vagal tone may result in a sinus bradycardia.
- Drug induced: anesthetics and sedatives, digoxin, calcium channel blockers, beta-blockers.
- Pathologic: hypothermia, hypothyroidism, sick sinus syndrome (SSS).
- Specific therapy for sinus bradycardia is not warranted unless clinical signs (weakness, reduced cardiac output) have developed as a result of the arrhythmia.

SINUS TACHYCARDIA

- A sinus rhythm with an abnormally rapid rate.
- May be physiologic—associated with exercise, stress, anxiousness, pain.
- Drug induced: atropine or glycopyrrolate, methylxanthines (theophylline), excessive thyroid supplementation, catecholamines (epinephrine, dobutamine).
- Pathologic: fever, shock, hyperthyroidism, anemia, hypoxia, congestive heart failure.
- Specific therapy for sinus tachycardia is usually indicated for rate control in patients with congestive heart failure.

Key Point

The underlying cause of the sinus tachycardia should be identified and addressed if necessary.

DISTURBANCES OF SUPRAVENTRICULAR IMPULSE FORMATION

ATRIAL PREMATURE COMPLEXES

- An APC is an abnormal beat occurring prematurely and originating in atrial tissue.
- Electrocardiographic features in the following (Fig. 3.11F):
  - Presence or absence of ectopic P wave; the P wave is usually of normal shape.
  - Ectopic P wave may be superimposed on the preceding T wave.
  - Premature QRS may have identical or nearly identical appearance to normal QRS.
  - A compensatory pause may follow the APC.
  - If the APC occurs when the ventricles are refractory, then an isolated ectopic P wave will be noted; it is nonconducted because of the refractoriness of the ventricles.
- Causes generally include any disease associated with atrial enlargement, such as degenerative valve disease, congenital heart disease, or cardiomyopathy. Other causes include hypoxia, atrial neoplasia, and chronic obstructive pulmonary disease.
- Therapy is not generally warranted for infrequent APCs. Antiarrhythmic therapy may be needed if APCs are frequent and thought to be compromising cardiac output or if there is a concern of impending atrial tachycardia or atrial fibrillation.

ATRIAL TACHYCARDIA

- A paroxysmal tachycardia originating from atrial tissue (other than SA node)

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- Electrocardiographic features (Fig. 3.11G)
  - There is a rapid rate—from 200 to 350 beats/min.
  - There is usually a regular rhythm. If originating from multiple atrial sites, then an irregular rhythm may occur.
  - P waves may be difficult to discern. QRS complexes are generally normal but may widen, or electrical alternans may develop.
  - The onset and termination of arrhythmia is sudden.
  - It may occur as a reentrant arrhythmia within the AV node.
- Same causes as for APCs
- May be clinically significant depending on rate, frequency of runs, and length of runs

ATRIAL FLUTTER

- An uncommon arrhythmia characterized by a rapid atrial rate (greater than 250 beats/min) and altered atrial depolarization resulting in bidirectional saw-toothed atrial complexes (F waves). Ventricular rate varies depending on refractoriness of AV node.
- It is usually a result of severe structural heart disease.
- Clinical significance depends on ventricular rate. If excessive, then cardiac output is reduced.

ATRIAL FIBRILLATION

- A common arrhythmia in the dog characterized by lack of P waves, rapid ventricular rate, and irregularity of ventricular depolarizations. In atrial fibrillation, there are numerous sites of ectopic atrial depolarization and varying AV nodal refractoriness (Fig. 3.11H). Atrial fibrillation is uncommon in the cat.
- Baseline may be flat or may exhibit fine fibrillation potentials.
- Causes include the following:
  - Structural heart disease (advanced degenerative valve disease, dilated cardiomyopathy, atrial neoplasia, congenital heart disease)
  - Lone atrial fibrillation, which occurs in large- to giant-breed dogs without structural heart disease
  - May occur as a complication of noncardiac disease, such as gastric dilatation-volvulus or other disorders altering vagal tone
  - May be drug induced (e.g., digoxin)
- Clinical significance depends on ventricular rate in most cases. With atrial fibrillation there is also loss of atrial contraction (atrial kick), which may reduce ventricular performance. If the rate is not controlled, then lone atrial fibrillation may cause myocardial deterioration and secondary dilated cardiomyopathy.

JUNCTIONAL PREMATURE COMPLEXES

- A junctional premature complex is an abnormal beat occurring prematurely and originating in the AV nodal area.
- Electrocardiographic characteristics include abnormal-appearing P wave (often inverted), which may precede QRS, be superimposed on QRS, or follow QRS complexes. The QRS complex is usually unaffected.
- Causes are the same as for APCs.
- Clinical significance is the same as APCs.

ATRIOVENTRICULAR JUNCTIONAL TACHYCARDIA

- A paroxysmal or sustained rhythm originates from AV nodal tissue.
- Electrocardiographic features.
  - Rate is greater than 60 beats/min (inherent rate of AV nodal tissue is approximately 40–60 beats/min).
  - There is a regular rhythm.
  - Abnormal-appearing P wave (often inverted in lead II) may precede QRS, be superimposed on QRS, or follow QRS complexes.
  - QRS complexes may be normal or may be widened as a result of aberrant conduction.
- Causes include digoxin toxicity and structural heart disease.
- It may be clinically significant depending on ventricular rate.

Key Point

AV junctional tachycardia may be difficult to distinguish from atrial tachycardia.

SUPRAVENTRICULAR TACHYCARDIA

- SVT refers to a rapid arrhythmia originating from supraventricular tissue but in which the exact site of origin cannot be determined. Atrial tachycardia and AV junctional tachycardia are often indistinguishable, so the term *SVT* may be more precise in these cases.

DISTURBANCES OF IMPULSE CONDUCTION

SINUS BLOCK

- The SA node discharges normally, but the impulse is blocked by neighboring tissue.
- It produces a pause equal to twice the preceding R-R interval, with immediate restoration of rhythm.
- Causes are the same as for sinus arrest.
- Sinus block is usually not clinically significant.

ATRIAL STANDSTILL

- Atrial standstill is characterized by an absence of P waves and can be temporary in nature or persistent. The most common cause of temporary atrial standstill is hyperkalemia. Persistent atrial standstill is caused by an atrial muscular dystrophy, most commonly occurring in English Springer Spaniels.

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- In atrial standstill caused by hyperkalemia, SA nodal discharge occurs, but atrial depolarization is blocked by the effects of hyperkalemia. Because there is no atrial depolarization, P waves are absent. The impulse originating from the SA node reaches the AV node by way of internodal fibers. Hyperkalemia also slows the rate of SA nodal discharge and affects ventricular depolarization and repolarization.
- Electrocardiographic characteristics of persistent atrial standstill include the following:
  - Absence of P waves
  - HR usually slow (<60 beats/min)
  - Rhythm regular with supraventricular-appearing QRS complexes
  - No increase of HR with atropine administration
- The following are electrocardiographic characteristics of atrial standstill caused by hyperkalemia (Fig. 3.12A):
  - As serum potassium increases, P-wave amplitude diminishes. The absence of P waves occurs when the potassium approaches 8.0 mEq/L.
  - The ventricular rate is slow: the rhythm is termed *sinoventricular*, and the rate is approximately 20 to 40 beats/min.
  - As serum potassium increases, the T-wave amplitude increases and becomes peaked.
  - QRS duration progressively increases, and the R-wave height decreases as the serum potassium levels increase.
  - HR may increase slightly with atropine administration.
- Causes of hyperkalemia include hypoadrenocorticism, anuric or oliguric renal failure, uncontrolled diabetic ketoacidosis, metabolic acidosis, urethral obstruction, and rupture of the urinary bladder.

Key Point

Atrial standstill caused by hyperkalemia is usually life threatening.

VENTRICULAR PREEXCITATION SYNDROME

- In these syndromes, an abnormal accessory pathway exists, which bypasses the AV node. This results in a shortening or loss of the PR interval due to premature activation of the ventricles. With an accessory pathway, there are two electrical connections between the atria and the ventricles, and both may simultaneously be relaying impulses. There also exists the potential for reentry because the impulse may leave the atria through one pathway and immediately reenter through the other. The accessory pathway may connect the atria to the distal AV node, to the bundle of His, or directly to ventricular tissue.
- Electrocardiographic features include the following (Table 3.2 and Fig. 3.13):
  - The PR interval is shortened.
  - If the accessory pathway terminates in ventricular tissue, then initial slurring of the QRS complex (delta wave) occurs.
  - There is paroxysmal reentrant tachycardia in some cases.
- Causes include congenital anomalies, feline hypertrophic cardiomyopathy, and other structural heart diseases.
- Clinical significance depends on the frequency and severity of secondary tachycardia resulting from the accessory pathway.

FIRST-DEGREE ATRIOVENTRICULAR BLOCK

- Conduction through the AV node is delayed.
- Produces prolongation of the PR interval (more than 0.13 second for the dog, more than 0.09 second for the cat) (see Fig. 3.12B).
- Causes include fibrosis of the AV node, vagal stimulation, drug induced (digoxin), and electrolyte imbalance.
- Isolated first-degree AV block is not clinically significant but may be an early indicator of progressive AV nodal dysfunction.

SECOND-DEGREE ATRIOVENTRICULAR BLOCK

- There is intermittent blockage of conduction through the AV node.
- After discharge of the SA node and atrial depolarization, there is no associated ventricular depolarization.
- Electrocardiographic features include absence of QRS-T complexes following the P wave (see Fig. 3.12C).
  - In a Mobitz type I (Wenckebach) block, there is progressive prolongation of the PR interval, followed by the occurrence of a second-degree AV block.
  - In a Mobitz type II block, there is a second-degree AV block without preceding prolongation of the PR interval.
  - A pattern of block may exist; a 2:1 AV block would refer to the presence of two P waves for every QRS complex.
  - Advanced or high-grade AV block consists of more than two consecutive blocked atrial depolarizations.
- Causes are identical to those of first-degree AV block.
- High-grade AV block may reduce cardiac output and result in clinical signs.

Key Point

Second-degree block may progress to complete AV block. This is more common with Mobitz type II AV block.

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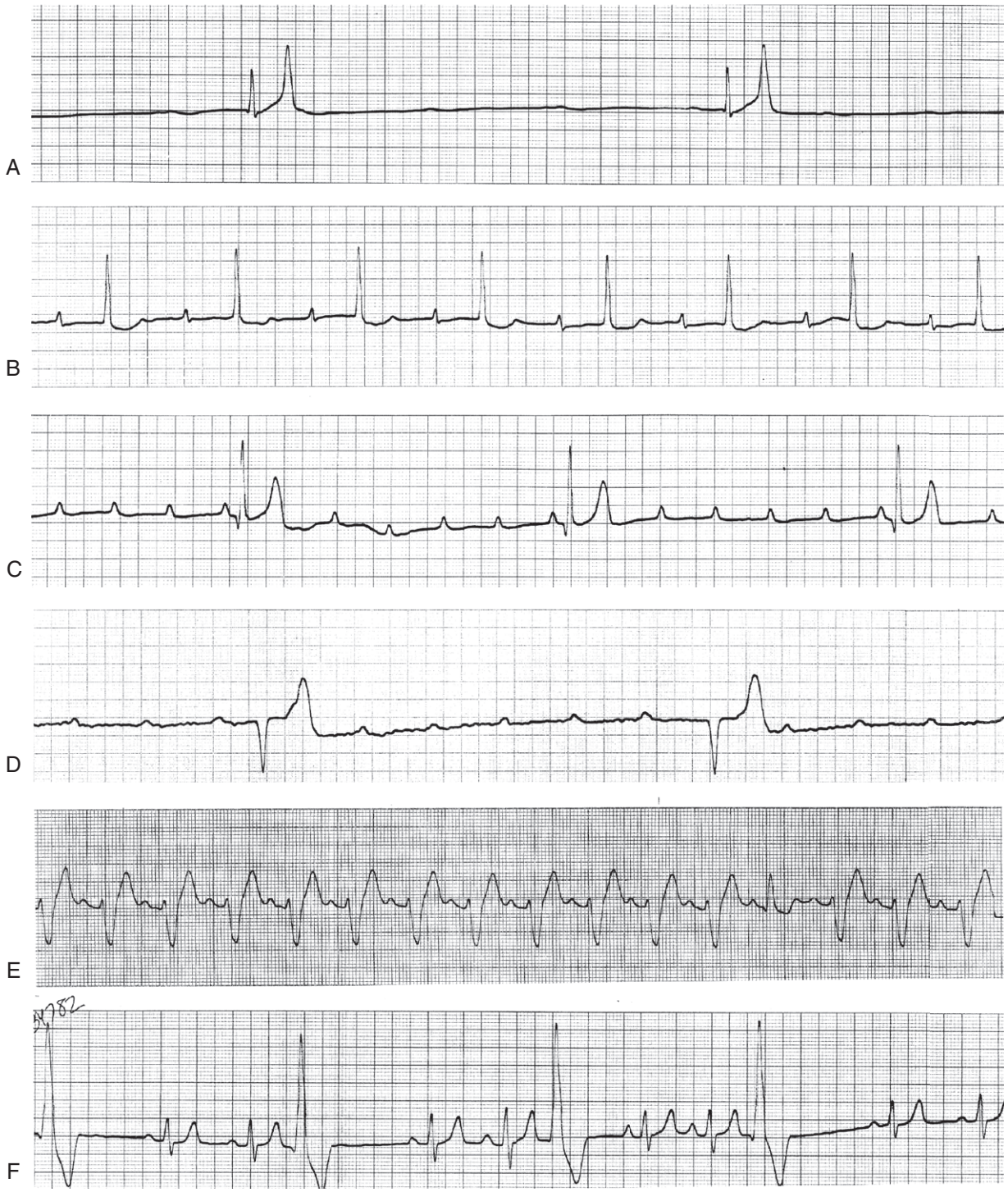


Fig. 3.12 **(A)** Electrocardiogram (ECG)–atrial standstill. **(B)** ECG–first-degree atrioventricular (AV) block. **(C)** ECG–second-degree AV block. **(D)** ECG–third-degree AV block. **(E)** ECG–right bundle branch block. **(F)** ECG–unifocal ventricular premature complexes (VPCs).

THIRD-DEGREE ATRIOVENTRICULAR BLOCK (COMPLETE HEART BLOCK)

- All conduction through the AV node is blocked. Atrial and ventricular depolarizations are no longer coordinated and occur independently of one another. Ventricular depolarization is initiated by discharge of a ventricular escape focus.
- Electrocardiographic features (see Fig. 3.12D) include the following:
  - There is no association between P waves and QRS-T complexes.
  - P waves are of normal shape and usually occur at a normal rate.
  - QRS complexes are of ventricular origin shape.
  - Ventricular rate is typically 30 to 50 beats/min.

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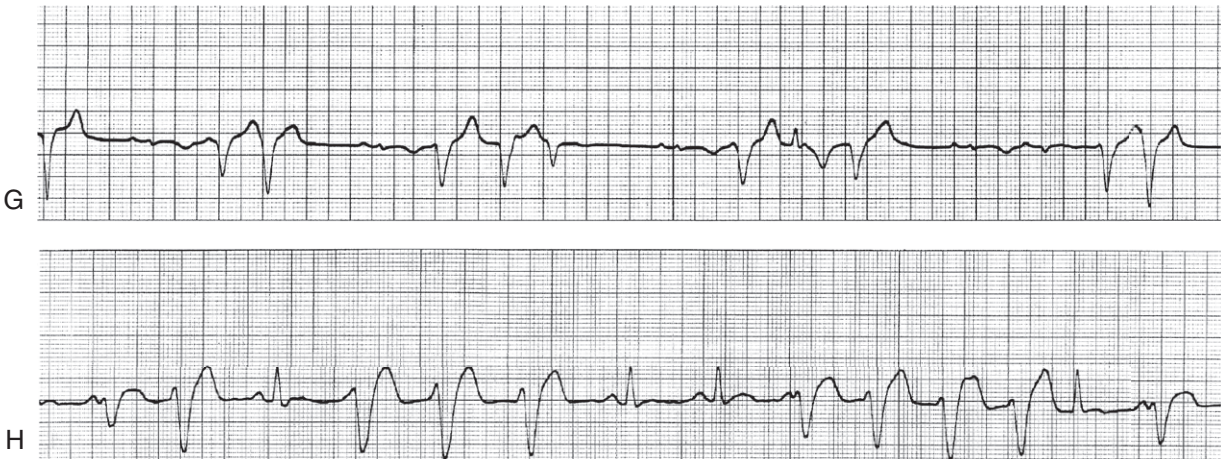


Fig. 3.12, cont'd **(G)** ECG–multiform VPCs. **(H)** ECG–run of VPCs.

Table 3.2 Summary of Preexcitation Syndrome

CARDIAC SEQUENCE	MECHANISM	ELECTROCARDIOGRAM
Sinus impulse	Normal	—
Atrial depolarization	Normal	Normal P wave
AV node accessory pathways	Relatively rapid conduction, skirting the A-V nodal system	Short PR interval
Early ventricular depolarization	One ventricle activated early	Initial QRS slurred (delta wave) (widened QRS)
Retrograde conduction from ventricles to atria	Atrial reentry impulse	Tendency to supraventricular paroxysmal arrhythmias
Late ventricular depolarization	Fusion between normal and anomalous ventricular activation	Delay, often with altered direction, of terminal QRS

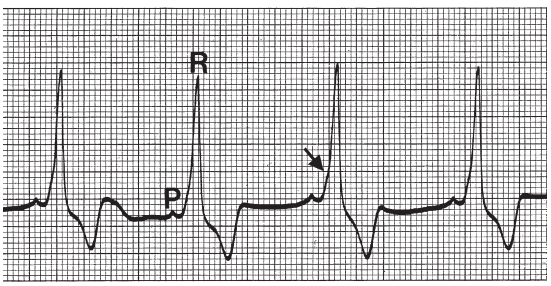


Fig. 3.13 Ventricular preexcitation in a dog. A short PR interval and a widened QRS complex with slurring or notching (*arrow*) of the upstroke ("delta" wave) are present. (From Fox PR, Sisson D, Moise NS, eds: *Textbook of canine and feline cardiology*, St. Louis, 1999, Elsevier.)

- Causes include fibrosis of the AV node, drug induced (digoxin), infiltrative disease, Rickettsial myocarditis, and hyperkalemia.
- Usually associated with clinical signs of weakness or collapse. A complete AV block warrants implantation of a permanent pacemaker in most cases.

LEFT BUNDLE BRANCH BLOCK

- A conduction delay or block in both the left posterior and the left anterior fascicles of the left bundle. A supraventricular impulse activates the right ventricle first through the right bundle branch. Left ventricular depolarization is delayed.
- Electrocardiographic features include the following (Fig. 3.14):
  - QRS duration is prolonged (>0.08 second for the dog; >0.06 second for the cat)
  - QRS is wide and positive in leads I, II, III, and aVF.
  - Unlike VPCs, the QRS complex is associated with a preceding P wave.
  - It may be difficult to distinguish from a left ventricular enlargement pattern.
- Causes include structural heart disease (cardiomyopathy, congenital anomalies, neoplasia, trauma, fibrosis).
- The presence of a LBBB does not impair cardiac performance directly but is a marker of significant heart disease.

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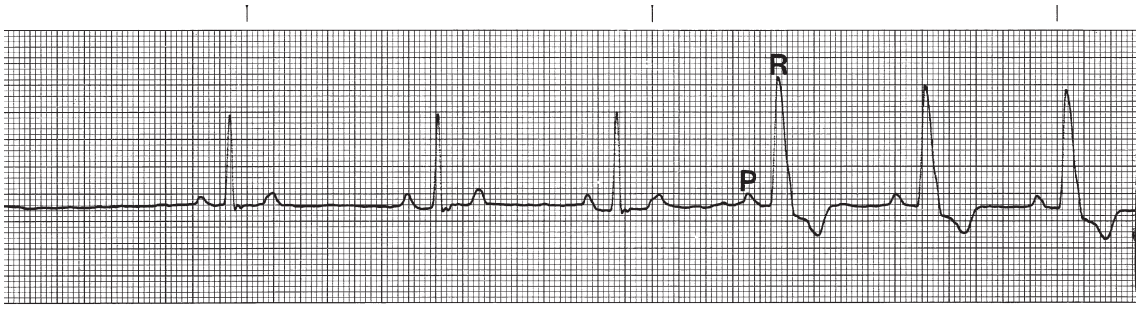


Fig. 3.14 Intermittent left bundle branch block in a dog. The QRS complexes are wider and taller than normal in the fourth, fifth, and sixth complexes. (From Fox PR, Sisson D, Moise NS, eds: *Textbook of canine and feline cardiology*, St. Louis, 1999, Elsevier.)

Key Point

Complete heart block will occur if right bundle branch block (RBBB) develops in an animal with LBBB.

LEFT ANTERIOR FASCICULAR BLOCK

- There is a block in the left anterior fascicle of the left bundle branch slowing depolarization of the left ventricle.
- Electrocardiographic features include the following:
  - QRS duration within normal limits
  - Left-axis deviation (dog, less than 40 degrees; cat, less than 0 degrees)
  - Small Q wave and tall R wave in leads I and aVL (small Q wave not consistent)
  - Deep S waves in leads II, III, and aVF (exceeding R wave)
  - Associated with feline hypertrophic cardiomyopathy, other diseases associated with left ventricular hypertrophy, hyperkalemia, ischemia, and postcardiac surgery

Key Point

Left anterior fascicular block (LAFB) does not directly impair cardiac function, but if present, one should look for underlying causes.

RIGHT BUNDLE BRANCH BLOCK

- There is a block of the right bundle branch, delaying depolarization of the right ventricle.
- Electrocardiographic features include the following (see Fig. 3.12E):
  - Increase of QRS duration
  - Prominent S waves in leads I, II, III, and aVF
  - Right-axis shift
- It is associated with structural heart disease, Chagas disease, heartworm disease, acute pulmonary thromboembolism, and hypokalemia.

Key Point

As in the case of LAFB, RBBB does not significantly impair cardiac function, but one should look for underlying causes.

DISTURBANCES OF VENTRICULAR IMPULSE FORMATION

VENTRICULAR PREMATURE COMPLEXES

- An abnormal beat originates in the ventricles and occurs earlier than expected in relation to the existing rhythm. A compensatory pause often follows a VPC.
  - Unifocal VPCs occur from one ventricular site and have identical shapes (see Fig. 3.12F).
  - Multifocal VPCs usually occur from more than one ventricular site and have differing shapes. Electrophysiologic studies have demonstrated that a single ventricular site may produce VPCs of differing shape; this is due to altered conduction of the VPCs rather than multiple ectopic sites. It is more precise, therefore, to use the term *multiform* rather than *multifocal* (see Fig. 3.12G).
  - When the VPC does not alter the underlying normal rhythm, it is considered to be interpolated.
  - When normal ventricular depolarization is interrupted by a VPC, a fusion beat occurs; this is essentially the electrocardiographic merging of a normal QRS and VPC.
- Electrocardiographic features include the following (see Fig. 3.12F–H):
  - As the site of depolarization is ventricular, there is no AV association. P waves are not associated with the QRS complexes of the VPCs.
  - QRS complexes are wide and bizarre, consistent with ventricular origin.
  - T-wave polarity is often reversed.
  - Compensatory pause following VPC is typical.
  - The following are patterns of VPCs:
  - Two consecutive VPCs are referred to as a couplet.
    - Three or more consecutive VPCs are a salvo or run (see Fig. 3.12H).
    - When every other complex is a VPC, ventricular bigeminy exists.
    - When every third complex is a VPC, ventricular trigeminy exists.
    - R-on-T phenomenon occurs when the VPC occurs immediately following a normal beat within the T wave. This may predispose to development of ventricular tachycardia.

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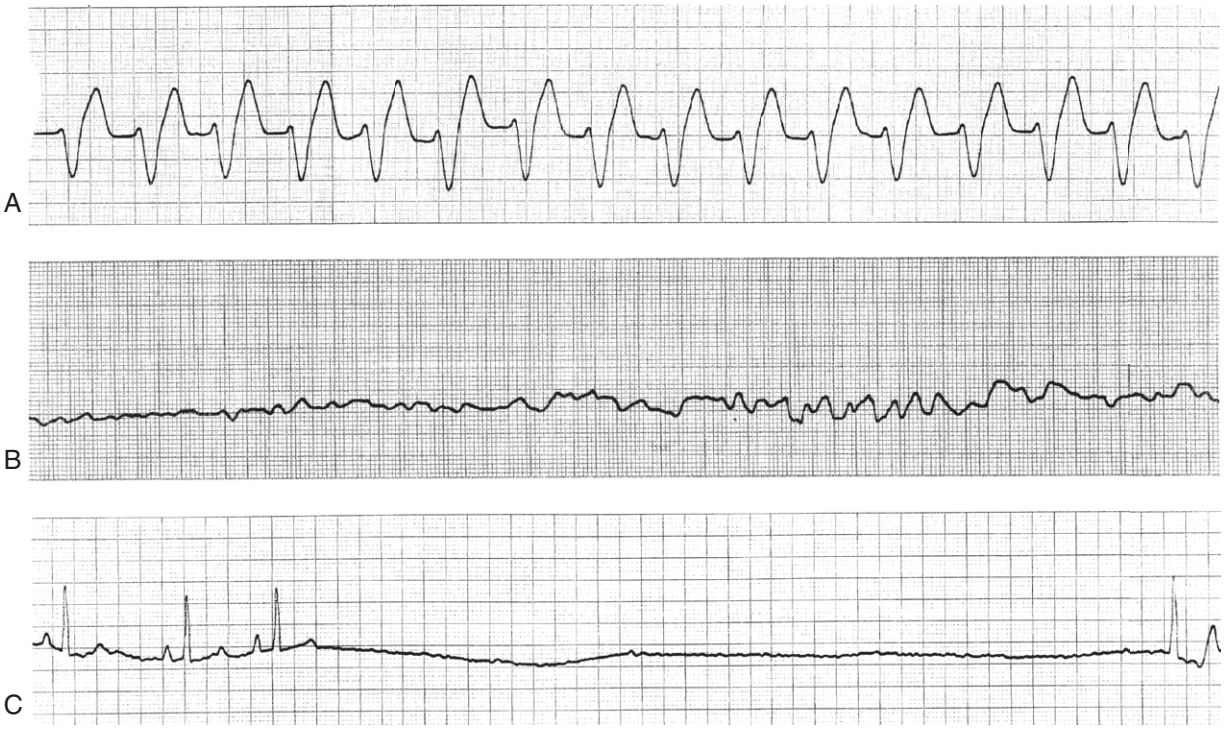


Fig. 3.15 **(A)** Electrocardiogram (ECG)–ventricular tachycardia. **(B)** ECG–ventricular fibrillation. **(C)** ECG–sick sinus syndrome.

- Causes are numerous and include structural heart disease, inheritance in young German Shepherds, arrhythmogenic right ventricular cardiomyopathy (Boxer cardiomyopathy), hypoxia, anemia, uremia, gastric dilatation-volvulus, splenic torsion and splenic neoplasia, pancreatitis, myocarditis, and use of drugs (digoxin, anesthesia).
- Clinical significance depends on the frequency of VPCs, pattern of ectopy, and cause of the arrhythmia.
  - Isolated VPCs usually pose no significant problems but may signal the presence of progressive disease and potential for more serious arrhythmia.
  - Runs of VPCs do suggest the potential for ventricular tachycardia and possibly ventricular fibrillation.

Key Point

The finding of isolated VPCs in a young to middle-aged Doberman Pinscher or Boxer is highly suggestive of occult dilated cardiomyopathy or arrhythmogenic right ventricular cardiomyopathy, respectively.

VENTRICULAR TACHYCARDIA

- These are runs of VPCs occurring in succession at a rate usually greater than 100 beats/min. The inherent discharge rate of the ventricle is approximately 40 to 50 beats/min (seen with complete heart block). In normal animals, this is overdriven by the sinus rhythm. An accelerated idioventricular rhythm refers to a ventricular rhythm at a rate of 60 to 100 beats/min. Ventricular tachycardia may be sustained or paroxysmal.
- Electrocardiographic features include the following (Fig. 3.15A):
  - P waves not associated with QRS complexes
  - QRS complexes wide and bizarre, consistent with ventricular origin
  - Regular rhythm, as contrasted with atrial fibrillation and RBBB
- Causes are the same as for VPCs.
- Ventricular tachycardia indicates significant heart disease or systemic disease. Cardiac output may be significantly impaired, and the arrhythmia predisposes to ventricular fibrillation.

VENTRICULAR FIBRILLATION

- Completely irregular, chaotic variable fibrillation potentials, indicating lack of organized ventricular depolarization and impending death
- Electrocardiographic features (Fig. 3.15B)
  - No evidence of organized cardiac depolarization (absence of P-QRS-T waves)
  - Wavy, undulating baseline
  - Coarse ventricular fibrillation that is characterized by large wavelets
  - Fine ventricular fibrillation that is characterized by small wavelets

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Key Point

Ventricular fibrillation indicates cardiopulmonary arrest, and immediate restoration of rhythm is required to preserve life.

VENTRICULAR ASYSTOLE

- There is lack of any significant ventricular electrical activity.
- ECG demonstrates flat baseline; occasional ventricular escape complexes may occur.
- A terminal rhythm requires immediate restoration of rhythm to preserve life.

DISTURBANCES OF BOTH IMPULSE FORMATION AND IMPULSE CONDUCTION

SICK SINUS SYNDROME

- Sick sinus syndrome (SSS) is a progressive heart disease characterized by a variety of arrhythmias, including sinus bradycardia, sinus arrest, paroxysmal atrial tachycardia (bradycardia-tachycardia syndrome), intermittent AV nodal block, and lack of ventricular escape complexes (Fig. 3.15C).
- Breeds predisposed include Miniature Schnauzers, Cocker Spaniels, Dachshunds, Pugs, and West Highland White Terriers. SSS is most common in older female dogs.
- The cause is unknown but likely involves idiopathic degeneration of the conduction system.
- Most patients have a history of intermittent weakness and collapse.
- Medical therapy may be successful in some cases; many require pacemaker implantation.

ATRIAL PREMATURE COMPLEXES WITH ABERRANT CONDUCTION

- When the impulse of an APC encounters an area of refractoriness (AV node, bundle of His, or ventricular myocardium), it may terminate or continue with aberrant conduction. The latter may result in a wide and bizarre QRS configuration resembling a beat of ventricular origin.
- Most often occur at slow HRs.
- QRS may take the form of RBBB.

ESCAPE RHYTHMS

JUNCTIONAL ESCAPE BEAT

- When not activated by atrial depolarization, the junctional AV nodal area may spontaneously discharge. This impulse results in ventricular depolarization in a normal fashion. Junctional escape beats may occur when there is a significant pause in the sinus rhythm.
- Electrocardiographic features include the following:
  - Inverted P wave, occurring before, during, or just after QRS complex
  - Normal or relatively normal QRS complex
  - Occurrence after significant pause in sinus rhythm
- Clinical significance—this complex is adaptive and helps to maintain cardiac output in the face of a slow rate or sinus arrest.

Key Point

Junctional escape beats should not be suppressed.

JUNCTIONAL RHYTHM

- This rhythm is a succession of junctional escape beats in absence of adequate sinus node function.
- Rate of junctional escape rhythm is typically 40 to 60 beats/min.
- This rhythm is adaptive and should not be suppressed. Correction of the cause of sinus node dysfunction is warranted.

VENTRICULAR ESCAPE BEAT

- When not activated by atrial depolarization, a ventricular focus may spontaneously discharge. This impulse results in an abnormal ventricular depolarization, but contraction is not affected. Ventricular escape beats may occur when there is a significant pause in the sinus rhythm and lack of junctional escape complexes.
- Electrocardiographic features include the following:
  - Occurrence of ventricular escape beat after a pause in the rhythm
  - QRS wide and bizarre, consistent with ventricular origin
- Clinical significance—this complex is adaptive and helps to maintain cardiac output in the face of a slow rate or sinus arrest.

Key Point

These ventricular escape beats should not be suppressed.

VENTRICULAR ESCAPE RHYTHM

- This rhythm is a succession of ventricular escape beats in absence of adequate sinus node function.
- Rate of ventricular escape rhythm is typically 30 to 40 beats/min.
- This rhythm is adaptive and should not be suppressed. Correction of the cause of sinus node dysfunction is warranted.

MISCELLANEOUS

ARTIFICIAL PACEMAKER

- A permanent pacemaker is used to control bradyarrhythmias that caused clinical signs and were unresponsive to medical therapy.

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- Transvenous implantation with an endocardial lead is the preferred means and is much less invasive than abdominal surgery and epicardial lead implantation.
- Most pacemakers used in dogs are set at 100 beats/min.
- Electrocardiographic features include the following:
  - If the sinus node is functioning and the HR is greater than 100 beats/min (or the pacemaker's discharge rate), then there will be no electrocardiographic changes.
  - Once the HR decreases below the pacemaker's minimum rate, a pacing spike will appear, followed by a wide and bizarre QRS (ventricular origin of impulse).

Key Point

The beats generated by the pacemaker are not of normal shape; this is expected.

PARASYSTOLE

- In parasystole, there is an independent focus discharging spontaneously. There is an entrance block, so the focus is not overdriven by the normal cardiac impulse. The parasystolic focus will discharge at a regular rate and may cause complete depolarization of the atria or ventricles.
- Atrial parasystole focus is located within atrial myocardium and produces small P waves, usually unassociated with QRS complexes.
- Ventricular parasystole focus is located within ventricular myocardium and produces regularly spaced QRS complexes. When the focus discharges during the ventricle's refractory period, a QRS will not be created.

ASHMAN'S PHENOMENON

- Tendency of premature supraventricular beats to have aberrant ventricular conduction when a short cycle follows a long one

Key Point

Ashman's aberrance may mimic a VPC.

Frequently Asked Questions

IF A VETERINARIAN AUSCULTS AN ABNORMALITY THAT COMES AND GOES AND THE ECG DOES NOT SHOW AN ARRHYTHMIA, WHAT SHOULD BE DONE?

Repeat ECGs may be required to pick up changes in heart activity occurring only in a paroxysmal pattern. In some cases, a Holter monitor must be used for a 24-hour recording so that the pattern and frequency of abnormality can be properly tracked.

INTERFERENCE IS OCCURRING ON AN ECG TRACING; IT APPEARS TO BE 60 CYCLE (SMALL WAVEFORMS) AND IS NOT IMPROVED WHEN THE FLUORESCENT BULBS AND ALL ELECTRICAL EQUIPMENT IN THE ROOM ARE TURNED OFF. WHAT COULD CAUSE THIS ONGOING INTERFERENCE?

Sometimes interference may travel through the walls from the adjacent room, and proximity to power supply in the wall could also send off interference. Electrical supply to the facility must be properly grounded, so if the problem persists when all of those factors have been controlled, an electrician should examine the electrical system of the building.

WE MUST DO A SURGERY THAT REQUIRES AN UNUSUAL POSITION FOR THE DOG. WILL ABNORMAL RELATIVE AND ABSOLUTE POSITIONING OF THE CLIPS INTERFERE WITH OUR ROUTINE INTRAOPERATIVE MONITORING?

Position is not critical for routine intraoperative electrocardiography, as opposed to position required for a primary ECG with multiple leads for diagnostic purposes, and a useful capture can be done despite unorthodox limb or animal position. Any position for the recording will still always give an analysis of an arrhythmia, conduction abnormality, or both. Watching that the extreme positioning required will not lead to the lead wires or clips touching a metal surgical table is important because such contact can cause interference.

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Chapter 4

Echocardiography and Doppler Ultrasound

Virginia Luis Fuentes

INTRODUCTION

Echocardiography is the most important diagnostic technique for the diagnosis of canine and feline heart disease. The interaction between the heart and ultra high frequency sound waves produces a cardiac imaging technique of great versatility. Echocardiography allows us to evaluate a wide range of cardiac features, including cardiac morphology, cardiac motion, and blood flow velocity. Echocardiography is complementary to physical examination, radiography, and electrocardiography (ECG). Echocardiography is a valuable resource at every level of small animal cardiology, from focused 2D imaging (point-of-care ultrasound) in emergency and first-opinion settings to interventional echocardiography, where transesophageal echocardiography and 3D echocardiography play a key role in cardiac interventions.

TYPES OF IMAGING

TWO-DIMENSIONAL ECHOCARDIOGRAPHY

- A sector-shaped beam of ultrasound waves is reflected by the interfaces of cardiac tissue to provide a 2D cross-sectional (tomographic) image in real time (Fig. 4.1).

Applications of Two-Dimensional Echocardiography

- Demonstrating cardiac lesions
- Demonstrating abnormal cardiac chamber geometry
- Demonstrating abnormal cardiac wall motion
- Demonstrating pleural and pericardial effusions

M-MODE ECHOCARDIOGRAPHY

- M-mode ("motion-mode") echocardiography uses a single narrow beam of ultrasound, with the positioning of a cursor guided by a 2D image to display cardiac tissues according to depth over time (Fig. 4.2). Historically M-mode echocardiography was used for the measurement of cardiac chamber dimensions because of its superior time resolution, but 2D echocardiography is also used as frame rates have increased.

Applications of M-Mode Echocardiography

- Time-dependent measurements (left heart chamber dimensions at end diastole and end systole; left ventricular wall motion)

DOPPLER ECHOCARDIOGRAPHY

- Doppler echocardiography uses the Doppler principle to estimate the *velocity* and *direction* of blood flow or myocardial motion.
- The change in frequency (or Doppler shift) when an ultrasound wave is reflected by a moving red blood cell depends on the transmitted frequency and the direction and velocity of the red blood cells. If the transmitted frequency and the received frequency are known, the blood flow velocity can be calculated.
- Aligning the beam parallel with flow (or at least within 20 degrees of flow) is essential to avoid underestimating blood flow velocity.
- There are several types of Doppler echocardiography, with the direction and velocity of motion displayed in different formats or modes.

Spectral Doppler Echocardiography

- With spectral Doppler, the velocity of blood flow is calculated within a region of interest, selected by aligning a cursor parallel with flow on a 2D echocardiographic image and then plotted as a graph of blood flow velocity against time (Fig. 4.3).
- The angle of interrogation of the Doppler beam can be seen in two dimensions with 2D Doppler, but further adjustments in probe position are still necessary to optimize alignment in the third plane.
- Blood flow velocities toward the transducer are displayed as positive velocity signals (above the baseline), and blood flow velocities away from the transducer are displayed as negative (below the baseline).
- Spectral Doppler can be further subdivided into pulsed wave (PW) Doppler and continuous wave (CW) Doppler.

Pulsed-Wave Doppler

- In PW Doppler, the ultrasound waves are transmitted as pulses of waves, with the transducer acting at different times as a receiver or transmitter of ultrasound

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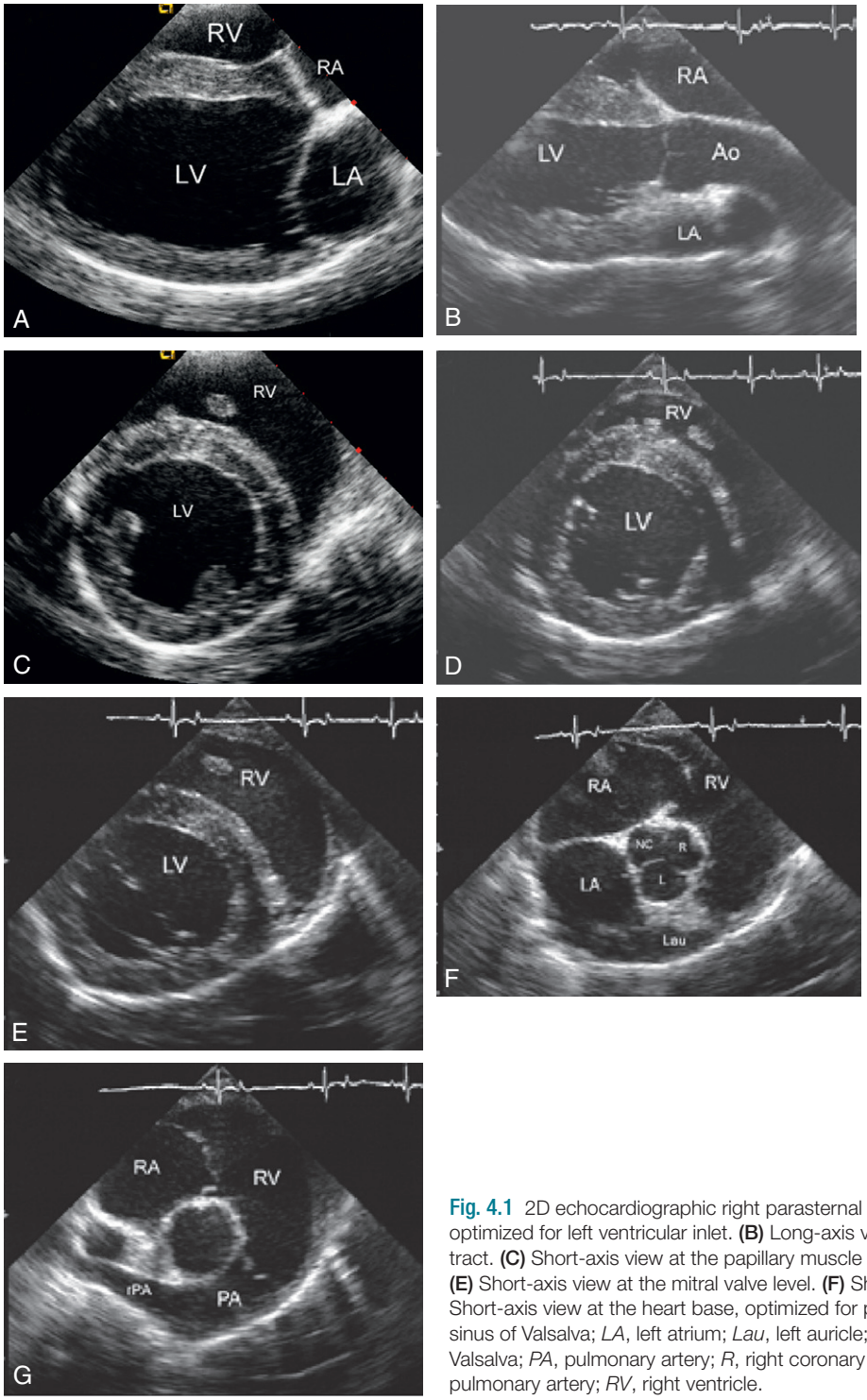


Fig. 4.1 2D echocardiographic right parasternal (RP) views. **(A)** Long-axis four-chamber view optimized for left ventricular inlet. **(B)** Long-axis view optimized for left ventricular outflow tract. **(C)** Short-axis view at the papillary muscle level. **(D)** Short-axis view at the chordal level. **(E)** Short-axis view at the mitral valve level. **(F)** Short-axis view at the aortic valve level. **(G)** Short-axis view at the heart base, optimized for pulmonary artery. *Ao*, Aorta; *L*, left coronary sinus of Valsalva; *LA*, left atrium; *Lau*, left auricle; *LV*, left ventricle; *NC*, noncoronary sinus of Valsalva; *PA*, pulmonary artery; *R*, right coronary sinus of Valsalva; *RA*, right atrium; *rPA*, right pulmonary artery; *RV*, right ventricle.

waves, allowing the interrogation of blood flow velocities within a specific region of interest.

- This region of interest is represented as a *sample volume* on the cursor.
- PW Doppler has limitations in the maximum velocities that can be displayed: high velocities result in "aliasing," where blood flow will be displayed as both positive and negative velocities (i.e., the signal wraps around the baseline).
- The velocity at which aliasing will occur depends on the *Nyquist limit* (half the pulse repetition frequency). Aliasing is most likely to occur with higher velocities, higher transducer frequencies, and with increasing depth.

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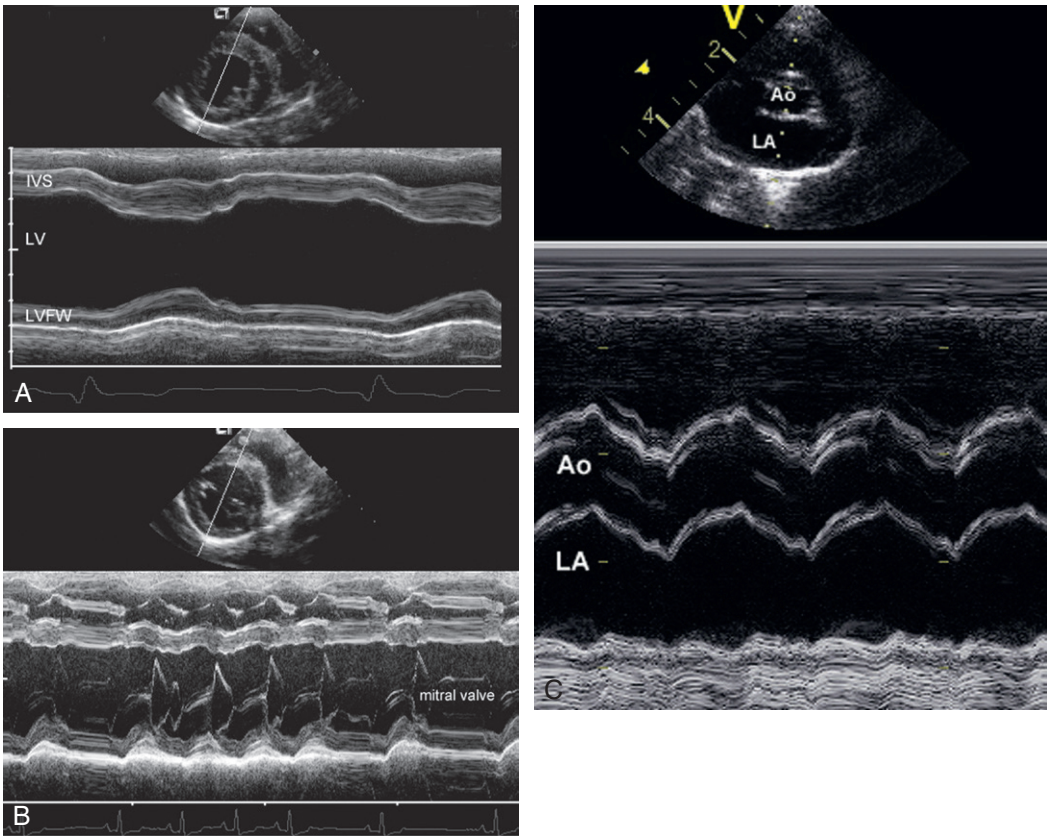


Fig. 4.2 M-mode echocardiograms from right parasternal short-axis views: **(A)** at the papillary muscle level; **(B)** at the mitral valve level; (C) at the aortic (*Ao*) and left atrial (*LA*) level in a cat. *IVS*, Interventricular septum; *LV*, left ventricle; *LVFW*, left ventricular free wall.

Continuous-Wave (CW) Doppler

- With CW Doppler, echocardiography, ultrasound waves can be transmitted and received simultaneously. This allows much higher velocities to be displayed, but recorded velocities may originate from any point along the cursor (i.e., there is *range ambiguity*).

Color Flow Doppler Echocardiography

- Color flow Doppler echocardiography represents the velocity and direction of blood flow in color, superimposed on a black-and-white 2D image (Fig. 4.4). In effect, the color is displayed within a very large sample volume superimposed on the 2D image. Blood flow away from the transducer is shown in blue, and blood flow toward the transducer is displayed in red ("BART": *b*lue *a*way, *r*ed *t*oward). Disturbed or turbulent flow may be displayed in green or yellow. Aliasing may also occur in color flow Doppler echocardiography with high-velocity blood flow.

Tissue Doppler Imaging

- More sophisticated machines may include facilities for recording the velocity of myocardial motion (tissue Doppler imaging or TDI). The signals reflected by the moving myocardium have high amplitude but low velocity. These myocardial velocities can be displayed in spectral format, or as a color display (Fig. 4.5E). Although TDI indices are less likely to be affected by preload and afterload than conventional echocardiographic indices, they are not completely independent of load.

Applications of Doppler Echocardiography

Doppler echocardiography is used to display abnormal direction or velocity of blood flow and to indicate the origin of turbulent blood flow. Doppler echocardiography can also be used to estimate differences in intracardiac pressures between chambers and assess systolic and diastolic function.

- *Abnormal blood flow direction* is associated with valvular regurgitation or intracardiac shunts.
- *Abnormal blood flow velocity* can be used to quantify abnormal chamber pressures because the velocity of blood flow across an orifice is chiefly determined by the difference in pressure (pressure gradient [PG]). If there is a large difference in the pressures of the chambers on each side of a valve, then the velocity of flow across the valve will be high. For example, during systole the pressure in the left ventricle is very high, and the pressure in the left atrium is very low. If the mitral valve is incompetent and regurgitation occurs, then the velocity of the regurgitant

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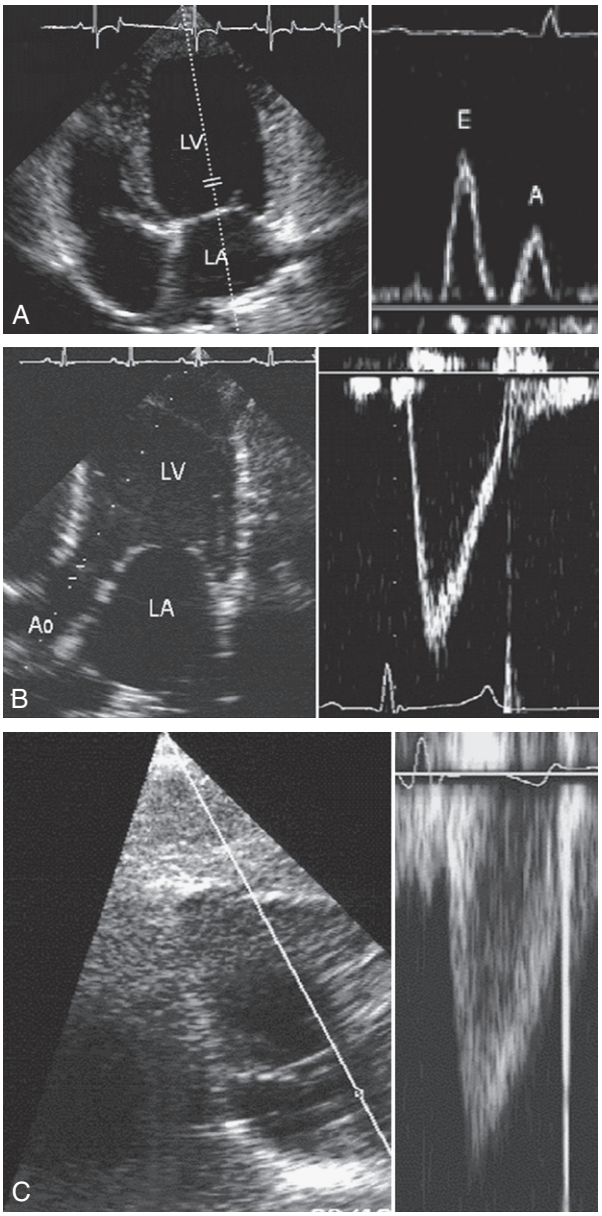


Fig. 4.3 Doppler echocardiographic studies. **(A)** Mitral inflow obtained from the left caudal parasternal (apical) four-chamber view, with the cursor placed parallel with mitral inflow, and a small sample volume placed at the tips of the mitral valve leaflets when the mitral valve is open. The typical spectral Doppler waveform of mitral inflow displays an early diastolic (*E*) wave and an atrial contraction (*A*) wave of filling. **(B)** Aortic flow obtained from the left caudal parasternal (apical) "five-chamber" view, showing left ventricular outflow tract and placement of pulsed-wave sample volume in ascending aorta. The typical spectral Doppler waveform of aortic flow is displayed. **(C)** Aortic flow obtained from the subcostal view, showing continuous-wave cursor positioned in ascending aorta. The typical spectral Doppler waveform of aortic flow is displayed.

jet traveling from the left ventricle to the left atrium will be very high, reflecting the large difference in pressures. Conversely, for much of diastole, the pressure in the left atrium is only slightly higher than the pressure in the left ventricle so that the velocity of the early filling (*E*) wave is relatively low. This velocity information can therefore be used to derive information about intracardiac pressures.

- *Turbulent blood flow* is relatively uncommon in the normal heart, but when present, it most likely occurs where velocity is highest (i.e., within the aorta). Blood flow will be turbulent whenever velocity is high, and valvular regurgitation or stenosis almost inevitably results in turbulent blood flow. Turbulent blood flow may be displayed in a different color (green/yellow) or color distribution (mosaic pattern); this is termed *variance*. In most cases, turbulent flow will be present when a murmur is audible, making Doppler echocardiography particularly useful in conditions where a murmur is present.
- *Measurement of a PG* is based on the velocity of blood flow. The modified Bernoulli equation is used to calculate the difference in pressure across a valve or between chambers: $PG = [4 \times (\text{blood flow velocity})^2]$.

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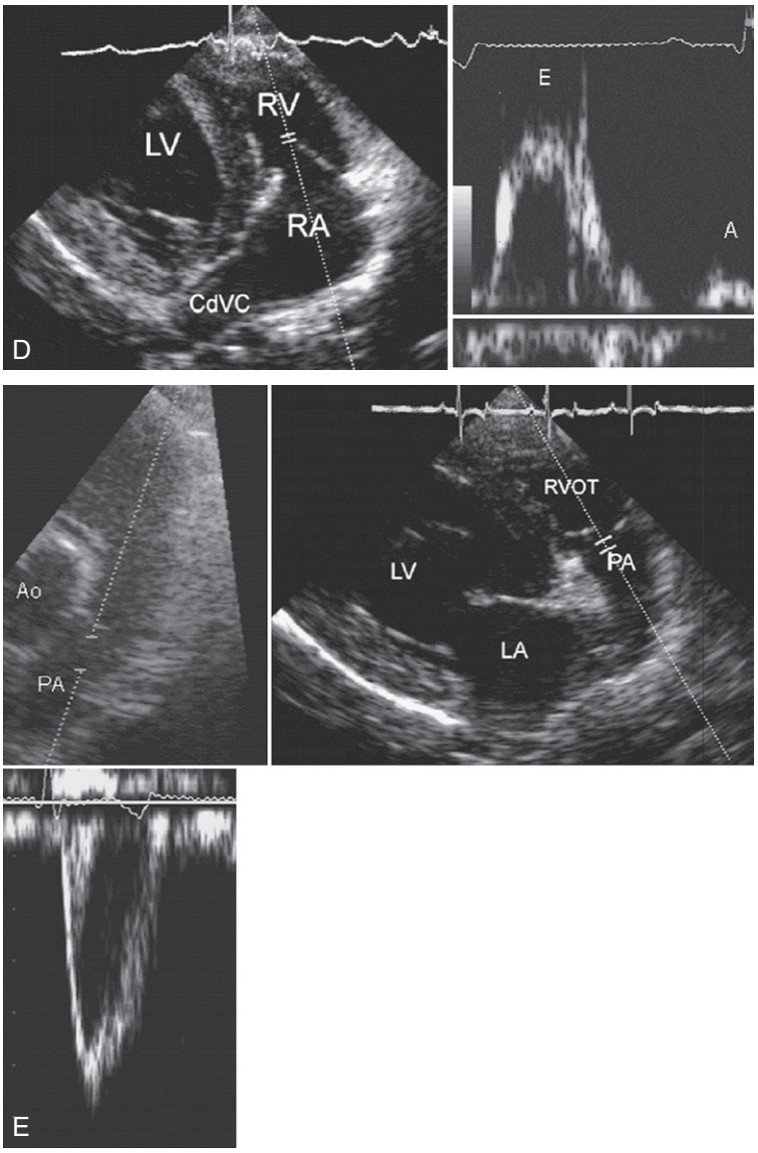


Fig. 4.3, cont'd **(D)** Tricuspid valve flow obtained from the left cranial parasternal view optimized for right ventricular inflow. The pulsed-wave sample volume is placed at the tricuspid leaflet tips, with the probe in a cranial position. The spectral waveform is similar to that of mitral inflow, although sometimes an additional systolic forward flow wave is recorded. **(E)** Pulmonary artery flow from the right parasternal short-axis view and from the left cranial parasternal view optimized for the pulmonary artery. The typical spectral Doppler waveform of pulmonary artery flow is displayed. *Ao*, Aorta; *CdCV*, caudal vena cava; *LA*, left atrium; *LV*, left ventricle; *PA*, pulmonary artery; *RA*, right atrium; *RV*, right ventricle; *RVOT*, right ventricular outflow tract.

- *Diastolic dysfunction* can also be assessed with Doppler echocardiography, although many measurements are influenced by loading conditions. Evaluation of transmitral and pulmonary venous flow in conjunction with TDI indices can suggest abnormal left ventricular (LV) relaxation and filling pressures.

ECHOCARDIOGRAPHIC TECHNIQUE

EQUIPMENT

Echocardiography Machines

- Ultrasound machines aimed at the echocardiography market will be capable of 2D and M-mode imaging, and most will also have the facility for Doppler echocardiography (although not always both PW and CW spectral Doppler). Usually appropriate software for 2D and M-mode measurements are also included. Image quality remains the most important feature, and appropriate transducer choice is also essential. High-specification machines may have additional options, such as TDI or even 3D echocardiography.
- Most echocardiography machines have ECG cables, allowing a concurrent ECG to run throughout the scan. This helps with the accurate timing of measurements within the cardiac cycle and with the interpretation of blood flow patterns. It also allows screening for intermittent arrhythmias.

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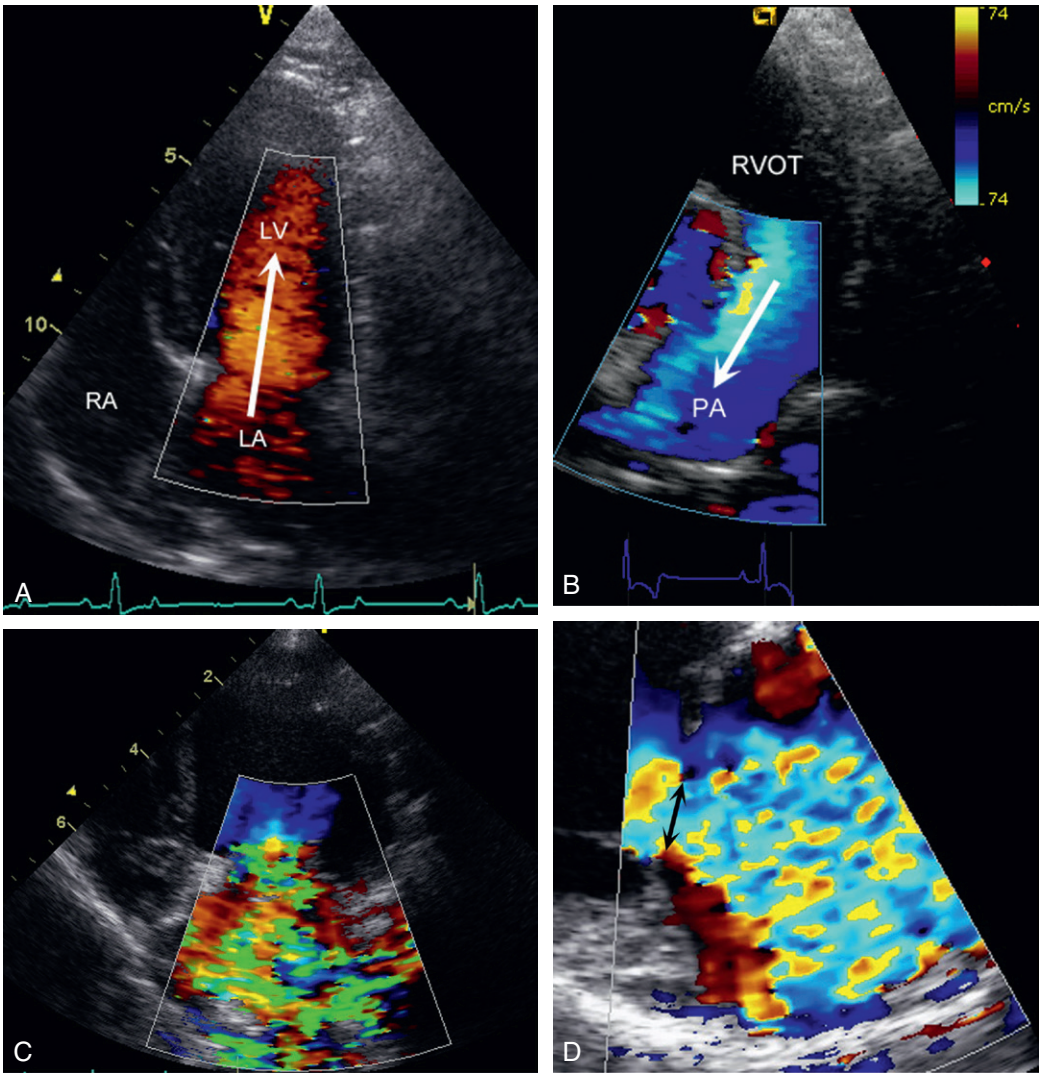


Fig. 4.4 Color flow Doppler echocardiographic images. **(A)** Normal mitral inflow in a left caudal parasternal view, showing blood flow toward the transducer in red. **(B)** Normal pulmonary artery flow in a right parasternal short-axis view, with blood flow away from the transducer shown in blue (a few pixels are coded as yellow because of "aliasing" when blood flow velocity exceeds the Nyquist limit; in this case, at >74 cm/s). **(C)** Severe mitral regurgitation in a left apical four chamber, with a large color jet of mitral regurgitation showing variance (disturbed flow) in green. **(D)** Right parasternal long-axis view of a dog with severe mitral regurgitation, showing a large vena contracta diameter (*black arrow*). This corresponds to the width of the mitral regurgitant orifice. *LA*, Left atrium; *LV*, left ventricle; *PA*, pulmonary artery; *RA*, right atrium; *RVOT*, right ventricular outflow tract.

- **Transducers** Linear probes are unsuitable for echocardiography because of the limited acoustic window available between the ribs.
- Sector probes produce a fan-shaped arc of ultrasound waves, so they will provide a wide image of the far field from a small acoustic window. These are ideal for cardiac applications.
- Types of sector probes include the following:
  - *Curvilinear* probes are similar to linear probes but have a rounded surface and perform better than linear probes for cardiac imaging. Rib shadows are a problem, however, when they are rotated through 90 degrees.
  - *Phased array* transducers have an array of crystals fired electronically in sequence to produce the fan-shaped wavefront. Generally the footprint is rectangular. Wider sector angles are possible, as well as better beam focusing.
- There is always a trade-off between penetrating power and resolution. Low-frequency transducers have high penetrating power but poor resolution, whereas high-frequency transducers have low penetrating power but higher resolution.
- Most transducers have a wide frequency bandwidth that allows a single probe to operate at more than one frequency, but ideal results are not necessarily obtained across the entire bandwidth.
- Higher-end machines may have *harmonic (or octave) imaging*, which results in a better signal-to-noise ratio.

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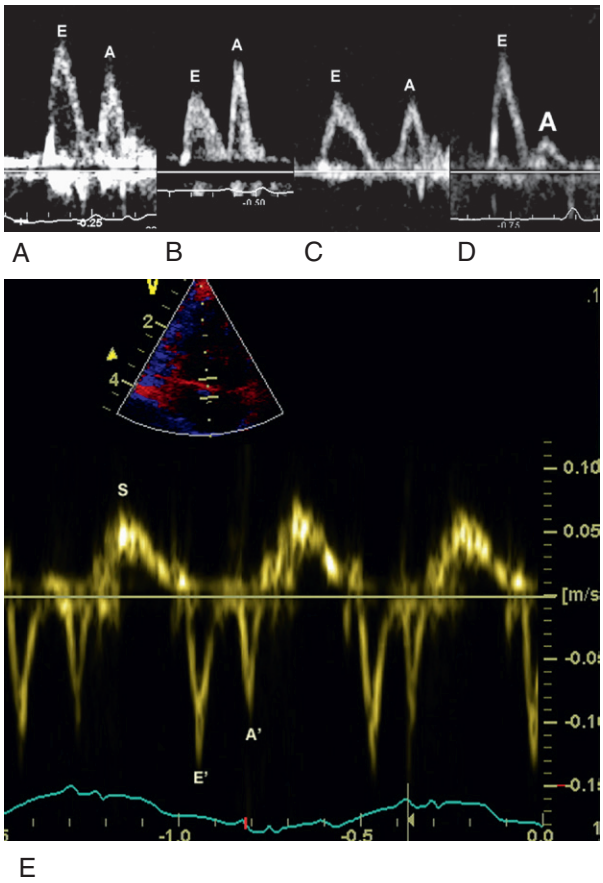


Fig. 4.5 Spectral Doppler transmitral flow showing different diastolic filling patterns. **(A)** *Normal* transmitral flow pattern demonstrating an early filling wave (*E*) with higher velocity than the atrial contraction wave (*A*). **(B)** *Delayed relaxation* transmitral flow pattern (reduced E wave amplitude and an enhanced A wave). **(C)** *Pseudonormal* transmitral flow pattern (normal E:A velocity ratio from delayed relaxation with increased left atrial [LA] pressures). **(D)** *Restrictive* transmitral flow pattern. High E wave amplitude and diminished A wave from high LA pressures and decreased LV compliance (sometimes with atrial systolic dysfunction). **(E)** Pulsed-wave tissue Doppler imaging (TDI) of mitral annulus velocity, displaying a systolic wave (*S*), early diastolic wave (*E'*), and atrial wave (*A'*).

Key Point

Although the information that can be obtained from echocardiography is greatly affected by the ultrasound machine and transducer, the echocardiographer's level of skill and training have the greatest impact.

- **Echo Table** The ideal echocardiography table should be covered with a comfortable but hygienic surface, and it is helpful if the table can be raised or lowered.
- Purpose-built echocardiography tables usually have a wedge-shaped (or rectangular) cutout to allow the probe to be positioned on the lowermost chest wall when the animal is in lateral recumbency.
- An alternative option is to have a custom-made table-top platform with a cutout that can be positioned on top of a standard examination table.
- Some clinicians prefer to scan with the animal standing or sitting.

PATIENT PREPARATION

Clipping

- Unless the hair coat is very sparse, dogs will usually need to be clipped for echocardiography. Ultrasound gel should be used generously, and in cats that object to the clippers this might provide adequate acoustic contact, but there may be some delay before the image is optimized if air is trapped in the coat.

Sedation

- Echocardiography can be performed without sedation in calm animals and with skilled assistants, but stress should be avoided as much as possible. Trazodone (in dogs) and gabapentin (in cats) administered at home can help to prevent stress, and may be sufficient alone. If not, butorphanol can be added. In severely uncooperative patients, alfaxalone may be given in addition. General anesthesia and alpha-2 agonists can affect systolic function and may affect ventricular dimensions, so should be avoided.

ECHOCARDIOGRAPHIC VIEWS

TWO-DIMENSIONAL IMAGING

- A consistent technique should be adopted so that the same views are obtained in the same sequence with each study. Some views may be more difficult to obtain in some patients. The most common imaging planes are the *right parasternal (RP) views* obtained with the transducer on the right side of the chest (see Fig. 4.1). Views obtained from the left apical windows (*caudal and cranial left parasternal views*; see Fig. 4.3) are mainly used for Doppler echocardiography applications, but as with radiography, it is helpful to confirm abnormalities in more than one view.

DOPPLER IMAGING

- Poor alignment of the cursor with blood flow will lead to underestimation of velocity, so the highest value obtained for blood flow velocity is likely to be the most accurate. Blood flow across all four valves can be recorded from the left parasternal views, with the subcostal view preferred for aortic velocities. Color Doppler echocardiography used in the RP views can be used for screening for mitral and aortic regurgitation. The features of blood flow across each valve are listed below. Each wave of blood flow velocity graphically displayed against time is called an *envelope*.

Pulmonary Artery Flow

- Pulmonary artery flow can be recorded from an RP short-axis view or the left cranial parasternal

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position (see Fig. 4.3E). A large sample volume should be used to minimize aliasing.
- There is usually one single systolic envelope, away from the transducer (below the baseline) with a peak velocity of <1.5 m/s.
- There may be some positive flow (toward the transducer) during diastole associated with pulmonic regurgitation. A small amount of pulmonic insufficiency is common in healthy dogs and is usually considered a normal finding.

Aortic Flow
- Aortic flow is recorded from the left caudal parasternal (apical) five-chamber (see Fig. 4.3B) or from the subcostal position (see Fig. 4.3C). The subcostal view usually provides the best alignment with flow. A large sample volume should be used to minimize aliasing.
- There should be one single systolic envelope, away from the transducer (below the baseline) with peak velocities <1.7 m/s (left caudal parasternal view) or <2.0 m/s (subcostal).

Mitral Inflow
- Mitral inflow is recorded from the apical four- or two-chamber view (see Fig. 4.3A). As small a sample volume as possible should be used, with the sample volume placed at the open leaflet tips during diastole.
- Two peaks of flow are generally recorded, corresponding to early passive filling at the start of diastole (the E wave: 0.5–1.0 m/s) and filling across the mitral valve during atrial contraction (the A wave: 0.3–0.6 m/s).

Tricuspid Inflow
- Tricuspid flow is recorded in one or two intercostal spaces cranial to the position for mitral inflow (see Fig. 4.3D). As small a sample volume as possible should be used. Two peaks of flow are recorded, corresponding to early passive filling at the start of diastole (the E wave: 0.3–0.9 m/s) and filling across the tricuspid valve during atrial contraction (the A wave: 0.3–0.6 m/s).

2D ECHOCARDIOGRAPHIC MEASUREMENTS
- 2D echocardiography provides qualitative information on cardiac chamber geometry and structural lesions, as well as quantitative assessment of cardiac dimensions and function. Echocardiography can also be used to assess systolic and diastolic performance, valve function, and hemodynamic estimates (e.g., intracardiac pressures).
- Echocardiographic measurements should be obtained from three to five consecutive cardiac cycles (or more with atrial fibrillation) to obtain an average. Ectopic beats and postectopic beats should be avoided for measurements.

LEFT VENTRICULAR DIAMETER
- LV diameter is typically measured from M-mode images (Fig. 4.6A).

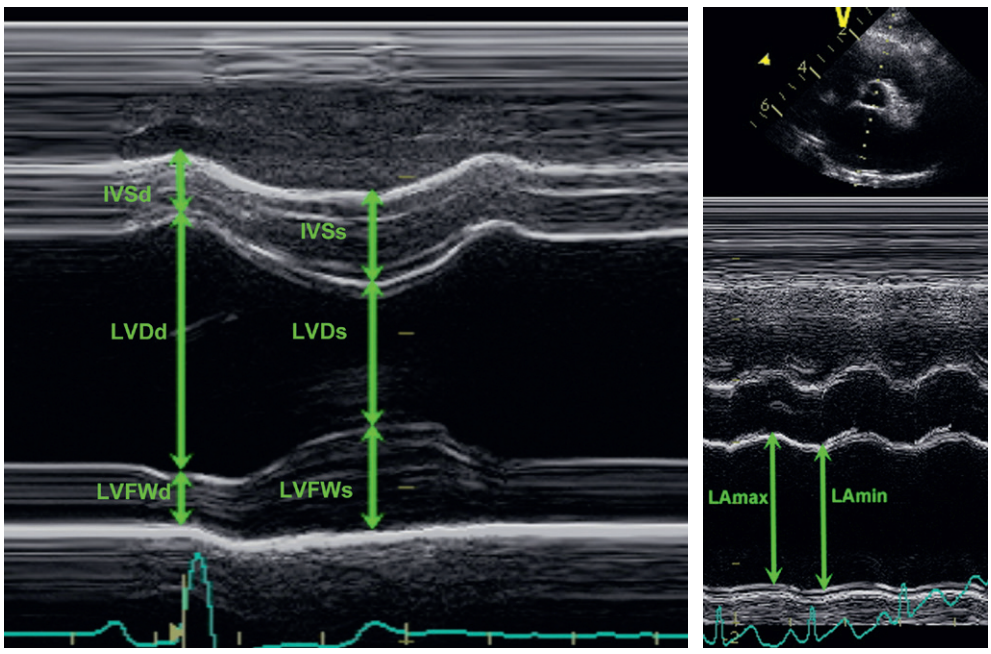


Fig. 4.6 M-mode measurements. **(A)** M-mode at papillary muscle level, showing measurements at end diastole and end systole. **(B)** M-mode at the aortic level in a cat, showing measurement of left atrial fractional shortening (LAFS%). *IVSd*, Septal thickness in diastole; *IVSs*, septal thickness in systole; *LAmax*, maximal left atrial diameter; *LAmin*, minimal left atrial diameter; *LVDd*, left ventricular diameter in diastole; *LVDs*, left ventricular diameter in systole; *LVFWd*, left ventricular free wall in diastole; *LVFWs*, left ventricular free wall in systole.

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M-Mode Measurement Technique

- Measure from the "leading edge" of the right ventricular (RV) endocardial border of the interventricular septum to the "leading edge" of the LV endocardial border (see Fig. 4.6A).
- For the LV free wall, measure from the leading edge of the LV endocardial border to the leading edge of the epicardium/pericardium.
- Diastolic measurements are made at the peak of the R wave on the ECG.
- Systolic measurements are made at peak excursion of the interventricular septum.

Problems With LV Diameter Measurements

- Oblique views may result in diameter inaccuracies.
- Because of the difficulties in establishing normal reference intervals for a widely varying canine population, various strategies can be used to take account of variation in body size:
  - Allometric scaling, where LVDd is normalized for body weight (LVDdN) by dividing the measured end diastolic diameter by [body weight]$^{0.299}$
  - For a normalized LV end-diastolic diameter measured with M-mode (LVDdN^{MM}), LVDdN is predicted to fall between 1.2 and 1.64 in 95% of healthy dogs.
  - Ratio indices, with LVDd divided by aortic diameter.
  - Breed-specific reference intervals.

Left Ventricular Wall Thickness

- Detection of LV hypertrophy is more important in cats than in dogs because of the high prevalence of hypertrophic cardiomyopathy (HCM) in cats. M-mode imaging planes may miss localized areas of hypertrophy or erroneously include papillary muscles, so wall thickness is best measured from 2D images if the frame rate is adequate (i.e., >60 frames/s). 2D echocardiography has the advantage of allowing measurements irrespective of the location of hypertrophy.
- Measurements *must* be made at end diastole, when the walls are at their thinnest. There is a risk of overestimating wall thickness from 2D images with machines with slow frame rates.
- LV septal and free wall thickness should be measured in RP long-axis and RP short-axis 2D views, taking care to avoid including the linear endocardial structures ("false tendons") that are often present.
- LV hypertrophy is usually defined in cats as an end-diastolic septal or free wall thickness of ≥6 mm, and LV wall thickness in most normal cats will be <5 mm, with a "gray zone" of uncertainty between these values. There is a small increase in LV wall thickness with increasing body weight.

Left Atrial Diameter

- Left atrial (LA) size can be measured from a 2D short-axis view and a 2D long-axis view.

Right parasternal short-axis view

- The ideal RP short-axis view includes the aortic valve with leaflets visible throughout the cardiac cycle, the left atrial appendage, and a pulmonary vein entering the left atrium.
- The maximum LA diameter is measured in early diastole at aortic valve closure, although the aortic leaflets can be difficult to image at this phase of the cardiac cycle.
- The aortic annulus is measured from inner edge to inner edge, from the middle of the right coronary cusp to the commissures of the left and noncoronary cusps.
- The maximum (early diastolic) LA diameter/aortic diameter ratio (LA/Ao) should be <1.6.
- The minimum LA diameter is measured in late diastole after atrial contraction and before the aortic valve opens. The LA diameter is measured from the trailing edge of the aortic annulus to the LA free wall. The presence of a pulmonary vein can complicate identification of the far LA wall (Fig. 4.7A).

Right parasternal long-axis views

- The aortic and LA diameters are measured from different RP views.
- The LA diameter is measured at end systole in an RP long-axis four-chamber view, from the midpoint of the interatrial septum to the midpoint of the LA free wall, using an inner edge to inner edge technique (see Fig. 4.7B).
- The aortic diameter is measured from an RP long-axis five-chamber view as the distance between the open aortic leaflets during systole (see Fig. 4.7C).
- The RP long-axis LA/Ao ratio should be =2.4.
- In cats, there is no need to normalize the RP long-axis LA diameter to the aortic diameter; the absolute diameter in this view should be ≥1.6 cm (note, however, that this is a technically difficult view to achieve in cats).
- LA volumes can also be measured using the Simpson's method of discs (SMOD).

Key Point

LA size is one of the most powerful predictors of left-sided heart failure, and it is an essential measurement in staging myxomatous mitral valve disease and feline cardiomyopathy. It is also a key measurement in the assessment of patients with respiratory distress, where the presence of LA enlargement increases suspicion of congestive heart failure.

Left Atrial Fractional Shortening

- LA fractional shortening is also a very important prognostic indicator in cats and is relatively easy to measure from M-mode images at the level of the

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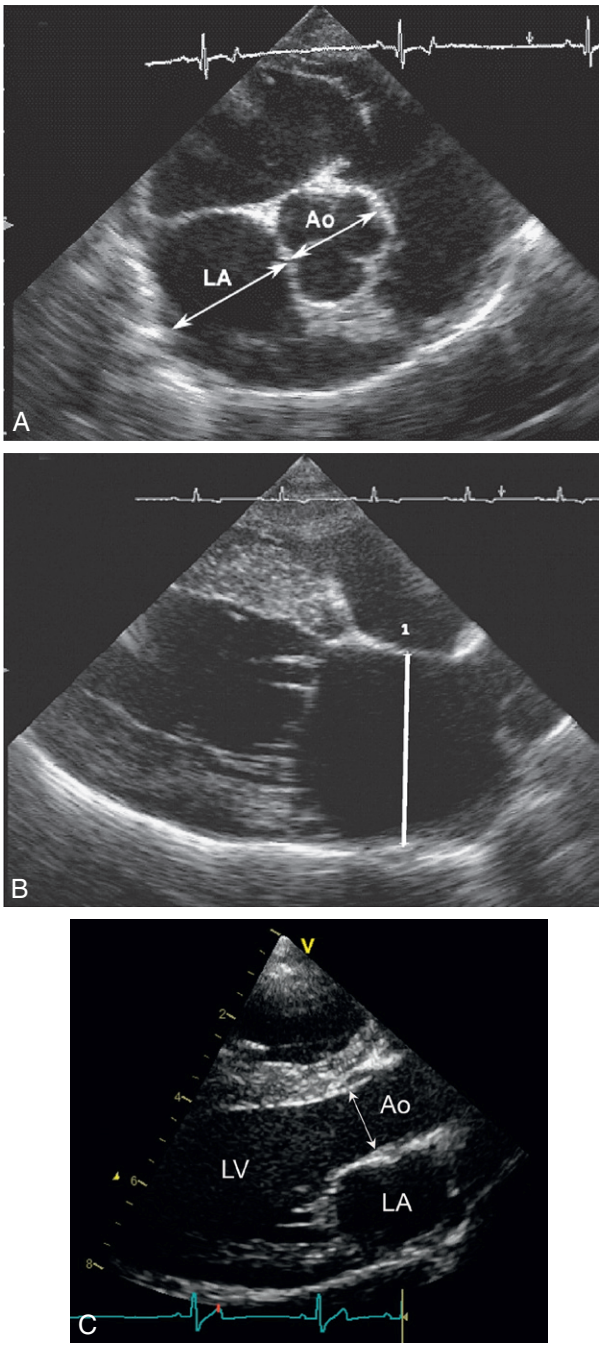


Fig. 4.7 **(A)** Measurement of the left atrial (*LA*) and aortic (*Ao*) diameters from the right parasternal short-axis view **(B)** Measurement of the LA diameter from the right parasternal long-axis view in systole optimized for the left ventricular inlet. **(C)**. Right parasternal long-axis view in systole optimized for the left ventricular outflow tract. The aortic diameter is measured from the inner edge to inner edge of the aortic leaflets.

aortic valve in an RP short-axis view (see Fig. 4.6B). LA fractional shortening (LAFS%) is the percentage change in LA diameter, measured from the maximal LA diameter (LAmax) and the minimal LA diameter (LAmin). It is calculated as follows: **LAFS % = [(LAmax – LAmin)/LAmax] x 100.**

- LAFS% in normal cats is generally >24%.

Left Ventricular Systolic Function

- Many different echocardiographic measurements can be made to assess systolic performance, but all are affected to some extent by ventricular loading conditions (i.e., abnormal preload and afterload).
- Common indices used for assessing LV systolic function include LV fractional shortening (LVFS%), LV end-systolic volume index (LVESV-I), and LV ejection fraction (EF%).

LV fractional shortening

- LV fractional shortening (FS%) is the most commonly used echocardiographic index of LV systolic function (Fig. 4.8A) and is calculated using the following formula: **FS% = [(LVDd – LVDs)/LVDd] x 100.**
- FS% will increase with improved contractility, increased preload, or decreased afterload, but it only assesses ventricular shortening in the minor axis dimension.
- Normal reported mean values range from 25% to 40%, with lower values in larger dogs and some athletic dogs.

LV Volumes

- LV volumes should be measured from 2D images, *not* using M-mode (such as with the Teichholz formula).
- A modified SMOD is generally used to calculate LV end-diastolic volume (LVEDV) and LV end-systolic volume (LVESV) (see Fig. 4.8B).
- The endocardial borders of the LV are traced in an RP long-axis or left caudal parasternal view. Ideally both should be measured, selecting the view with the largest volume.
- LV volumes have traditionally been indexed to body surface area, but the relationship with body weight in kilograms is also linear and easier to calculate (LVEDV-I and LVESV-I in milliliters per kilogram). Median LVEDV-I is 2.2 mL/kg (90% CI 1.3–3.3) in healthy non-sighthounds and 3 mL/kg (90% CI 1.9–4.2) in healthy sighthounds. Median LVESV-I is 0.9 mL/kg (90% CI 0.3–1.5) in healthy non-sighthounds and 1.4 mL/kg (90% CI 0.7–2.1) in healthy sighthounds.

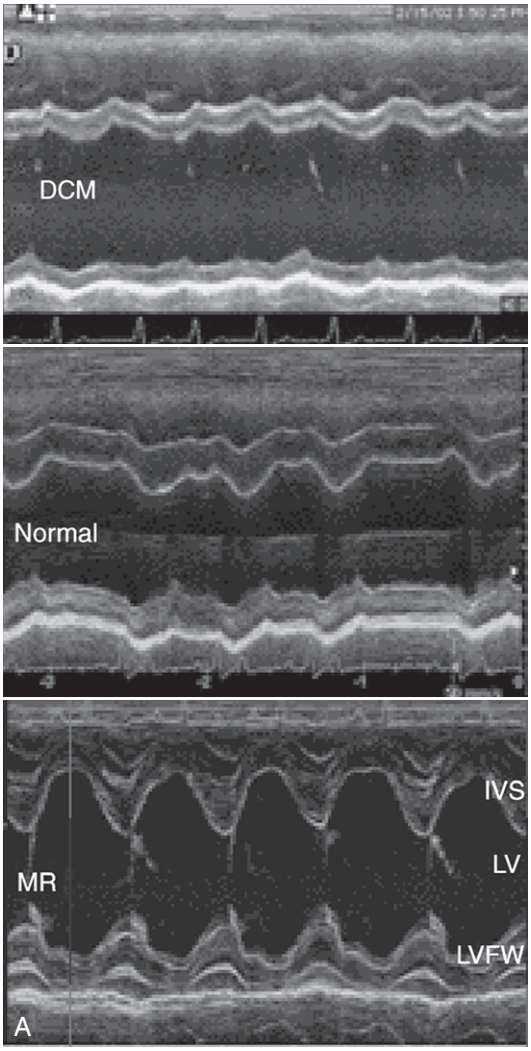
Ejection fraction

- Calculation of LV volumes allows calculation of ejection fraction (EF%):
- LV EF% = [(LVEDV – LVESV)/LVEDV] × 100
- Median EF% is 57% (90% CI 41%–75%) in healthy non-sighthounds and 53% (90% CI 41%–65%) in healthy sighthounds.

Valve Function

- Color Doppler can be used as a quick screen for valve function, but caution should be used in determining the severity of valvular regurgitation by regurgitant

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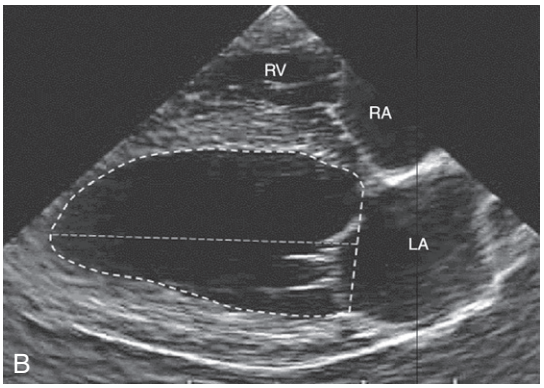


Fig. 4.8 Echocardiographic evaluation of ventricular systolic function. **(A)** M-mode of ventricular hypokinesis from a dog with dilated cardiomyopathy (*DCM*) (*top panel*), normal ventricular motion from a healthy dog (*middle panel*), and ventricular hyperkinesis from a dog with mitral regurgitation (*MR*) (*bottom panel*). *IVS*, Interventricular septum; *LV*, left ventricle; *LVFW*, left ventricular free wall. **(B)** Measurement of left ventricular volume from a right parasternal long-axis view. The endocardial borders are traced, with the left ventricular length measured from a line drawn across the mitral annulus to the apex. *LA*, Left atrium; *RA*, right atrium; *RV*, right ventricle.

jet size alone or in relying on the presence of turbulent signals to identify valvular stenosis.

Mitral Regurgitation

- 2D echocardiography is very important in the assessment of mitral regurgitation. It can be used to identify abnormal mitral valve morphology and motion and increase chamber dimensions.
- Mitral regurgitation can be assumed to be severe when the following criteria are present:
  - Dilated left atrium and left ventricle (in myxomatous mitral valve disease)
  - Increased mitral E wave velocity
  - Increased width of the vena contracta (see Fig. 4.4D)
  - Presence of a large proximal flow convergence region
  - High-intensity CW spectral Doppler signal
- Large regurgitant jet area compared with LA area.
- Similar criteria apply to tricuspid regurgitation on the right side of the heart.

Aortic Regurgitation

Less common than mitral regurgitation, aortic regurgitation is considered severe when the following criteria are present:

- Large aortic regurgitation jet diameter compared with aortic valve area in a short-axis view
- Increased width of vena contracta
- Rapid deceleration of aortic regurgitation signal on spectral Doppler signal

Aortic and Pulmonic Stenosis

- The severity of aortic or pulmonic stenosis is generally assessed in terms of the magnitude of the PG across the stenosis.
- The greater the degree of stenosis, the higher the blood flow velocity beyond the stenotic valve. A PG across a valve or between chambers can be estimated with the modified Bernoulli equation: PG (mm Hg) = 4 × (velocity of blood flow beyond the stenosis^2).

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Diastolic Function

- Diastolic function is complex and includes multiple factors that influence ventricular filling.
- Clinically relevant aspects of diastolic function include LV relaxation, LV compliance, LA pressures, LA systolic function, heart rate and rhythm, and pericardial constraint.
- No single echocardiographic measurement can provide a complete overview of diastolic function, but some clinically important aspects can be identified with echocardiography.
- At its simplest, echocardiography can reveal LA enlargement, implying increased LA pressures. A range of Doppler measurements can be used to give a composite assessment of diastolic function or highlight specific aspects.

Transmitral Flow

- Transmitral flow reflects the instantaneous PG across the mitral valve.
- During diastole, there is a forward flow across the mitral valve.
- The initial wave of filling (the E wave) will be delayed if the rate of LV relaxation is slow, and the E wave velocity will increase if LA pressures are increased.
- The second wave of filling (the A wave) occurs with atrial contraction, and usually has a smaller contribution to LV filling (the A wave velocity is usually low).
- There is a progressive change in the ventricular filling pattern with advancing disease across a range of underlying cardiac diseases (see Fig. 4.5A–D as the severity of diastolic function increases):
  - *Delayed LV relaxation pattern:* normal LA pressures (E wave < A wave)
  - *Pseudonormal pattern*: LA pressures increase but LV relaxation is still delayed (E wave > A wave).
  - *Restrictive pattern*: very high E wave velocities (from very high LA pressures) and very low A wave velocities (E wave >> A wave).
- Pulmonary venous flow patterns can be used when transmitral E and A waves are fused.

COMMON ACQUIRED CARDIAC CONDITIONS

DEGENERATIVE (MYXOMATOUS) MITRAL VALVE DISEASE

- Myxomatous mitral valve disease (MMVD) is relatively straightforward to diagnose in older small-breed dogs, but echocardiography plays a vital role in staging MMVD.

Role of Echocardiography in MMVD

- Diagnosis
  - Prolapse of thickened mitral leaflets (Fig. 4.9)

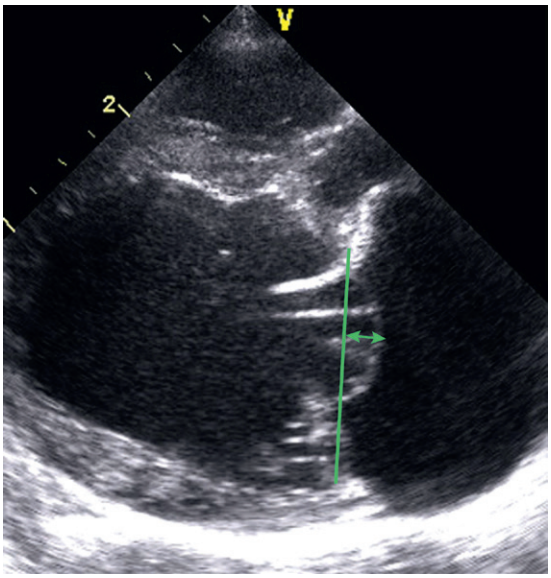


Fig. 4.9 Myxomatous mitral valve disease. Right parasternal long-axis view showing thickened, distorted mitral leaflets with prolapse of the anterior leaflet (*green arrow*, extending beyond line drawn across mitral annulus).

  - Color Doppler jet of mitral regurgitation (often eccentric)
  - Hyperdynamic LV wall motion with severe mitral regurgitation (see Fig. 4.8A)
  - "Flail leaflet" following the rupture of a chordae tendineae
- Staging subclinical mitral valve disease
  - Asymptomatic dogs with a murmur of mitral regurgitation, typical 2D lesions of MMVD and normal left heart dimensions (stage B1) remain free of clinical signs for longer than asymptomatic dogs with left heart enlargement (stage B2).
  - Dogs with stage B2 MMVD are likely to benefit from administration of pimobendan.
- Identifying pulmonary hypertension
  - Echocardiography is the main tool for diagnosing pulmonary hypertension of any cause. Dogs with high-velocity tricuspid regurgitation in the absence of RV outflow tract obstruction are very likely to have pulmonary hypertension.

DILATED CARDIOMYOPATHY

- In the presence of clinical signs of congestive heart failure, dilated cardiomyopathy (DCM) is a relatively easy diagnosis to make, with global hypokinesis of a dilated heart in the absence of any other lesions (Fig. 4.10). Occult DCM is more difficult, and caution is advised when diagnosing DCM in asymptomatic dogs. DCM should not be diagnosed on the basis of a low fractional shortening value alone (especially in dogs of nonpredisposed breeds).

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Role of Echocardiography in Dilated Cardiomyopathy

- Diagnosis
  - Dilation of LA and LV
  - ± Dilation of right atrium (RA) and right ventricle (RV)
  - Global hypokinesis of LV (decreased FS%, EF%)

HYPERTROPHIC CARDIOMYOPATHY

- HCM is very common in cats, with a wide spectrum of disease. Even in severely affected cats, HCM can be difficult to diagnose without echocardiography. Differentiating cats with mild HCM from normal cats using echocardiography can be challenging, but echocardiography is very useful for prognosis.
- Moderate to severe LA dilation is easy to recognize with echocardiography and is a powerful prognostic marker that can be used to identify cats at high risk of congestive heart failure or aortic thromboembolism (Fig. 4.11).

Role of Echocardiography in Hypertrophic Cardiomyopathy

- Diagnosis:
  - End-diastolic septal or LV free wall thickness ≥6.0 mm (ideally with two views on 2D echocardiography) in the absence of other causes of LV hypertrophy.
  - Systolic anterior motion of the mitral valve is common in cats with HCM and causes dynamic LV outflow tract obstruction.
- Prognosis
  - The most important indicators of a poor prognosis are all based on echocardiography: low LAFS%, LA dilation, LV systolic dysfunction, and extreme LV hypertrophy (≥9 mm).
  - Additional markers of increased congestive heart failure or thromboembolic risk include presence of spontaneous echocontrast ("smoke") and a restrictive diastolic filling pattern.

PERICARDIAL DISEASE

- *Pericardial effusions* can be identified as an echo-free space around the heart (not to be confused with pleural effusions).
- Tamponade is suggested by phasic collapse of the right atrial wall (Fig. 4.12).
- The heart may be affected by a number of different neoplasms that can result in pericardial effusions, and these are most easily imaged while some pericardial fluid is present.
- *Chemodectomas* generally involve the heart base and may be imaged as homogeneous soft tissue densities encircling the aortic and pulmonary artery roots.

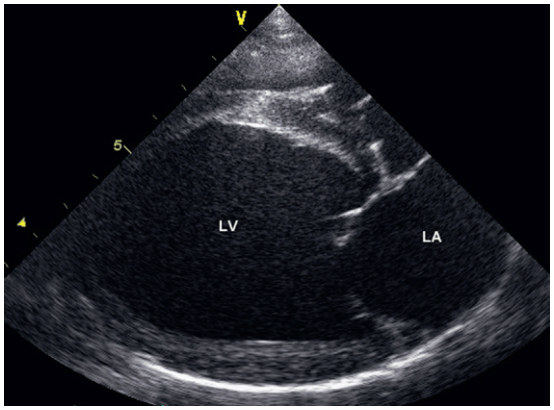


Fig. 4.10 Dilated cardiomyopathy (DCM) in a dog. Right parasternal long-axis view, showing a rounded left ventricle (*LV*) with normal mitral valve appearance (and no mitral valve prolapse). *LA*, Left atrium.

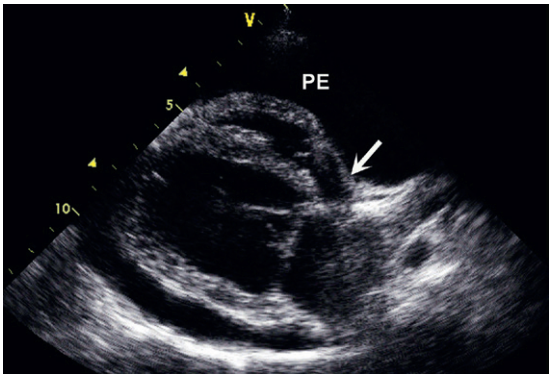


Fig. 4.12 Pericardial effusion. The echolucent space around the heart is a pericardial effusion (*PE*), which is causing collapse of the right atrium (*RA*) (cardiac tamponade, *arrow*).

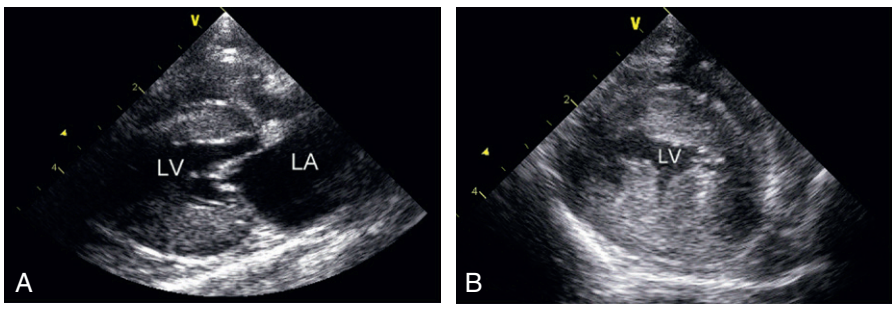


Fig. 4.11 Hypertrophic cardiomyopathy (HCM) in a cat. Right parasternal long-axis view (**A**) and short-axis view at the papillary muscle level (**B**), showing marked left ventricular (*LV*) hypertrophy and left atrial (*LA*) enlargement.

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- *Hemangiosarcomas* frequently affect the RA, although they can be difficult to image at this site. They may also infiltrate other areas of the heart (such as the septum and ventricular walls) where they may have an irregular echotexture compared with surrounding myocardium. Some pericardial effusions are considered idiopathic, although failure to identify a mass lesion does not rule out a neoplastic cause (mesotheliomas are extremely difficult to diagnose with echocardiography).

Tears in the LA can cause intrapericardial hemorrhage, usually secondary to chronic severe myomatous mitral valve disease.

FREQUENTLY ASKED QUESTIONS

CAN ANY ULTRASOUND MACHINE BE USED FOR ECHOCARDIOGRAPHY?

A. Most ultrasound machines in small animal practices can be applied to imaging the heart, although a sector probe will be required. The echocardiographer's level of training and experience is usually a bigger issue. For novice echocardiographers, a point-of-care ultrasound can be used to identify pleural effusions, pericardial effusions, and B-lines with some training and practice. Assessment of LA size is also achievable, although more challenging. Time and effort spent on obtaining (and interpreting) an RP short-axis view at the LA level is very worthwhile.

HOW IMPORTANT IS IT TO MEASURE CARDIAC CHAMBER DIMENSIONS?

A. Measurements provide a means of tracking disease progression over time, and are essential for accurate staging. However unless the recorded image is of high quality and the measurer uses a consistent technique, the results will be unreliable. For this reason, for less experienced echocardiographers, it may be more reliable to make a *qualitative* echocardiographic assessment than to base a clinical decision on suboptimal measurements.

HOW DOES ONE INTERPRET A LOW VALUE FOR FRACTIONAL SHORTENING IN AN OTHERWISE HEALTHY DOG?

A. Caution should be used when making a diagnosis of DCM in an asymptomatic dog based solely on a low fractional shortening value. Multiple other echocardiographic variables should be assessed, including evidence of left heart chamber dilation, any increase in LVESV-I, and whether the EF% is also subnormal. Normal dogs of some breeds and some athletic dogs can have a reduced FS%, but other indices of chamber size and function are within normal limits for their breed (e.g., sighthounds).

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Chapter 5

Special Diagnostic Techniques for the Evaluation of Cardiac Disease

Sonja S. Tjostheim

Many special diagnostic techniques are available for the evaluation of animals with cardiovascular disease. Ambulatory electrocardiographic (ECG) equipment is available through commercial services, continuous in-hospital ECG monitoring equipment is widely available, and several large diagnostic laboratories provide specialized clinical pathology services specific to the heart. Additionally, veterinary cardiology referral centers are increasingly available for special diagnostic techniques such as cardiac catheterization and cardiac computed tomography (CT).

CONTINUOUS IN-HOSPITAL ELECTROCARDIOGRAPHIC MONITORING

Continuous ECG monitoring is recommended for hospitalized patients at risk of heart rate or rhythm disturbances. These patients include the following:

- Patients with congestive heart failure
- Patients hospitalized with clinical signs (e.g., syncope) secondary to arrhythmia
- Patients with systemic disease that puts them at risk for arrhythmias (e.g., shock, sepsis, and gastric dilation-volvulus)

TECHNIQUE

- Continuous ECG monitoring requires an ECG unit with an oscilloscope or light-emitting diode display.
- The use of a chest lead configuration with adhesive electrode patches will minimize artifacts on the tracing while allowing the patient the most freedom for mobility.
- Clip two 2- to 3-cm square areas at the left apex and the heart base (where the apex beat is palpable on the left chest and at the heart base caudal to the right or left scapula).
- Clean and defat the area with 70% isopropyl alcohol. Allow to dry.
- Place the positive electrode patch at the left apical site and the negative electrode at the heart base site. A ground electrode may be placed at either site (Fig. 5.1).
- Apply a vest or light chest wrap to secure the electrodes and wires.
- If a multiple-lead ECG unit is being used, then use the left arm electrode for the positive electrode, the right arm electrode for the negative electrode lead, and lead I for display/recording. Alternatively, a quadtrode ECG electrode patch may be placed over the left hemithorax. The quadtrode design incorporates four electrodes into a single adhesive patch.
- If the patient is recumbent and unlikely to move, leads attached directly to the patient's limbs can be used.
- Depending on the unit available, the signal is transmitted to the central station monitor by cables or by radiotelemetry.
- Recorded continuous ECG tracings can be reviewed retrospectively by the clinician. Some units allow clinicians to access the ECG data remotely from their tablet or computer.
- Many ECG units will print out an ECG strip, which can be added to the permanent patient record.

LIMITATIONS

- Excessive patient motion may result in the displacement of electrodes or motion artifacts on the ECG.
- Adhesive patches may occasionally result in contact dermatitis.
- Electrical interference can occur from other monitoring or therapeutic machinery (e.g., fluid pumps, heating pads, and blood pressure measurement equipment).

PROVOCATIVE ELECTROCARDIOGRAPHIC TECHNIQUES

In some patients the history or baseline ECG is suggestive of pathologic arrhythmias, but a definitive diagnosis is elusive. In these cases vagal stimulation or abolition may be informative. A provocative vagal maneuver will transiently elevate parasympathetic tone. In normal animals the technique typically slows the heart rate or has no effect. However, when there is sinoatrial (SA) or atrioventricular (AV) nodal dysfunction or an abnormal sensitivity to parasympathetic tone, transient sinus arrest or significant AV block may occur. Similarly, a vagal maneuver may be diagnostic or therapeutic for an ectopic supraventricular tachycardia (SVT). A sinus tachycardia typically slows over several seconds, while some types of SVT may terminate abruptly (see Frequently Asked Questions).

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Fig. 5.1 Chest lead preparation in a Boxer. The negative lead should be placed in the upper patch behind the scapula, and the positive lead should be placed at the left apex (behind the elbow). This configuration can be used for in-hospital continuous electrocardiographic monitoring or ambulatory monitoring.

An ECG recorded immediately after exercise may demonstrate cardiac arrhythmias associated with increased sympathetic tone or abolition of arrhythmias caused by high resting vagal tone. Likewise, an atropine response test can also be used to determine if a slow heart rate is associated with elevated vagal tone (as can occur with respiratory disease, gastrointestinal disease, or neurologic disease) or primary cardiac conduction system disease. If the bradycardia is due to elevated vagal influence, then atropine administration or exercise will result in its abolition.

VAGAL MANEUVER

- The following techniques can be used separately or concurrently to elevate vagal tone. The patient should be restrained and calm so that a good-quality lead II ECG is obtained before and during the technique.

Ocular Pressure

- Moderately firm digital pressure is applied over the closed eyelid to one or both eyes for a period of 5 to 10 seconds or until significant slowing of the heart rate occurs.

Carotid Body Massage

- The carotid bodies are located in the area behind the larynx. Apply moderate digital pressure around the larynx while monitoring the ECG. Initiation of a gag response or a cough yields similar results and suggests adequate pressure has been applied.

INHIBITION OF VAGAL TONE

- Two techniques are possible for abolishing vagal tone and are useful if the patient is suspected of having a vagally mediated bradycardia. With either technique, it is important to first obtain a baseline ECG.

Postexercise Electrocardiography

- Typically strenuous leash running is used; however, the duration of exercise is not standardized. Evidence of exertion, such as panting, is sufficient to stop exercise, and an immediate postexercise ECG is obtained. A delay of as little as 30 seconds may alter results, and heavy panting can result in artifacts, making the ECG difficult to interpret.

Atropine Response Test

- Atropine (0.04 mg/kg intravenously or intramuscularly) will result in abolition of vagal influence. An ECG should be obtained 15 minutes after administration of the drug if given intravenously or 30 minutes after administration if given intramuscularly. The results of this ECG are then compared with the baseline ECG.

CLINICAL UTILITY

Vagal Maneuver

The vagal maneuver is clinically useful in two scenarios:

- In the evaluation of dogs with a history of syncope, a vagal maneuver may demonstrate sinus arrest or AV block, suggestive of parasympathetic hypersensitivity or primary nodal disease.
- In dogs with tachycardia, abrupt termination of the arrhythmia with a vagal maneuver suggests an ectopic supraventricular origin because ventricular tachycardias are not usually sensitive to vagal tone; however, a lack of response is not helpful in differentiating the origin of the tachycardia. The maneuver can also be useful to differentiate sinus tachycardia (no response or gradual slowing of heart rate) from pathologic ectopic SVT (no response or abrupt termination of tachycardia).

Postexercise Electrocardiography

- This test can be performed in the evaluation of dogs with subaortic stenosis or occult cardiomyopathy. In affected dogs the combination of exercise, myocardial disease, and left ventricular hypertrophy may result in ECG indicators of myocardial ischemia (e.g., ST segment depression or ventricular ectopy). Lack of response does not necessarily rule out heart disease. In dogs with bradycardia, abolition of the arrhythmia after exercise suggests a vagally mediated etiology. The increased availability of ambulatory ECG recordings has markedly reduced the use of postexercise ECGs in clinical practice.

Atropine Response Test

- An appropriate response to atropine may be defined as (1) development of a sinus tachycardia (heart rate greater than 140 beats/min in dogs or 240 beats/min in cats) or, failing that, at least a 50% increase in

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heart rate above the baseline heart rate; (2) resolution of AV nodal conduction block, if present.
- A positive response to atropine is suggestive of a vagally mediated bradyarrhythmia affecting the SA node, AV node, or both. In addition, this response suggests that medical management with vagolytic therapy may be effective if the bradycardia is associated with clinical signs, such as syncope or collapse, and implantation of a pacemaker is not possible. Atropine administration can induce a transient type I or II AV block. Parasympathomimetic effects occur most rapidly, and parasympatholytic effect is greatest after intravenous administration.

Key Point

Provocative ECG techniques are easily performed, relatively inexpensive, and can be very helpful for diagnostic and therapeutic purposes in animals with bradycardias and/or tachycardias.

AMBULATORY ELECTROCARDIOGRAPHY (HOLTER MONITORING AND CARDIAC EVENT RECORDING)

INDICATIONS

Routine in-hospital electrocardiograms provide only a glimpse of daily ECG activity. Moreover, arrhythmia detection may be confounded by changes in autonomic nervous system activity. A 24-hour ambulatory (Holter) recording of the ECG increases the sensitivity of arrhythmia detection. Major indications include the following:
- Detection of transient arrhythmias associated with syncope or periodic weakness
- Screening of breeds at high risk for cardiomyopathy (e.g., Boxers, Doberman Pinschers)
- Evaluation of frequency, severity, and significance of arrhythmias detected on in-hospital ECG
- Monitoring efficacy of antiarrhythmic therapy (e.g., control of heart rate in patients with chronic atrial fibrillation)
- Determining the true incidence and type of arrhythmia in patients with heart disease

TECHNIQUE

Holter monitors use a high-fidelity digital recorder to capture and store cardiac electrical activity for 24 hours. Some digital recorders can monitor the patient for up to 14 days. Two to three simultaneous ECG chest leads are typically recorded.
- Electrode sites are prepared by clipping, shaving, cleaning, and drying the chest.
- Adhesive patches are firmly adhered to the skin (see Fig. 5.1).
- A chest wrap is essential for securing electrodes, wires, and the recorder. A vest or harness can also be used (Fig. 5.2).



Fig. 5.2 A vest can be placed over the light wrap, which secures Holter or cardiac event recordermonitor electrodes in place. The vest has pockets to hold the monitor and helps keep the device in place.

- Once the monitor has been applied, the animal returns home to resume normal activity. Very small dogs and cats that find it cumbersome to ambulate with the monitor in place may respond better to hospitalization with cage restraint during the 24-hour recording session.
- Owners or caretakers should maintain a diary of significant changes in activity, such as sleeping and exercising. Any clinical signs, such as syncopal events, must be noted.
- The animal should be evaluated for at least 24 hours to assess a full circadian cycle.
- At the conclusion of the recording period, the components are removed, and the monitor is analyzed with the aid of automated computer-based software. Operator interaction and editing are essential for diagnostically accurate results.
- Consultation with a veterinary cardiologist is recommended regarding the significance of any identified arrhythmias and the need for therapy. Many normal dogs will have infrequent ventricular premature complexes or sinus pauses noted during a 24-hour period. Assessment of an arrhythmia's significance and the risk-benefit ratio of antiarrhythmic medications are essential considerations.

CARDIAC EVENT RECORDERS

The cardiac event recorder (CER) is an ambulatory microprocessor with a solid-state memory loop capable of storing portions of ECG tracings when activated. The CER is lightweight, activated by a person witnessing an event (e.g., weakness or syncope), and can be worn by small dogs and cats without restricting activity. Unlike most Holter monitors, which are worn for only 1 or 2 days, the CER can be worn for up to a week, increasing the diagnostic yield in animals with infrequent clinical signs.
- CERs can be programmed to store up to five separate, 1-minute-duration, single-channel ECGs or fewer ECGs of longer duration.

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- Recording is activated by pressing the event button on the device. The CER uses a memory loop to store the ECG (most commonly 30 seconds before activation to 30 seconds after activation); however, these times may be changed as indicated for the individual patient.
- After one or more events, the CER is detached, and the stored ECG is transmitted and downloaded to a receiving station for computer-based analysis and interpretation.

Applications

- The CER uses two adhesive electrodes in a base-apex configuration and is relatively easy to apply.
- A light chest wrap is used to secure the unit over the dorsum. As with Holter monitoring, the patient may be discharged to resume routine activity.

Use of Cardiac Event Recorders

- The CER will not store an ECG unless the activation button is pressed; therefore the event must be witnessed by the owner. If an event does not occur during the time the unit is worn, a definitive diagnosis is not possible.
- CERs may be rented from a commercial service, or the patient may be referred to a specialty practice offering this service.

Implantable Cardiac Event Recorders

- Implantable loop recorders (ILRs) are available for cases in which syncope is intermittent and difficult to capture with a 24-hour Holter recording or 7-day CER. These devices are small enough to implant subcutaneously in most dogs or cats. They are capable of monitoring the ECG for longer than 18 months and can be activated to store the ECG by a person observing an episode or can be programmed to store the ECG if the heart rate is slower or faster than the programmed limits (set at the time of implantation). The device is interrogated by a radiotelemetry device (similar to those used for pacemakers) to determine if unobserved episodes occurred. The devices require specialized programming and telemetry equipment, similar to that used in pacemaker implantation; they can be very helpful in cases with rare clinical signs (syncope).

Key Point

If syncope is infrequent, then a CER or ILR, rather than a 24-hour Holter recording, is much more likely to result in definitive diagnosis.

NONSELECTIVE ANGIOGRAPHY

INDICATIONS

- Nonselective angiography is occasionally helpful to identify congenital or acquired abnormalities of intracardiac or intravascular blood flow. Abnormalities of the right side of the heart (e.g., right atrium, right ventricle, pulmonary arteries) are most readily identified by this technique. Echocardiography and cardiac CT have superseded the need for this technique at most facilities.

TECHNIQUE

- Sedation or a light plane of anesthesia is usually necessary.
- A large-bore catheter (18 gauge or larger) is placed intravenously (preferably in the jugular vein).
- The animal is placed in the most appropriate position for opacification of the structures of interest (i.e., lateral recumbency for most cardiac defects, sternal recumbency to visualize pulmonary arteries).
- A large bolus of contrast is rapidly injected intravenously. Typically, 1 mL of radiopaque contrast per kilogram of body weight is used for the injection. Alternatively, the dose of iodine to be injected can be calculated, with 400 mg iodine per kilogram body weight as the desirable dose.
- A fluoroscopy unit allows continuous assessment. If fluoroscopy is not available, radiographic exposures are obtained 2 to 8 seconds after the injection is initiated (depending on the structures of interest and cardiovascular performance). Shorter times should be used for the evaluation of the right-side of the heart and pulmonary arteries and longer times for evaluation of structures on the left side of the heart or in animals with heart failure and slow circulation.

CLINICAL UTILITY

- Nonselective angiography is an alternative technique primarily used to evaluate lesions that involve in the right side of the heart, particularly when echocardiography, cardiac CT, or cardiac catheterization is either not an option or inconclusive.

LIMITATIONS, RISKS, AND COSTS

- Dilution of contrast material occurs as it moves through the circulation. Thus nonselective angiography results in poor opacification of structures that are very distal to the site of injection (e.g., left side of the heart and systemic arteries).
- Timing of radiographs is difficult to predict, and several attempts are often required unless fluoroscopy is available.
- Intravenous contrast agents may result in transient hypotension, cardiac arrhythmias, nephrotoxicity (especially in patients with preexisting renal dysfunction), and allergic reactions.

Key Point

Nonselective angiography is significantly limited, particularly in the assessment of left-sided heart structures. Echocardiography is preferred, and if the results remain inconclusive, cardiac CT or cardiac catheterization and angiography are more likely to be diagnostic than nonselective angiography.

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CARDIAC CATHETERIZATION

INDICATIONS

- In general, cardiac catheterization is defined as a combined angiographic and hemodynamic study undertaken for therapeutic or diagnostic purposes. The standard cardiac catheterization procedure for the evaluation of congenital or acquired cardiac diseases typically includes the measurement of intracardiac pressures, blood oximetry, and selective angiocardiography. The most common indication for cardiac catheterization in veterinary species is to ameliorate congenital heart disease (e.g., pulmonic stenosis or patent ductus arteriosus). For the purposes of diagnosis, advances in echocardiography have markedly reduced the need for routine cardiac catheterization.

TECHNIQUE

- Anesthesia is usually required.
- Surgical preparation of the neck (carotid artery or jugular vein) or inguinal area (femoral vein or artery) is required.
- Vascular access can be obtained by dissecting down to the vessel or with a percutaneous catheter introducer system with a modified Seldinger technique.
- Catheter advancement to the chamber of interest is performed under fluoroscopic or pressure waveform guidance (Fig. 5.3).

Oximetry

- Blood samples are obtained from various cardiac or great vessel locations to measure oxygen saturation and calculate shunt fraction in animals with congenital shunting defects.

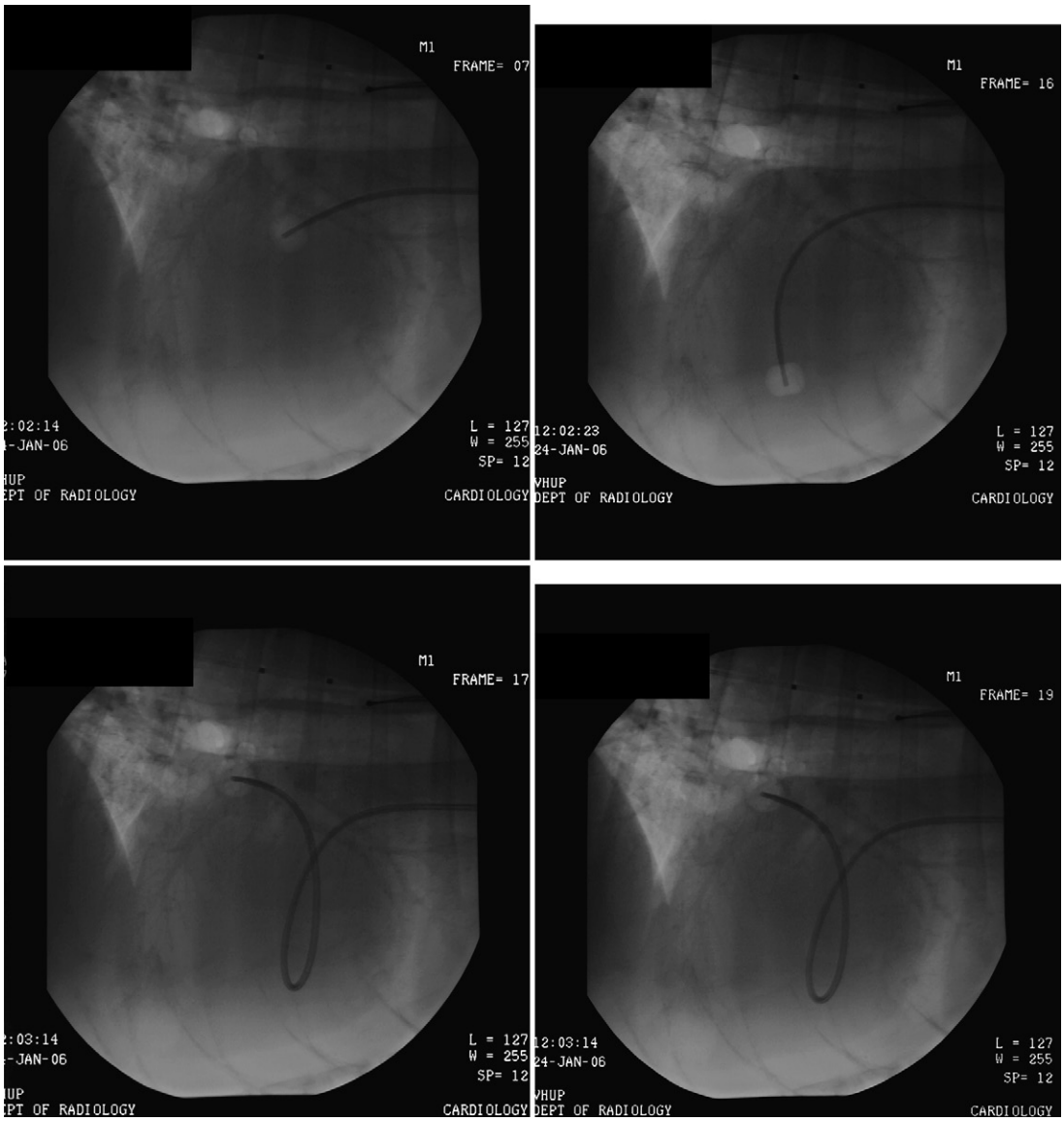


Fig. 5.3 Fluoroscopically obtained images of a balloon-tipped cardiac catheter being placed into the right side of the heart. The catheter is advanced to the heart via the jugular vein. The balloon on the tip of the catheter facilitates traversing the tricuspid valve because it will tend to follow blood flow.

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Angiocardiography

- A radiopaque contrast material is injected through the catheter(s) located at the appropriate areas of interest, and the image is recorded on videotape, radiographic film, or cineangiography; it can also be digitally stored (Figs. 5.4 and 5.5). Postprocessing can be performed on digital images with digital subtraction techniques. The primary advantage over nonselective angiography involves superior opacification of structures of interest.

Hemodynamics

- Cardiac output may be determined with thermodilution or indicator dye techniques.
- Intravascular or intracardiac pressures are recorded from chambers of interest. Pressures are typically recorded before and after therapeutic interventions (e.g., balloon valvuloplasty) to assess procedure efficacy.

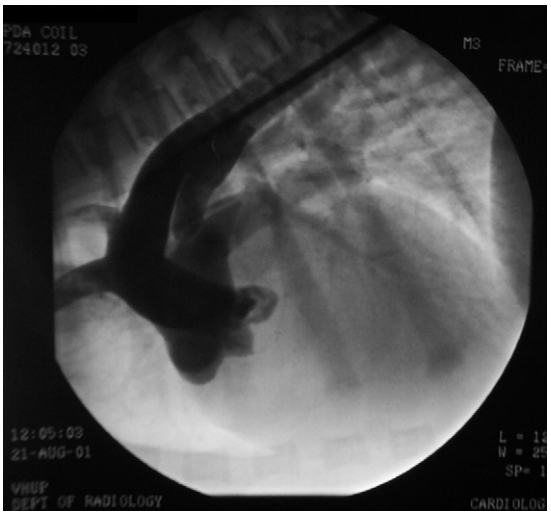


Fig. 5.4 Selective aortogram demonstrating a patent ductus arteriosus with radiographic contrast material crossing the ductus and entering the pulmonary artery (left to right flow).

- Additional procedures, such as balloon valvuloplasty, patent ductus arteriosus occlusion, endomyocardial biopsy, heartworm retrieval, stenting, and cardiac pacing, can be performed.

CLINICAL UTILITY

- Angiocardiography is valuable to diagnose cases of complex heart disease and to guide therapeutic interventions such as balloon valvuloplasty or patent ductus arteriosus occlusion. The approach provides useful morphologic and physiologic information regarding interventional responses to therapy.

LIMITATIONS, RISKS, AND COSTS

- Cardiac catheterization requires specialized equipment and training and is typically limited to tertiary care facilities.
- The technique can be time consuming.
- Contrast solutions can result in hemodynamic abnormalities. Patients with severe heart disease or renal disease are at a relatively higher risk.
- Infection, cardiac arrhythmias, air embolism, vascular thromboembolism, or perforation are possible complications. With appropriate experience and case selection, mortality rate is low.

SEROLOGIC TESTING

INDICATIONS

Animals with clinical signs suggestive of myocardial dysfunction resulting from infectious or immune-mediated causes and animals at risk of myocardial toxicity from chemotherapeutic agents are candidates.

- Trypanosoma titer. Animals with a travel history to Mexico, southern Texas, or other regions where Chagas disease is endemic. Chagas disease should

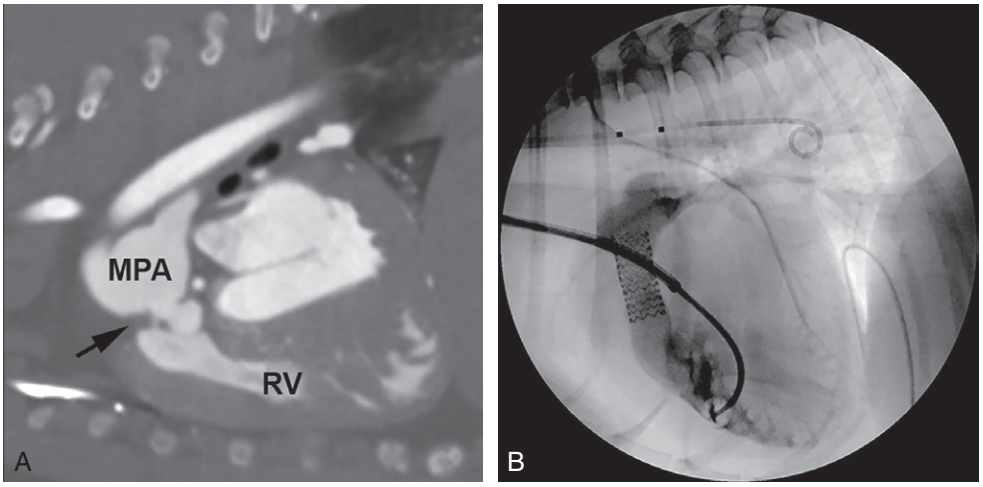


Fig. 5.5 (A) Sagittal cross-sectional view of the heart during a presurgical electrocardiography-gated cardiac computed tomography following the injection of iodinated contrast. There is a markedly thickened pulmonary valve that has resulted in stenosis at the level of the sinotubular junction (*arrow*), necessitating the placement of a transvalvular stent. (B) Selective angiocardiogram following transvalvular stent placement to treat severe valvular pulmonic stenosis. *MPA*, Main pulmonary artery; *RV*, right ventricle.

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be considered a differential diagnosis in cases that are diagnosed with a dilated cardiomyopathy phenotype, heart failure, ventricular tachyarrhythmias, and/or advanced second-degree or third-degree AV block.
- Antinuclear antibody titer. Animals with heart failure or arrhythmias, in addition to other clinical signs, are suggestive of immune-mediated diseases.
- Toxoplasmosis titer. Cats with myocardial dysfunction, fever, pneumonia, neurologic disease, chorioretinitis, or other signs compatible with toxoplasmosis.

TECHNIQUE

- Serum or plasma should be submitted to a laboratory that has the appropriate facilities to perform the indicated serologic testing.

CLINICAL UTILITY

- In selected cases, these tests can be useful in establishing an etiologic agent and in monitoring patients at risk for myocardial toxicity. Results must be interpreted in concert with the patient's clinical signs.

LIMITATIONS, RISKS, AND COSTS

- These tests are limited only by correct interpretation. Otherwise, there are no particular risks. Costs are dependent on the laboratory.

NEWER CARDIAC IMAGING TECHNIQUES

- These techniques are becoming more widely available, and familiarity with their potential applications is helpful to identify referral candidates. CT and magnetic resonance imaging (MRI) are useful adjunctive techniques that are less invasive than angiocardiography and may be advantageous over echocardiography for surgical planning and for the identification of certain diseases, such as cardiac neoplasia and pericardial disease. Nuclear scintigraphy is particularly useful for the quantitative assessment of intracardiac shunts.

COMPUTED TOMOGRAPHY

- CT is an x-ray technique that displays cross-sectional images of the body.
- A diagnostic cardiac CT evaluation requires hardware and software that optimize temporal resolution to overcome cardiac motion, ECG gating that allows for the reconstruction of different phases of the cardiac cycle, and spatial resolution to allow for a precise definition of cardiac anatomy (Fig. 5.5).
- An intravenous injection of nonionic iodinated contrast agent is used to define the blood pool.
- CT is becoming widely available for use in veterinary patients. Multiple veterinary case reports have demonstrated the utility of cardiac CT for identifying or confirming less common structural heart diseases such as aortic coarctation, intrathoracic vascular abnormalities, and vascular ring anomalies.
- Acquisition of the images is generally very quick but does require general anesthesia or heavy sedation in veterinary patients.

MAGNETIC RESONANCE IMAGING

- With MRI, a high natural contrast exists between blood and cardiovascular structures; therefore contrast medium is not required to discriminate the blood pool.
- Physiologic gating of the imaging sequence is necessary for cardiac imaging, and this capability may not be available at all facilities that offer MRI.
- Despite this limitation, as MRI has become more available, reports have demonstrated that it can add useful information regarding complex congenital heart disease and acquired diseases such as cardiac neoplasia.
- MRI gives useful information on cardiovascular morphology, function, and tissue character without ionizing radiation.
- General anesthesia is required to perform the imaging study. Acquisition of the images takes much longer than cardiac CT, and some patients with complex congenital heart disease may not tolerate the anesthesia.

NUCLEAR CARDIOLOGY

- Gamma ray-emitting radiopharmaceuticals (radionuclides) are injected intravenously and either are extracted by the myocardium or remain in the blood pool. A scintillation (gamma) camera interfaced with a computer analyzes and stores the data.
- The direction and severity of congenital intracardiac shunts can be determined. Regional distribution of myocardial perfusion can also be visualized.

LIMITATIONS

The required technical expertise and expense of equipment limit the use in general veterinary cardiology.

? Frequently Asked Questions

HOW IS A VAGAL MANEUVER HELPFUL WHEN A DOG WITH A SUSPECTED SUPRAVENTRICULAR TACHYCARDIA IS ASSESSED?

An effective vagal maneuver will transiently increase vagal tone. The technique can be helpful to differentiate between sinus tachycardia, which occurs during a normal physiologic response to pain, fever, and so on, and a pathologic ectopic SVT, which occurs with paroxysmal atrial tachycardia. A gradual slowing of the heart rate (over several seconds) suggests the focus is sinus because the sinus node accelerates and decelerates gradually. An abrupt cessation of the tachycardia is suggestive of a pathologic focus (i.e., SVT). Lack of response to a vagal maneuver can occur with either condition and is nondiagnostic. Occasionally, an SVT will respond to a vagal maneuver with slowed AV nodal conduction, resulting in second-degree AV block with an underlying rapid P-wave rate (Fig. 5.6).

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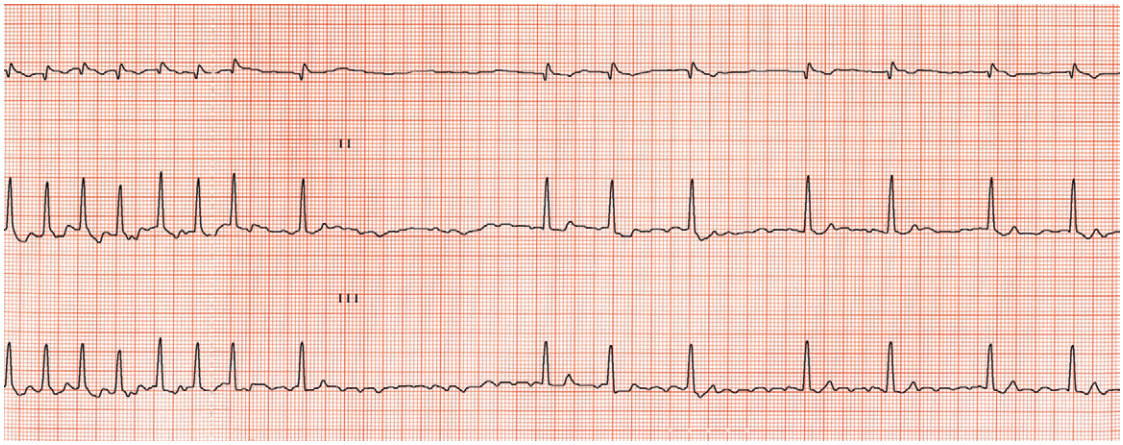


Fig. 5.6 Simultaneous leads I, II, and III ECG showing the effect of a vagal maneuver on a supraventricular tachycardia (atrial fibrillation in this example). Note the dramatic slowing of the heart rate, which results in clearer demonstration of the hallmarks of atrial fibrillation (irregularly irregular rhythm, lack of P waves and fibrillation waves). 50 mm/s; 10 mm/mV.

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Chapter 6

Acquired Valvular Disease

Jonathan A. Abbott

Acquired primary valvular disease in dogs and cats generally is degenerative or, much less commonly, infective. Other pathologic processes, such as neoplasia, rarely affect the cardiac valves. Myxomatous degeneration of the mitral valve is the most common cardiac disease in the dog. Mitral valve incompetence caused by valvular degeneration can result in progressive cardiac enlargement and, in some cases, heart failure (HF). Clinical signs, including cough that might be caused by compression of the mainstem bronchi by an enlarged left atrium, may precede the development of cardiogenic pulmonary edema. The clinical consequences of degenerative valvular disease are observed primarily in elderly, small-breed dogs.

Infective endocarditis (IE) is an uncommon form of acquired valvular disease that is observed occasionally in dogs and rarely in cats. Middle-aged medium- and large-breed dogs are affected most often. The clinical signs of IE relate to sepsis, thromboembolism, and HF.

DEGENERATIVE MITRAL VALVE DISEASE

Numerous designations for degenerative mitral valve disease (MVD) have been proposed on the basis of its clinical and pathologic features. The terms *myxomatous valvular degeneration, myxomatous transformation, mucoid degeneration, endocardiosis, chronic valvular disease*, and *degenerative valvular disease* all refer to the same disorder.

Key Point

Degenerative MVD is the most common cardiac disease in the dog; it is an acquired disease, and the prevalence is greatest in the geriatric population.

PREVALENCE AND INCIDENCE

- Degenerative MVD is the most common cardiac disease in the dog; it is an acquired disease, and the prevalence is greatest in the geriatric population.
- Clinical evidence of degenerative valvular disease is detected in approximately 30% of dogs aged 13 years and older.
- MVD is a progressive disease, and subtle changes in valve structure precede the development of clinically evident valvular dysfunction. Consequently, the prevalence of MVD detected by postmortem examination is higher than that reported in clinical studies.
- Postmortem evidence of advanced degenerative valvular disease was found in 58% of dogs older than 9 years; when mild degenerative changes are included, the postmortem prevalence exceeds 90% in dogs older than 13 years.
- MVD may affect any breed of dog, but clinical consequences of MVD are observed most often in small-breed dogs. Miniature Poodles, Pomeranians, Yorkshire Terriers, Chihuahuas, and other small dogs are commonly affected. The prevalence of MVD in Cavalier King Charles Spaniels is particularly high, and in dogs of this breed, the disease is sometimes clinically evident at a young age.
- Male dogs are affected somewhat more often than female dogs.
- Degenerative valvular disease is uncommon in cats, and when it occurs, it seldom results in clinical consequences.

PATHOLOGY

- MVD is grossly characterized by nodular distortion of the valve leaflets, as well as by thickening and, sometimes, lengthening of the chordae tendineae. The appearance of a small number of nodules at the free edge of the valve leaflet is the initial manifestation of disease. As the disease progresses, these nodules increase in number and size and coalesce. In severe cases, the leaflets are contracted, and the free edge of the leaflet rolls inward toward the ventricular endocardium (Fig. 6.1). When severe, these abnormalities prevent coaptation of the valve leaflets, resulting in mitral valve incompetence.
- MVD is histologically characterized by the deposition of mucopolysaccharides primarily within the spongiosa layer of the valve leaflet. Fibrosis of the valve is also present but is not the dominant histologic feature. Inflammatory infiltrates are absent; MVD is a sterile, degenerative disease that bears no known relationship to endocarditis.

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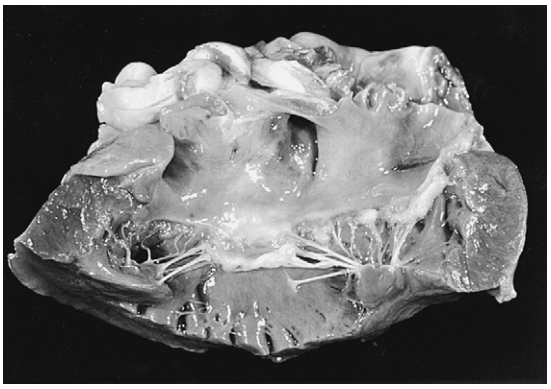


Fig. 6.1 A specimen that demonstrates the gross features of severe mitral valve degeneration. The mitral valve leaflets are abnormally thick and nodular. (Courtesy Department of Veterinary Pathology, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Canada.)

ETIOPATHOGENESIS

Key Point

The prevalence of endocarditis is no greater in dogs affected by MVD than in other, nonaffected dogs.

- The cause of MVD is unknown.
- Investigations of the molecular biology and biomechanics of MVD suggest that a complex interaction of mechanical stresses and signaling pathways leads to altered gene expression and induction of growth factors. A unified explanation for myxomatous transformation has not been established. However, an inductive effect of tensile and shear strains, conversion of valvular interstitial cells to a myofibroblastic phenotype, enzymes, and signaling molecules, including metalloproteinases, serotonin, and transforming growth factor, may all play a role.
- Because distinct breed predispositions are recognized, it is likely that there is a genetic predisposition for the development of MVD. Available evidence suggests that the tendency to develop MVD is not subject to simple Mendelian inheritance but is a polygenic trait or, if monogenic, subject to genetic or environmental comodification.
- In Cavalier King Charles Spaniels, parental status with respect to age and murmur intensity is an important determinant of the prevalence of murmurs in 5-year-old offspring.
- Genome-wide analyses of Cavalier King Charles Spaniels disclosed two chromosomal regions that are associated with MVD, but specific causative genes have not been identified. However, recently published data suggest that variants at the nebulette locus might be associated with severity of disease in patients of this breed.

PATHOPHYSIOLOGY

Key Point

The cause of MVD is unknown, but genetic factors are likely important.

- The mitral valve apparatus consists of the mitral valve leaflets, the fibrous valve annulus, the chordae tendineae, and the left ventricular papillary muscles.
- The two mitral leaflets are known as the aortic (anterior) and the mural (posterior) leaflets. In health, the mitral leaflets are thin, translucent structures that are tethered to the left ventricular papillary muscles by the chordae tendineae. The two left ventricular papillary muscles arise from the caudal (free) wall of the left ventricle. The basilar attachment of the mitral leaflets is to the fibrous left atrioventricular valve ring, known as the *mitral annulus*.
- The initiation of valve closure is a passive process; in early systole, when left ventricular pressure exceeds left atrial pressure, the mitral valve leaflets are forced into apposition. In normal individuals, the tethering effect of the chordae tendineae prevents prolapse, or bowing, of the leaflets into the left atrium.
- Coaptation of the normal mitral leaflets is complete, and there is little or no regurgitation through the valve orifice. Normal mitral valve function ensures that the entirety of the left ventricular stroke volume is ejected through the aorta. When the mitral valve is incompetent, a fraction of the left ventricular stroke volume (regurgitant fraction) is ejected through the valvular regurgitant orifice into the left atrium.
- Mitral regurgitation (MR) may be mild and have minimal consequences, or it can be severe. The severity of MR is determined principally by the size of the regurgitant orifice and the relationship between left atrial and left ventricular systolic pressure. Potentially, both of these determinants can be pharmacologically manipulated by administration of vasodilators.
- MR increases left atrial pressure, which potentially results in left atrial dilation. When the mitral valve leaks, the pulmonary venous return is augmented by the regurgitant volume; in consequence, the ventricle is filled in diastole not only by blood that has returned from the lungs but also by blood that has been regurgitated into the atrium. Therefore MR imposes a volume load on the left ventricle and the left atrium.
- High end-diastolic pressures and volumes result in ventricular dilation and hypertrophy. Hypertrophy of this type, in which the ratio of wall thickness and chamber size remains roughly unchanged, is known as eccentric hypertrophy.
- Severe MR may increase left ventricular filling pressures. High filling pressures are reflected backward,

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raising pulmonary vein pressure and potentially initiating the development of pulmonary edema.

- The syndrome of clinical signs and neuroendocrine activation that results from cardiac dysfunction is known as HF. Because veterinary patients cannot offer subjective observations—for example, the perception of breathlessness during exertion—the presence of congestive signs is generally used as an objective criterion for the diagnosis.
- Congestive heart failure (CHF) is the syndrome of clinical signs caused by venous pressure elevations that result from cardiac dysfunction. Left-sided CHF is defined by the presence of cardiogenic pulmonary edema. Right-sided CHF refers to clinical signs that result from systemic congestion; in dogs, ascites is the most common manifestation of right-sided CHF.
- Because of maladaptive neuroendocrine responses associated with cardiac dysfunction, and in the case of MVD, ongoing valvular valvular degeneration, the syndrome of HF tends to be progressive. The elimination of congestive signs does not signify resolution of the HF state, and when the responsible disorder cannot be definitively corrected, HF is a terminal syndrome.
- The imposition of a chronic volume load on the heart can result in deterioration of systolic myocardial function, a state sometimes known as cardiomyopathy of overload. In general, MR is relatively well tolerated by the myocardium possibly because the left atrium represents a low-pressure reservoir into which the ventricle can eject blood. In fact, dogs that have severe MR often develop CHF at a time when systolic myocardial function (contractility) is, according to echocardiographic indices, normal or only mildly diminished.
- MR may remain clinically silent until it is advanced. When HF results from MR, clinical signs may include weakness, syncope, cough, and dyspnea.
- Cough is a centrally mediated reflex, and most cough receptors are located in the large airways. Cough associated with MR in small-breed dogs may result from the following:
  - Pulmonary edema, when fluid floods the alveoli.
  - Reflexes mediated through stimulation of the juxtapulmonary (J) receptors; these receptors are associated with the pulmonary capillaries and are sensitive to increases in pulmonary venous pressure.
  - Compression of the mainstem bronchi by an enlarged left atrium.
  - Recent findings suggest that the pathogenesis of cough in patients with MVD is complex. For example, bronchoscopic evaluation of dogs with MVD that have a history of cough often reveals airway collapse, but this finding is not statistically associated with the presence of an enlarged left atrium. In spite of that, a recent retrospective investigation identified left atrial enlargement, but not radiographic pulmonary edema, as a risk factor for cough in patients with MVD.
- It is likely that the cause of cough observed in patients with MVD is multifactorial. Pulmonary congestion or edema, cardiac enlargement, and the presence of concurrent airway disease may all contribute.

CLINICAL PRESENTATION

Heart Failure

The number of distinct, extant definitions of "heart failure" is evidence of the semantic difficulty the term presents. It is now generally accepted that HF is a clinical syndrome characterized by congestive signs, exercise intolerance, or both that result from diseases that impair filling or emptying of the heart. In 2009 a working group of the American College of Veterinary Internal Medicine (ACVIM), Specialty of Cardiology, developed "Guidelines for the Diagnosis and Treatment of Canine Chronic Valvular Heart Disease." These guidelines were revised and updated in 2019 and published as an open-access document. A schema for "staging" of patients, modified from one proposed for the description of humans with HF, was presented.

According to the current ACVIM Guidelines, patients with MVD are classified as follows:

Stage A: patients predisposed to the development of MVD/HF

Stage B: patients with MVD that never have had clinical signs

- B1—without cardiac enlargement that defines B2
- B2—with radiographic and echocardiographic evidence of chamber enlargement meeting criteria that identify patients for which there is evidence that medical treatment delays the onset of clinical signs

Stage C: patients with MVD that have current *or prior* clinical signs

Stage D: patients with medically refractory HF

Traditionally, patients with HF had been categorized according to the degree of functional limitation that results from cardiac disease. Examples of this approach include the New York Heart Association Functional Classification and a similar system proposed by the International Small Animal Cardiac Health Council (ISACHC). However, classifications based on functional limitation are imperfect; such systems are poorly suited to the development of therapeutic guidelines partly because they do not reflect the progressive nature of the HF state. Furthermore, they do not take into account the fact that the outcome of patients that have *never* have had pulmonary edema generally is superior to those that have a *history* of edema that has resolved because of treatment. However, systems such as ISAACHC are not necessarily obsolete. Patients with fulminant edema caused by MVD and patients that are asymptomatic by virtue of therapy can both be

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described has having stage C MVD. It would therefore be reasonable to use both Staging *and* the ISACCH to resolve this dilemma; for example, the patient with fulminant edema might be described as stage C/class 3.

History

Key Point

It is important to recognize that cough can be associated with MVD in the absence of pulmonary edema. When this is the case, the cough might be a sign of heart disease or reflect primary respiratory tract disease but is not the result of pulmonary edema. This distinction is important because a diagnosis of pulmonary edema carries important prognostic and therapeutic implications.

- MVD exhibits a broad spectrum of severity. In most affected dogs, MVD does not cause clinical signs, and the disease is detected when a cardiac murmur is incidentally identified in patients presented for routine health care or for management of noncardiac disease.
- In cases in which MVD does become clinically apparent, cough is often the clinical sign that is first observed by the dog owner. When coughing is caused by pulmonary edema or congestion, other signs, such as exercise intolerance and tachypnea, are usually present. The expectoration of pink froth is sometimes observed in patients with fulminant pulmonary edema.
- Occasionally, syncope is the clinical sign that is seen first in dogs with MVD. Syncope is a transient loss of consciousness caused by a sudden and precipitous decline in cerebral perfusion. MVD can be responsible for syncope when cardiac enlargement predisposes to arrhythmias. In addition, exertional syncope might result when MR limits stroke volume so that cardiac output does not adequately increase to meet the physiologic demands of exercise. Alternatively, syncope on exercise or excitement or associated with paroxysmal cough can result from sudden onset of reflex-mediated bradycardia.
- Other clinical signs related to reduced cardiac performance, including tachypnea, exercise intolerance, and abdominal distention caused by ascites, occasionally prompt owners of affected dogs to seek veterinary attention.

Physical Findings

- The most notable feature of the physical examination is a systolic murmur that is usually heard best over the left cardiac apex. The murmur of MVD is indistinguishable from the murmur caused by other disorders, such as IE or dilated cardiomyopathy (DCM), which also can result in MR. Importantly, however, an acquired, left apical, systolic murmur in an older, small-breed dog is almost always caused by MVD. The intensity of the murmur depends on a number of factors, but severe MR usually causes a loud murmur. Severe MR associated with a nonrestrictive regurgitant orifice could hypothetically result in a soft murmur, but this is extremely uncommon in MVD. The murmur of MR typically has a plateau-shaped configuration, meaning that the murmur has a similar intensity throughout systole; when the murmur is loud, the second heart sound (S_2) may be obscured.
- An exaggerated apical impulse is often evident on precordial palpation of patients with moderate or severe MR. By definition, a precordial thrill—a palpable vibration of the chest wall—is present when the murmur intensity is grade 5/6 or greater.
- A high-frequency, midsystolic click is sometimes heard in older, small-breed dogs. These clicks may be associated with mitral valve prolapse. In many dogs, clicks are a precursor of MR. Often a soft systolic murmur of MR can also be heard in patients that have systolic clicks.
- When MR is severe, the third heart sound (S_3) is sometimes audible and results in an S_3 gallop. Care must be taken to distinguish a midsystolic click from a gallop. In general, a systolic click is louder than the S_3, and in patients with MVD, a click typically is heard in association with findings that suggest mild MR. In contrast, an S_3 gallop usually reflects severe MR and generally is heard in patients with loud murmurs. Note that S_3 is sometimes audible in patients with DCM. However, DCM is typically a disorder of large and giant dog breeds.
- The femoral arterial pulse is usually of normal strength when MR is present, but the pulse may have a rapid rise. Very severe MR can be associated with diminished pulse strength.
- Crackles may be heard in patients with pulmonary edema. It should be recognized that pulmonary crackles have low diagnostic specificity for identification of pulmonary edema, and the prevalence of primary respiratory diseases such as chronic bronchitis in the patients most often affected by MVD is high. Primary respiratory tract diseases can explain adventitious pulmonary sounds, such as crackles, in the absence of pulmonary edema.
- Abdominal palpation is usually normal in patients with MVD, but hepatic enlargement or even ascites may be present when there is severe tricuspid valve disease or when pulmonary hypertension complicates the presentation of MVD.

Patients With Mitral Regurgitation and Concurrent Respiratory Tract Disease

- Primary diseases of the respiratory tract, such as collapsing trachea and chronic bronchitis, are common in the patient group that is affected by MVD. In an individual patient, it can be difficult to determine whether cardiac disease or respiratory disease bears the greatest responsibility for the development of clinical signs.

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- In general, patients with severe MR are more likely than patients with respiratory disease to have poor body condition.
- Although loud murmurs of MR are sometimes clinically inconsequential, it is extremely uncommon for soft murmurs to indicate severe MR with cardiac enlargement.
- The presence of respiratory sinus arrhythmia (RSA) can also be of diagnostic value. Much of the moment-to-moment heart rate variability observed in healthy dogs is vagally mediated. In patients with severe cardiac disease, there is little vagal influence on heart rate and rhythm, and as a result, RSA is not prominent. In contrast, sinus arrhythmia is often preserved or even accentuated when primary respiratory tract disease is responsible for clinical signs. The physical finding of RSA is virtually incompatible with the presence of cardiogenic pulmonary edema.
- Although exceptions occur, clinical signs are usually related to respiratory tract disease in patients that are overweight and have sinus arrhythmia and a soft cardiac murmur.
- In contrast, the clinical signs of thin patients with loud murmurs and tachycardia are more likely to result from cardiac disease or CHF.
- Coughing in elderly, small-breed dogs that do not have cardiac murmurs is almost always due to primary respiratory tract disease (Table 6.1).

Large-Breed Dogs With Mitral Valve Disease

- A syndrome of severe MR and concurrent myocardial dysfunction is recognized in medium- and large-breed dogs.
- For reasons that are unknown but may relate to the geometry or pattern of contraction of inherently larger ventricles, large-breed dogs with MR are more apt to have echocardiographically evident myocardial dysfunction than are small-breed dogs.
- The echocardiographic appearance of valvular lesions in large dogs tends to be less impressive than it is in small dogs.
- Perhaps because myocardial dysfunction complicates MVD in large dogs more often than it does in small dogs, the prognosis may be worse than in smaller dogs.

Table 6.1 Guidelines for Clinical Assessment of Elderly Small-Breed Dogs With Cough and Cardiac Murmur[a]

	CARDIAC DISEASE	RESPIRATORY DISEASE
Body condition	Thin	Obese
Cardiac murmur	Loud	Often soft, occasionally loud
Heart rate	Rapid	Normal or slow
Rhythm	Regular, unless pathologic arrhythmias are present	Exaggerated respiratory arrhythmia may be present

[a]It is important to recognize that exceptions to these generalities occur. However, when a patient exhibits all of the findings in the left-side column, it is likely that cardiac disease or perhaps congestive heart failure is responsible for the cough. It must be recognized that some patients have both respiratory tract and cardiac disease. Ultimately the distinction between respiratory tract and cardiac disease is made through diagnostic imaging; thoracic radiographs are indispensable, and echocardiography often provides useful complementary data.

DIAGNOSTIC FINDINGS

Thoracic Radiography

In most cases, thoracic radiography is the most important element of the diagnostic approach to MVD. MVD is extremely common but progresses at a rate that varies greatly among individuals. Many patients with MVD are subclinical ("asymptomatic") and never develop clinical signs related to MR. Early in the course of MVD, the cardiac silhouette is normal. If clinically consequential MR develops, then there is enlargement of the cardiac silhouette. It should be recognized that the ability of thoracic radiographs to delineate specific cardiac chambers is limited. In general, the left atrium can be assessed with the greatest certainty. This is fortunate because, in the overwhelming majority of cases, left atrial enlargement precedes the development of clinical signs. A diagnosis of left-sided cardiogenic pulmonary edema secondary to MVD can rarely be supported in the absence of radiographic left atrial enlargement.

Radiographic Appearance of Left Atrial Enlargement

- The left atrium is left of, and caudal to, the right atrium. Radiographically, it occupies the caudodorsal area of the cardiac silhouette in the lateral projection.
- In the absence of left atrial enlargement, the caudal portion of the trachea curves ventrally over the caudal aspect of the cardiac silhouette.
- When the left atrium is enlarged, the caudal border of the cardiac silhouette straightens, and the trachea is forced dorsally to varying degrees. With marked left atrial enlargement, the left mainstem bronchus is narrowed, and the trachea adopts a path that is parallel to the thoracic vertebrae. Occasionally, severe left atrial enlargement has the appearance of a mass that splits the mainstem bronchi.
- In the ventrodorsal or dorsoventral projection, the left atrium is located near the center of the cardiac silhouette. When enlarged, the left atrium splits the mainstem bronchi to varying degrees. This is apparent in well-penetrated radiographs and results in an appearance that is sometimes known as the "crab sign" or the "bowlegged cowboy" (Figs. 6.2 and 6.3).

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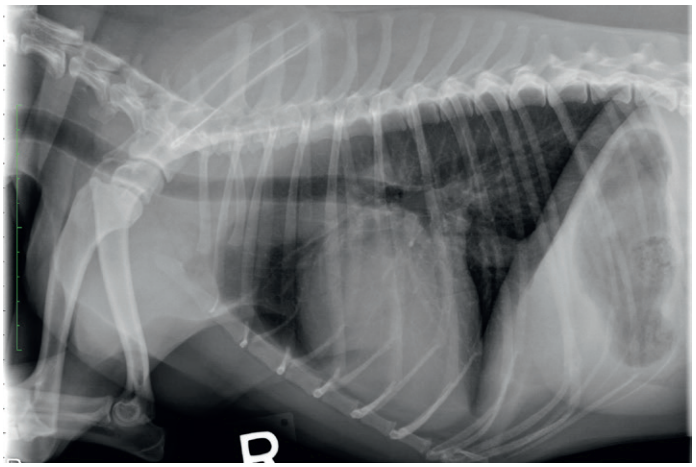


Fig. 6.2 Lateral thoracic radiograph obtained from an 11-year-old mixed-breed dog with degenerative mitral valve disease. There is relatively mild but distinct left atrial enlargement, as evidenced by elevation of the trachea and straightening of the caudal border of the cardiac silhouette.

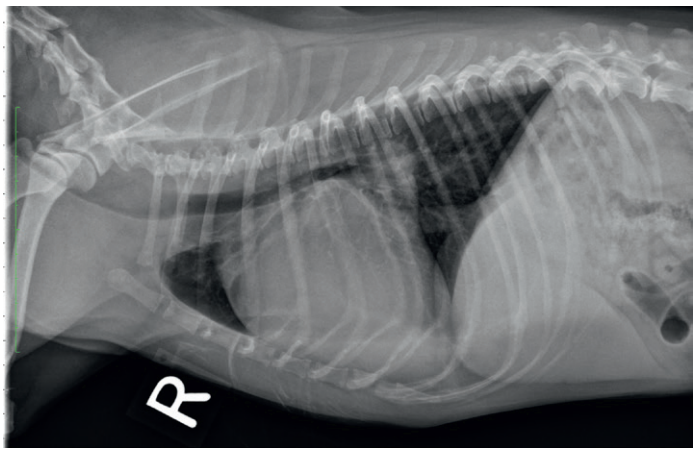


Fig. 6.3 A lateral thoracic radiograph obtained from an 8-year-old male castrated Pomeranian dog with severe mitral valve incompetence caused by degenerative disease. The left atrium is markedly enlarged, and the mainstem bronchi are compressed. The appearance of the trachea suggests concurrent tracheal collapse.

- In addition, in the ventrodorsal or dorsoventral view, enlargement of the left atrium may cause a bulge that represents the atrial appendage at the 3 o'clock position.

Radiographic Findings of Pulmonary Congestion and Edema

- The radiographic finding of pulmonary venous distention reflects increases in pulmonary venous pressure. Pulmonary venous distention suggests pulmonary congestion and may precede the development of pulmonary edema.
- A central, or perihilar, distribution can characterize cardiogenic pulmonary edema in dogs, but cardiogenic edema is commonly asymmetric and focal; there is a predilection for the right caudal lung lobe.
- The development of interstitial pulmonary edema precedes the appearance of alveolar edema.
- Blurring of vascular detail in the presence of left atrial enlargement and, sometimes, concurrent pulmonary venous distention characterizes the radiographic appearance of interstitial pulmonary edema.
- When tissue fluid weeps into the pulmonary alveoli, it provides contrast with air-filled structures such as the bronchi, resulting in air bronchograms. Alveolar pulmonary opacities, together with radiographic evidence of left atrial enlargement, are diagnostic of left-sided CHF (Fig. 6.4). The presence of alveolar pulmonary edema indicates severe HF that is almost invariably associated with noticeable respiratory distress.

Radiographic Cardiac Mensuration

Global cardiac size can be evaluated subjectively, but accurate subjective assessment requires expertise; as

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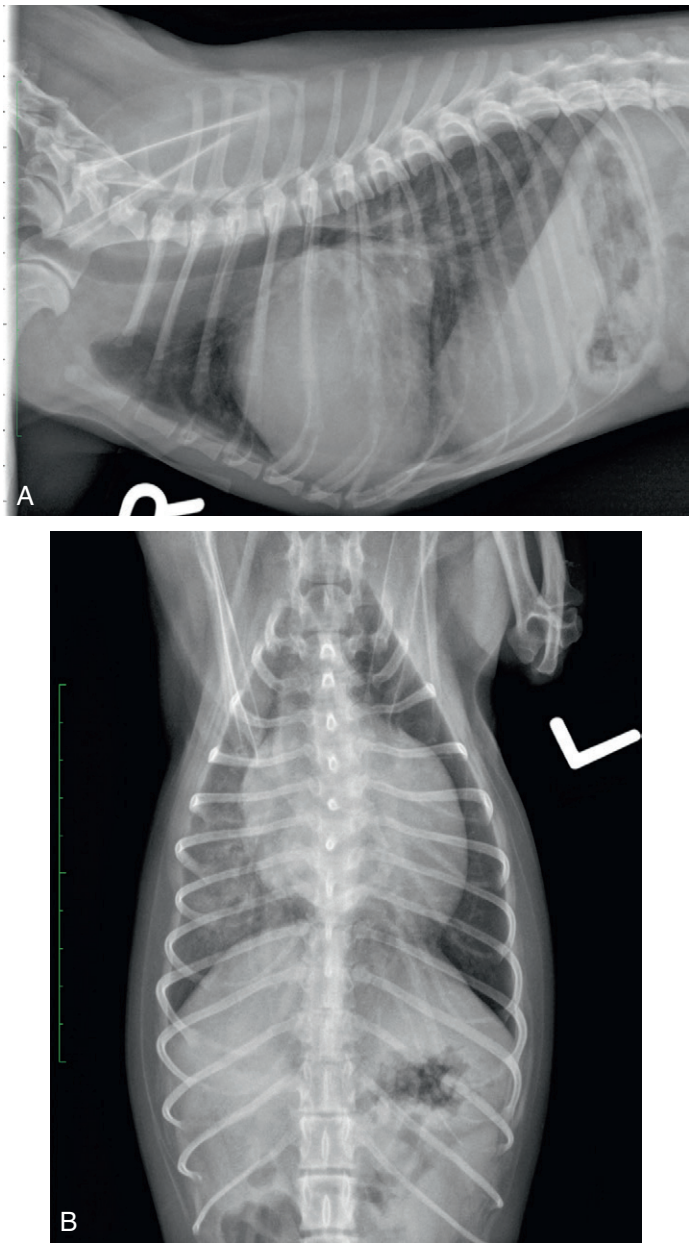


Fig. 6.4 Lateral **(A)** and ventrodorsal **(B)** thoracic radiographs obtained from a 10-year-old male castrated Chihuahua with degenerative mitral valve disease. The cardiac silhouette is markedly enlarged, and there is evidence of left atrial enlargement. Pulmonary opacities compatible with edema are distributed throughout the lung; the edema most noticeably affects the right caudal lung lobe.

a result, methods of radiographic cardiac mensuration might have a particular utility for generalists. The vertebral heart score (VHS) was first described by Buchanan. Although there are limitations to this technique—not least of these is a breed effect—it is a useful, objective means of evaluating the size of the cardiac silhouette. It is also relevant that a VHS in excess of 10.5 was an inclusion criterion for the clinical trial that determined the effect of pimobendan on canine patients with asymptomatic MVD, and therefore the VHS has therapeutic implications. There are minor variations in method, but the VHS is calculated from the two perpendicular dimensions of the cardiac silhouette viewed in the lateral projection. These two dimensions are defined in terms of the lengths of vertebral bodies, beginning at the fourth thoracic vertebra. In general, a VHS that exceeds 10.5 is evidence of cardiomegaly but there is an important breed effect. For example, Cavalier King Charles spaniels commonly have a VHS that exceeds this figure in the absence of echocardiographically evident cardiac disease.

Left atrial size is a surrogate marker of hemodynamic burden and reliable methods of identification of atrial enlargement are clinically useful. The canine vertebral left atrial size (VLAS)—measured from the ventral border of the bifurcation of the trachea to the union

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of the dorsal border of the caudal caval vein and the cardiac silhouette—can be measured in terms of vertebral bodies analogous to the VHS method (Fig. 6.5). VLAS that exceeds 2.5 generally reflects echocardiographic left atrial enlargement.

RADIOGRAPHIC IDENTIFICATION OF STAGE B2

- Optimally, contemporaneously acquired radiographic and echocardiographic data are used to stage canine patients with mitral valve disease. However, financial limitations on the part of pet owners often constrains the diagnostic approach. Therefore quantitative radiographic variables that predict that echocardiographic criteria for stage B2 have been met are of interest.
- Several investigations have addressed this clinical problem using different methods and patient populations and as a result, a number of different cut-points have been proposed. In general, a VHS in excess of 11.1 or a VLAS greater than 2.5 imperfectly, but with reasonable accuracy, predict that the echocardiographic criteria which define stage B2 mitral valve disease are met. These values optimize accuracy but, in an attempt to avoid unnecessary or at least potentially unhelpful treatment, it might be appropriate to use higher values that result in high positive predictive values, perhaps 11.4 or 2.8 respectively for VHS and VLAS. As a single measure, the VLAS might be superior to the VHS for identification echocardiographic stage B2 mitral valve disease and values greater than 2.8 are likely to be predictive while resulting in few "false positives."

Echocardiography

- Echocardiographic examination of patients with MVD demonstrates variable degrees of left atrial (Fig. 6.6) and left ventricular dilation. Hypertrophy is usually adequate to preserve a near-normal relationship between the diastolic lumenal dimension and wall thickness.
- The mitral leaflets may be noticeably thicker than normal, and prolapse of the leaflets into the left

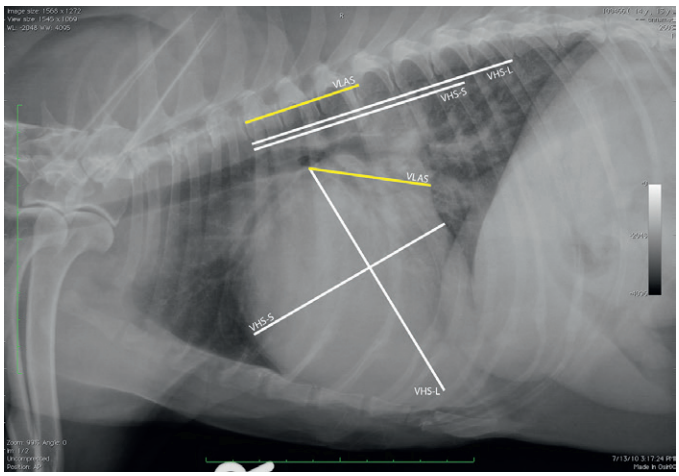


Fig. 6.5 A right lateral thoracic radiograph obtained from a canine patient with enlargement of the cardiac silhouette resulting from mitral valve regurgitation. Two perpendicular lines represent the dimensions that are summed to provide the vertebral heart score (*VHS*). The dimension that defines vertebral left atrial size (*VLAS*) extends from the tracheal bifurcation to the intersection of the dorsal border of the caudal caval vein and the caudal aspect of the cardiac silhouette. In this case, the VLAS is 3, and the VHS, 12.5 vertebral bodies.

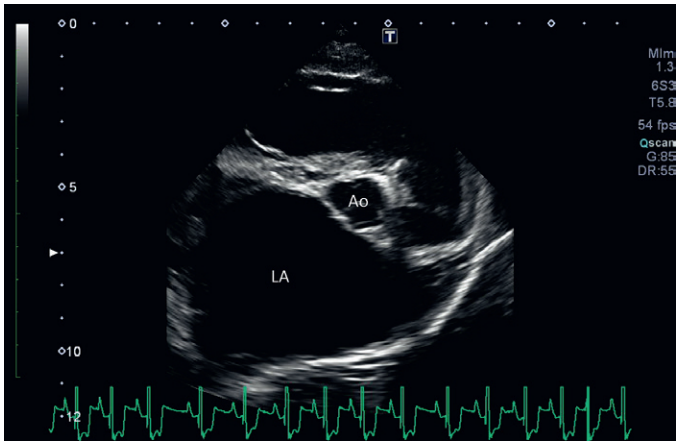


Figure 6.6 A right parasternal short-axis echocardiogram obtained from a 13-year-old female-spayed mixed-breed dog with severe mitral valve regurgitation. The left atrium (*LA*) is markedly enlarged; the dimension of the body of the atrium is more than twice the diameter of the aorta (*Ao*).

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atrium in systole is commonly observed (Fig. 6.7). Three-dimensional echocardiographic data provide evidence that the canine mitral valve annulus has a saddle shape. As a result, it is likely that the echocardiographic finding of leaflet protrusion in image planes in which the outflow tract visible has a greater diagnostic specificity for identification of prolapse than the same finding in "four-chamber" views. Veterinary patients are not often subject to surgical valve repair, so in most cases the presence of absence of prolapse has little clinical relevance.

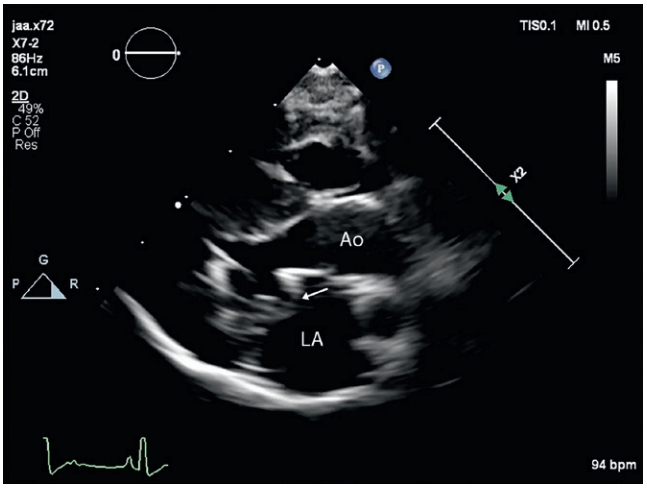


Fig. 6.7 A right parasternal long-axis echocardiogram obtained from a 11-year-old female-spayed mixed-breed dog with mitral regurgitation caused by degenerative disease. The image was obtained during systole. The left atrium (*LA*) is enlarged, and there is distinct prolapse (*arrow*) of the mitral valve leaflets. Because of the "saddle shape" of the mitral valve annulus, an image plane that includes the left ventricular outflow—as opposed to a four-chamber view—probably identifies prolapse with greater specificity. *Ao*, Aorta; *LV*, left ventricle.

- The echogenicity of affected leaflets is generally uniform, and nodular thickening is diffuse. In contrast, infective vegetations typically are localized, may exhibit oscillations that are independent of the valve leaflet, and are more, or less, echogenic than the valve leaflet.
- Often, the tricuspid leaflets are affected, although seldom as markedly as the mitral valve.
- Evaluation of myocardial function in patients with MR is difficult. When MR is moderate or severe, loading conditions imposed on the left ventricle are altered, and left ventricular emptying is hyperdynamic (Fig. 6.8), provided systolic myocardial function (contractility) is preserved.
- Ejection phase indices of systolic function such as fractional shortening are elevated because these variables are highly load dependent. When MR is present, impedance to ventricular emptying might be reduced because the ventricle is able to eject blood into the low-pressure reservoir of the left atrium. More importantly, end-diastolic ventricular stretch associated with MR increases the force of contraction and contributes to the finding of hyperdynamic ventricular performance. A normal or subnormal fractional shortening in the setting of MR that has

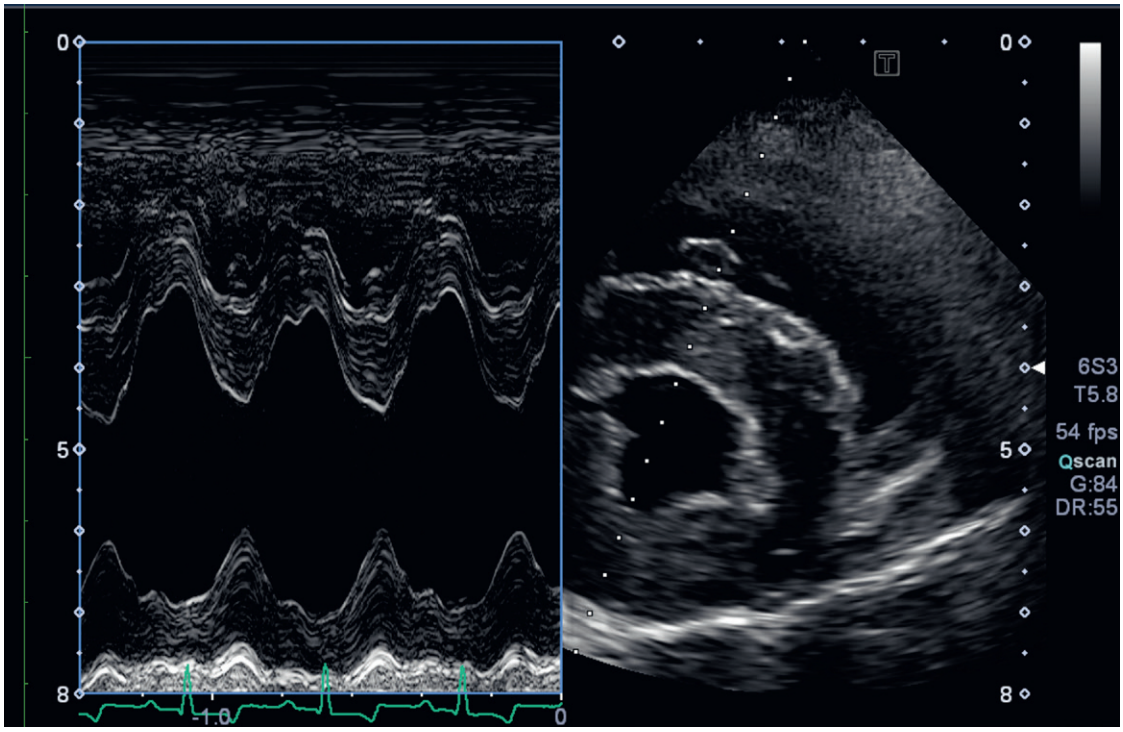


Fig. 6.8 M-mode echocardiogram obtained at the level of the left ventricular papillary muscles from a 9-year-old male castrated Cavalier King Charles Spaniel. Left ventricular dilation and hypertrophy are evident. Left ventricular systolic performance is hyperdynamic; the fractional shortening exceeds 50%.

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resulted in left ventricular dilation suggests systolic myocardial dysfunction (Fig. 6.9).

- Because the end-systolic left ventricular dimension is determined by relatively few factors, it is a better index of myocardial function; however, end-systolic left ventricular dimension must be interpreted in the context of body size. A method of echocardiographic mensuration in which cardiac dimensions are indexed to aortic diameter or the aortic diameter predicted based on body weight has been proposed. Both allometric scaling, and the use of aorta-based ratio indices, overcome some of the theoretic and practical limitations of comparing cardiac dimensions to body weight.
- The end-systolic volume index, which is calculated as $LVIDs^3/BSA$, where LVIDs is the end-systolic left ventricular dimension and BSA is body surface area, has been used in the assessment of myocardial function in dogs with MR, but it is of questionable value. The index performs poorly across the range of body sizes observed in the canine species because it is not reasonable to expect a linear relationship between a linear dimension and body weight.
- Doppler echocardiography is used to evaluate velocity, direction, and character of blood flow.
- Doppler evidence of disturbed flow within the left atrium during systole is noninvasive confirmation of the presence of MR (Fig. 6.10). When stroke volume is severely affected by MR or systolic failure, reductions in aortic outflow velocities may be apparent.
- Assessment of the severity of MR can be evaluated quantitatively or, more often, semiquantitatively by Doppler echocardiography.
- Quantitative methods include evaluation of the radius of color Doppler proximal flow convergence and the calculation of regurgitant fractions through volumetric flow analysis; however, these methods are time consuming and have not found widespread clinical application.
- The area of the color Doppler regurgitant jet relative to that of the receiving chamber is one means of semiquantitatively evaluating the severity of valvular regurgitation; however, many physiologic and technical factors influence the size of the jet, and this simple and intuitive method has limitations. The *width* of the regurgitant jet at its origin is another, perhaps more accurate, means of evaluating the severity of regurgitation; a greater width indicates a larger orifice and more severe regurgitation (Figs. 6.11 and 6.12). The appearance of proximal flow convergence—the color Doppler appearance of acceleration through the regurgitant orifice—generally suggests that MR is at least moderate in severity.

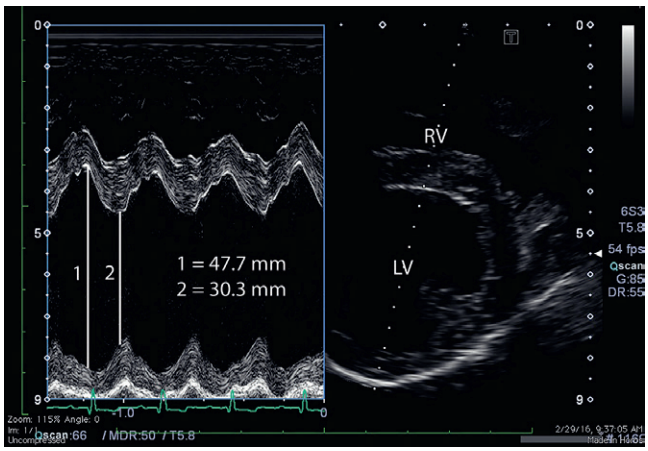


Fig. 6.9 M-mode echocardiogram obtained at the level of the left ventricular papillary muscles from a 14-year-old female-spayed Cocker spaniel weighing 11 kg. There was Doppler evidence of severe mitral regurgitation caused by degenerative valve disease. Left ventricular systolic performance evaluated by fractional shortening (36%) is within the reference range, but the end-systolic left ventricular dimension is markedly enlarged, which provides evidence of systolic myocardial dysfunction.

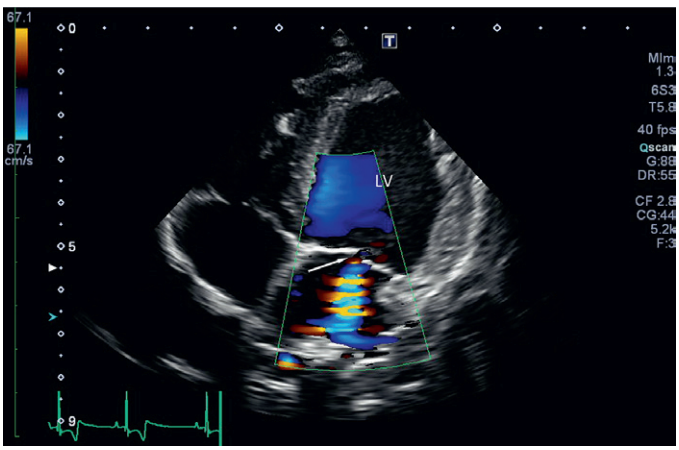


Fig. 6.10 An apical echocardiographic image obtained from a 9-year-old male castrated Miniature Schnauzer with mitral regurgitation (MR) caused by mild degenerative disease. Color-flow Doppler mapping demonstrates mild MR. The color mosaic occupies less than 50% of the area of the left atrium, and the jet is relatively narrow at its origin (*arrow*). *LV*, Left ventricle.

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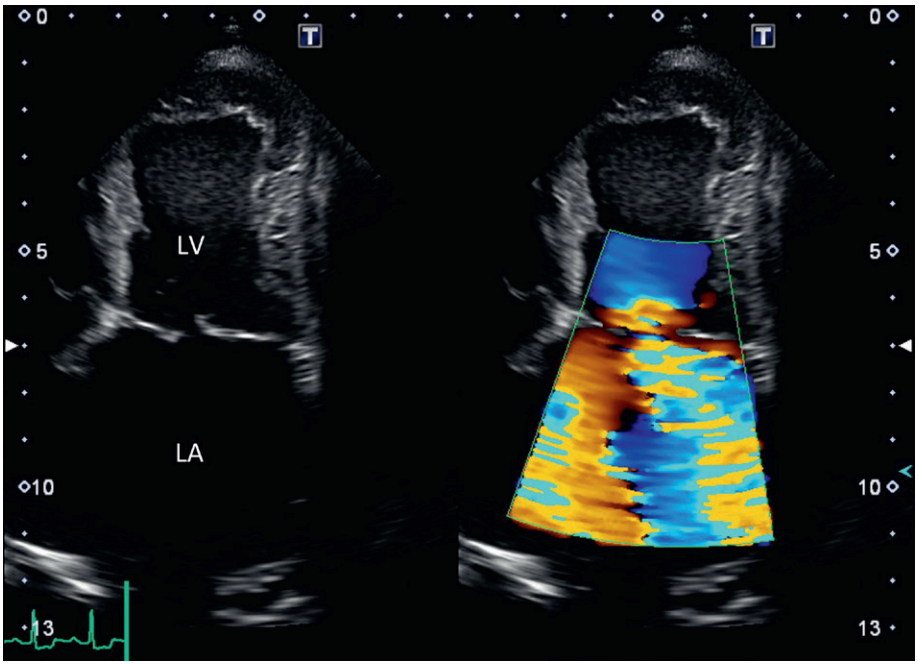


Fig. 6.11 Apical echocardiographic image obtained from a 13-year-old female-spayed mixed-breed dog with (*right*) and without (*left*) superimposed color Doppler map. There is marked mitral regurgitation caused by valvular degeneration. The color mosaic nearly fills the enlarged left atrium (*LA*), and more important, with respect to evaluation of the severity of regurgitation, the jet is broad at its origin. *LV*, Left ventricle.

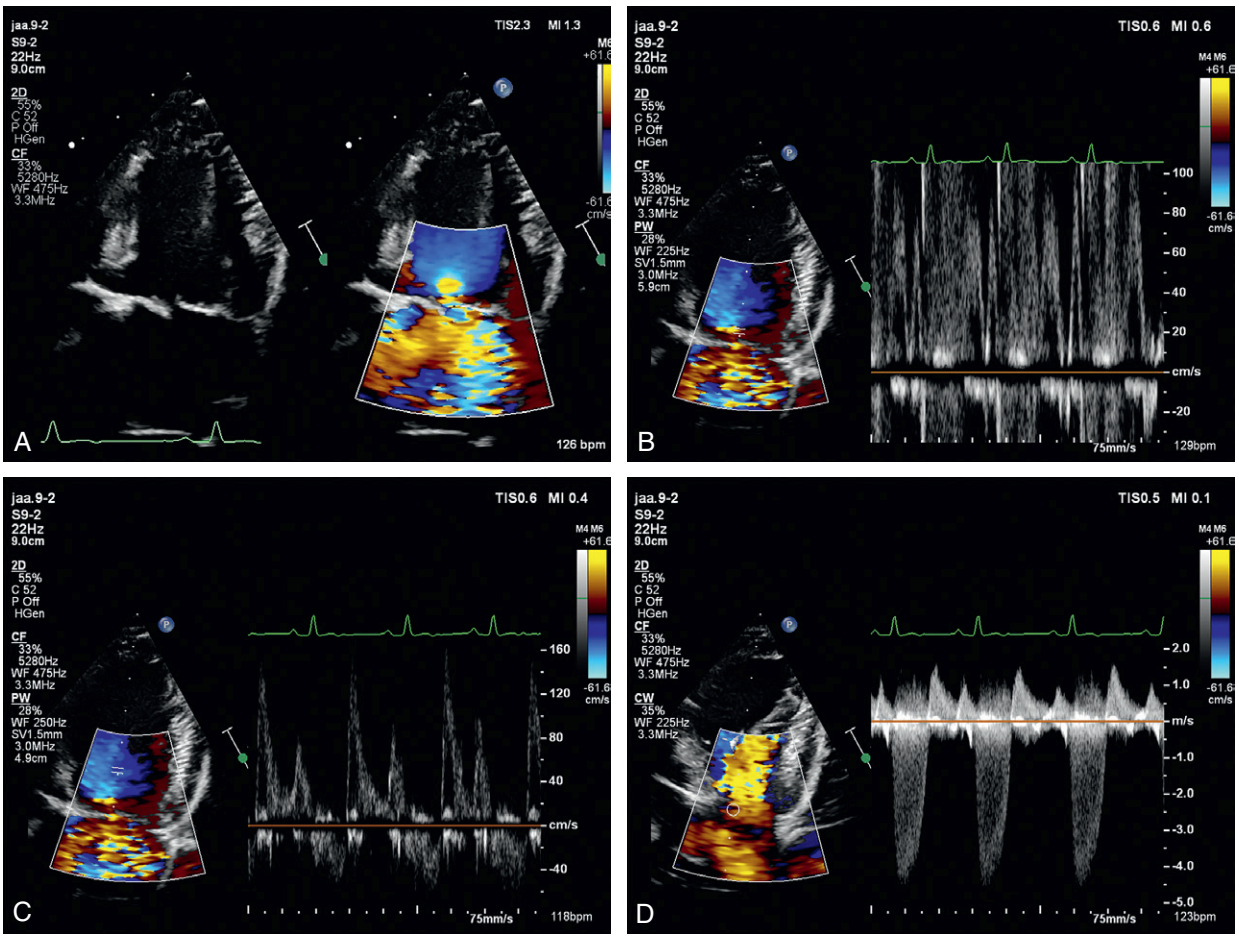


Fig. 6.12 **(A)**, Left apical four-chambered view with (right) and without (left) color Doppler mapping. There is severe mitral regurgitation; the regurgitant orifice is visible, proximal flow convergence is evident, and the vena contract is broad. **(B)** Pulsed-wave Doppler echocardiogram from the same patient. The pulsed-wave sample volume was placed within the left atrium; during systole there is an aliasing multifrequency signal. **(C)** Pulsed Doppler echocardiogram of mitral valve inflow. The sample volume has been placed at the tips of the mitral valve leaflets. Peak velocities are elevated which reflects the large regurgitant volume. **(D)** Continuous-wave Doppler study of the regurgitant jet. The signal is complete and dense, suggesting severe regurgitation; the profile is asymmetric because of abrupt deceleration during late diastole which might reflect left atrial hypertension. *LV*, Left ventricle; *MR*, mitral regurgitation.

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- The density of the regurgitant continuous-wave spectral Doppler signal is roughly proportional to the number of cells that move within the ultrasound beam and is an alternative means of semiquantitatively evaluating regurgitant severity.
- Ultimately it is important that the echocardiographic assessment is clinically relevant. In veterinary patients in whom valvular repair is seldom performed, the effect of valvular regurgitation might be of greater importance than its magnitude. Information regarding chamber size and myocardial function is essential in placing Doppler findings in the appropriate clinical context.
- Technologically advanced echocardiographic methods have recently been investigated in the setting of MVD. Tissue Doppler imaging, for example, which defines the velocity of the myocardium, provides potentially useful information regarding diastolic and systolic myocardial function. Determinations of myocardial strain and strain rate—measurements of the deformation of the heart muscle—also have been investigated in MVD. These modalities may have promise in the evaluation of myocardial function because they are, relative to conventional measures of cardiac dimensions, less dependent on ventricular loading conditions. The potential of real-time three-dimensional echocardiography has also been explored.

Relative Merits of Radiography and Echocardiography in Mitral Valve Regurgitation

MVD exhibits a broad spectrum of severity. Often, the presence of MR is incidental to the presentation, and clinical signs such as cough are not the result of HF or even heart disease but, rather, of primary respiratory disease. Therefore in many cases, the thoracic radiograph provides the most useful diagnostic and prognostic information in patients with MVD. Thoracic radiography not only provides an assessment of cardiac size but also allows visualization of the pulmonary vessels and parenchyma. Thus thoracic radiography provides an indirect assessment of cardiac performance, and while there is a role for pulmonary assessment by point-of-care sonography, radiography remains the "gold standard" diagnostic method for identification of cardiogenic pulmonary edema.

Echocardiography provides a noninvasive means by which to evaluate valvular structure, assess cardiac dimensions, evaluate left ventricular systolic performance, and, with Doppler studies, confirm the clinical diagnosis of MR. While echocardiographic indices of ventricular filling pressures have been proposed, echocardiography does not provide a definitive diagnosis of HF; it can only demonstrate that cardiac disease is sufficiently severe that a diagnosis of HF is plausible. Although the clinical signs associated with MVD may have a sudden onset, the disease process itself is chronic. Therefore left atrial dilation and, often, concurrent left ventricular dilation are expected before the onset of clinical signs. Echocardiographic evidence of MR in the absence of left atrial and left ventricular dilation is seldom of clinical importance. Echocardiographic evaluation plays a role in the optimal management of patients with MVD, and echocardiographic examination was consensus recommendation for patients in stages B, C, and D. However, financial limitations commonly constrain veterinary practice and, in many cases, echocardiography is not essential for the clinical management of many patients with MR. In patients with suspected MR, echocardiography is particularly likely to provide clinically useful information in the following scenarios:

- When the cause of a cardiac murmur is uncertain (for example, patients in which the signalment is atypical, when the murmur may possibly be congenital, or when the murmur is louder over the right, rather than left, hemithorax).
- When it is difficult to discern from thoracic radiographs whether the left atrium is enlarged.
- When sudden deterioration has occurred and rupture of the chordae tendineae or left atrium is suspected.
- When pulmonary hypertension is suspected.

Electrocardiography

- Electrocardiography is primarily useful for the diagnosis of arrhythmias, but it also can provide indirect evidence of chamber enlargement.
- The electrocardiogram is an insensitive gauge of cardiac chamber size. Nevertheless it is likely that findings such as P mitrale are relatively specific; that is, when P waves in the caudal frontal leads (II, III, and aVF) are wide, the left atrium is usually enlarged (Fig. 6.13).
- Arrhythmias can complicate the presentation of MVD. Most often, arrhythmias in MVD take the form of supraventricular tachyarrhythmias that reflect atrial stretch. Atrial premature complexes and paroxysms of atrial tachycardia are relatively common in patients with MVD. Atrial fibrillation develops occasionally and generally indicates advanced disease with marked atrial dilation. Ventricular arrhythmias (ventricular premature complexes) may develop in association with left ventricular dilation and myocardial fibrosis.

Blood-Borne Biomarkers

- Biomarkers are objectively determined characteristics that potentially have a role in diagnosis, risk stratification, evaluation of disease progression, and assessment of response to therapy. A number of blood-borne biomarkers, including endothelin, troponin I, atrial natriuretic peptide, and B-type natriuretic peptide (BNP), have been investigated in the settings of MVD and canine HF. BNP is released by atrial and ventricular cardiomyocytes in response to

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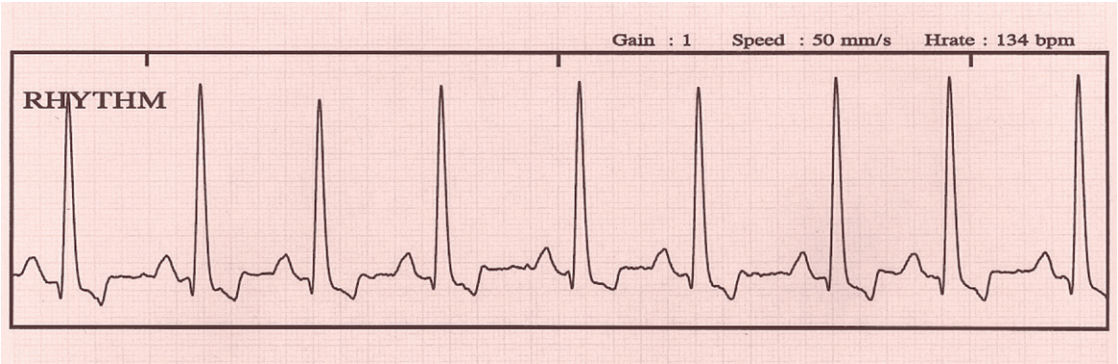


Fig. 6.13 An electrocardiogram recorded from a 13-year-old Beagle with physical findings of mitral regurgitation (MR). There is P mitrale, and the R amplitude exceeds 3 mV, suggesting left atrial enlargement and left ventricular hypertrophy, respectively. (Lead II, 50 mm/s, 1 mV = 1 cm.)

increases in ventricular filling pressures and therefore is a potential blood-borne diagnostic marker of the HF state.

- BNP concentration is associated with the severity of canine degenerative MVD, and statistically significant differences between BNP concentrations from healthy dogs and dogs with MVD have been identified. It is relevant, however, that the finding of statistically significant intergroup differences does not necessarily imply that a variable, in this case BNP, usefully discriminates between groups. Elevated BNP concentrations do distinguish patients with respiratory distress caused by HF from those with distress caused by respiratory tract disease with sensitivity and specificity that exceed 80%.
- The prognostic potential of BNP determination has also been evaluated in canine cardiac disease. Not only is BNP concentration related to severity of disease, but also, longitudinal investigations have disclosed a relationship between mortality and NT-ProBNP (*N*-terminal pro–B-type natriuretic peptide) concentration in dogs with MVD. Furthermore, a decrease in BNP concentration in association with medical therapy for MVD is predictive of outcome.

THERAPY

Stage A Mitral Valve Disease

- Patients that have stage A MVD do not have clinically identifiable valvular abnormalities but are predisposed to the development of mitral disease. Known predispositions related to genetics and relevant therapies have not been identified.

Stage B Mitral Valve Disease

- A theoretic ideal treatment for MVD could be used to prevent or reverse myxomatous degeneration. Unfortunately drug therapy that affects this pathologic process has not been identified. However, there is evidence that administration of pimobendan delays the onset of congestive signs in small-breed dogs with chamber enlargement caused by subclinical mitral valve regurgitation.
- The Evaluation of Pimobendan In dogs with cardiomegaly caused by preclinical mitral valve disease (EPIC), was a randomized, blinded placebo-controlled clinical trial designed to test the hypothesis that administration of pimobendan delays the onset of pulmonary edema in canine patients with asymptomatic mitral valve disease. The inclusion criteria of the trial are relevant because it was the result of this trial that prompted revision of the definition of stage B2 in the 2019 iteration of the ACVIM consensus statement. The criteria included:
  - Systolic murmur of grade 3/6 or greater intensity
  - Radiographic cardiomegaly defined by a VHS >10.5
  - Echocardiographic left atrial/aortic ratio >1.6
  - Echocardiographic end-diastolic left ventricular dimension indexed to allometrically scaled body size >1.7
- The primary endpoint of the trial was time until development of pulmonary edema or death/euthanasia. Interim analyses met a priori criteria for stopping the trial. The median time until endpoint for those receiving pimobendan was approximately 15 months greater than for those that received a placebo.
- The evidence in favor of administration of pimobendan to dogs that meet criteria that define the revised definition of stage B2 is strong and this was a Class I recommendation from the ACVIM Consensus Panel.
- The possibility that enalapril might delay the onset of HF in subclinical MVD has been addressed by two separate clinical trials. In both the Scandinavian Veterinary Enalapril Prevention (SVEP) trial and the Veterinary Enalapril Trial to Prove Reduction in Onset of Heart Failure (VETPROOF), dogs with subclinical MR were randomized to receive a placebo or **enalapril**. Neither trial demonstrated a statistically significant effect of enalapril on time to development of CHF. Although the result of the VETPROOF

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was not statistically significant with respect to the primary endpoint, the data did show a tendency toward a favorable treatment effect. Atrial enlargement was an inclusion criterion for VETPROOF but not for the SVEP trial; however, it is worthy of consideration that both trials included patients with relatively mild MR. Patients with mild and slowly progressive disease tend to experience few events of interest (e.g., death or occurrence of HF) even during relatively long periods of follow-up. The inclusion of mildly affected patients in a clinical trial might mask a treatment effect that is evident only for patients with severe disease. It is therefore possible that a subpopulation of preclinical patients with severe MR and cardiomegaly would benefit from ACE inhibition. Although this hypothesis has not been specifically tested, it is partly refuted by the results of the SVEP trial, in which a treatment effect was not observed in the subset of dogs that had radiographic cardiomegaly at study entry.

- Definitive evidence that ACE inhibitors improve prognosis in subclinical MVD is lacking. Administration of ACE inhibitors in stage B2 can be considered; it was not a consensus recommendation by the ACVIM panel.

Cough in the Absence of Radiographic Edema

- Some dogs with MVD have a cough that might partly result from compression of the mainstem bronchi by an enlarged left atrium. This cough, or cough related to primary respiratory tract disease, can be indistinguishable but in either case can occur in the absence of pulmonary edema.
- Radiographically, there is an enlarged cardiac silhouette with distinct evidence of left atrial enlargement. The mainstem bronchi may be noticeably narrowed.
- Primary respiratory tract diseases including tracheal collapse and chronic bronchitis are common in the same patient group that has MVD. With very few exceptions, clinical signs related to MVD do not occur in the absence of radiographic left atrial enlargement.
- When radiographic findings suggest that the cause of cough is airway compression or noninfective airway disease but not pulmonary edema, the use of an antitussive such as **hydrocodone** is rational.
- Interpretation of thoracic radiographs is difficult and in geriatric patients, distinguishing between mild interstitial edema and increases in radiographic opacity that might result from incomplete lung inflation or senescent change of the lung is challenging. Diuretic administration reduces cardiac volumes and is likely to reduce the severity of clinical signs related to pulmonary venous hypertension. There is no evidence that "prophylactic" diuretic administration—prior to the development of edema—is beneficial, but its empirical use in patients with respiratory signs associated with cardiac enlargement is reasonable when there is uncertainty regarding the presence or absence of edema and renal function is normal.

If there is compelling evidence of a favorable effect, concurrent long-term administration of pimobendan and an ACE inhibitor as described below is reasonable.

Stage C Mitral Valve Disease

- When MR causes clinical signs in humans, it is treated surgically. Mitral valve repair with preservation of chordal attachments is preferred to replacement of the valve with a prosthesis. Surgical treatment of canine mitral valve disease during cardiopulmonary bypass has been reported, but availability is limited to a few centers worldwide. A novel device that is guided to the mitral valve using a transapical approach after intercostal thoracotomy allowing beating-heart edge-to-edge repair is under investigation.
- In general, HF caused by canine MVD is treated medically (Table 6.2). Unless the cause can be definitively treated, HF is a terminal syndrome. Therefore medical management is intended to alleviate clinical signs and prolong life. Drug therapy for HF consists of interventions that manipulate the determinants of cardiac output and blunt the maladaptive neuroendocrine response to cardiac dysfunction.
- In CHF caused by MR, left ventricular filling pressure (preload) is excessive, and the consequent increase in venous pressures causes tissue fluid to weep into the pulmonary interstitium and alveoli. Drugs that reduce intravascular volume, such as diuretics, or that increase venous capacitance, such as nitroglycerin, are therefore mainstays of therapy.
- **Nitroglycerin** is usually administered transdermally and is sometimes used as short-term therapy in patients with fulminant edema or occasionally as adjunctive therapy in patients with advanced disease. The efficacy of transdermal nitroglycerin is uncertain, and in dogs with experimentally induced MR, the effect of nitroglycerin on filling pressure did not differ from that of a placebo.
- **Furosemide**, a potent agent that acts on the loop of Henle, is the diuretic that is used most often in veterinary practice. It can be administered orally or parenterally; the route of administration is based on the patient's clinical status. The resultant decrease in intravascular volume reduces left ventricular filling pressures and allows lymphatic drainage of tissue fluid and resolution of edema.
- **Torsemide** (torasemide) has recently been considered as an alternative to furosemide. Torsemide also is a loop diuretic. It is more potent than is furosemide, has pharmacokinetic properties that might be advantageous, and might have favorable neuroendocrine effects. The results of two clinical trials that enrolled canine patients with heart failure have been

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Table 6.2 Suggested Strategies for Diagnostic and Therapeutic Management of Canine Degenerative Valvular Disease

	STAGE A MVD	STAGE B MVD	STAGE C MVD	STAGE D MVD
Diagnostic approach	Diagnostic screening of predisposed breeds and genetic counseling are the only management strategies relevant to this stage of the disease.	Thoracic radiography and echocardiography for patients with murmur intensity of grade 3/6 or greater. ECG if auscultation suggests pathologic arrhythmia.	Thoracic radiography and echocardiography: determined by clinical circumstances and availability. ECG if auscultation suggests pathologic arrhythmia.	Thoracic radiography and echocardiography: determined by clinical circumstances and availability. ECG if auscultation suggests pathologic arrhythmia.
Therapeutic approach		Administration of pimobendan for patients that meet criteria that identify stage B2. Medical therapy is not indicated for patients with stage B1 MVD	"Quadruple Therapy" consisting of: • Furosemide • An ACE inhibitor • Pimobendan • Sprironolactone	Treatment must be tailored to the individual. The following are considerations: • Substitution of torsemide for furosemide • Amlodipine • Off-label, three times daily administration of pimobendan

ACE, Angiotensin-converting enzyme; *ECG*, electrocardiography.
Modest dietary salt restriction is reasonable in stages C and D. Home monitoring resting/sleeping respiratory rates is advisable in stages B2, C, and D. Periodic monitoring of renal function in patients receiving diuretic therapy and ACE inhibitors is recommended.

published; torsemide was compared to furosemide. Both trials demonstrated noninferiority of torsemide with respect to the trials' primary endpoints. There also was the suggestion that, relative to furosemide, torsemide might decrease cardiac mortality in canine patients with HF. However, both of these trials were of relatively brief duration and neither was primarily designed to determine mortality. Studies in people that were specifically intended to evaluate survival in patients receiving torsemide or other diuretics have not revealed differences in mortality. And evidence-based guidelines for treatment of people with heart failure recommend loop diuretics but do not favor one over others. When substituting torsemide for furosemide, an initial dose of 10% to 20% of the furosemide dose given once or twice daily is used.

- Duretics reduce preload; this decrease in ventricular filling pressures is generally well tolerated by patients with ventricular dilation and has obvious benefits when edema is present. However, excessive diuresis can result in hypotension related to low cardiac output, prerenal azotemia, and electrolyte disturbances. It is generally thought that the optimal dose of furosemide is the lowest one that controls signs of congestion.
- More importantly, many dogs with clinically evident MVD cough in the absence of pulmonary edema, and many of these patients have concurrent primary respiratory tract disease. Therefore aggressive diuresis after radiographically demonstrated resolution of pulmonary edema is to be avoided. In most cases of HF caused by MVD, the administration of furosemide rapidly and effectively resolves signs. Failure of patients with MR and respiratory distress to respond favorably to diuretic administration should cause the practitioner to question the diagnosis of HF and to consider the possibility that primary respiratory tract disease or pulmonary arterial hypertension is the cause of clinical signs.
- Most patients that have radiographic pulmonary edema caused by MR require lifelong diuretic therapy. Early in the course of the syndrome, an oral dose of 1 to 2 mg/kg furosemide q12h may be adequate, although the inevitable progression of MR and HF and renal tubular adaptations ultimately necessitate higher doses.
- Moderate dietary salt restriction is suggested for patients with CHF caused by MVD.
- **Pimobendan** is an "inodilator" that has complex pharmacologic properties. It inhibits phosphodiesterase and therefore causes vasodilation and an increase in the inotropic state. In addition, pimobendan increases the sensitivity of the contractile apparatus to available calcium, which also contributes to the inotropic effect. This latter property may be favorable because an increased inotropic state is associated with relatively low cost in terms of myocardial oxygen consumption.
- A randomized clinical trial (QUEST) compared pimobendan with benazepril against a background of conventional therapy that consisted of furosemide, digoxin, or both in dogs with stage C MVD. With respect to survival, pimobendan was superior

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to benazepril. Post hoc analyses of the data from this trial provide evidence that, relative to benazepril, pimobendan delayed intensification of cardiac therapy.

- The effect of pimobendan, when used with or without ACE inhibition in dogs with valvular disease, has recently been evaluated; the results suggest that the combination of pimobendan and an ACE inhibitor is not superior to pimobendan alone. However, other studies have provided evidence of effectiveness of ACE inhibitors in stage C mitral valve disease and administration of "triple therapy"—a diuretic, and ACE inhibitor and pimobendan—or "quadruple therapy"—those aforementioned together with spironolactone—has become standard and is consistent with the consensus recommendation included in the ACVIM guidelines.
- When atrial tachyarrhythmias, particularly atrial fibrillation, complicate MVD, the use of dilatiazem with or without digoxin (0.22 mg/m^2 PO q12h) for control of ventricular response rate is reasonable.

Neuroendocrine Modulation

- ACE inhibitors are part of the standard therapeutic approach to HF caused by MR. It is likely that the favorable effect of ACE inhibition is not simply the result of vasodilation. In addition to this mechanical effect, ACE inhibition serves to protect the heart from the apparently detrimental effects of RAS activation.
- Pharmacologic ACE inhibition generally is not complete, and because aldosterone may contribute to the development of myocardial fibrosis, more complete suppression of the RAS may yield positive results. Accordingly, the use of **spironolactone**, a weak diuretic that antagonizes the effect of aldosterone, is considered adjunctive therapy for patients with HF caused by MVD. In humans with HF, the use of subdiuretic doses of spironolactone prolongs survival.
- A placebo-controlled clinical trial provided evidence that administration of spironolactone to dogs receiving an ACE inhibitor with or without furosemide or digoxin for HF caused by MVD improved outcome. However, for reasons that relate to study design, the publication of the trial results provoked controversy.
- More recently, the results of a double-blind, randomized clinical trial designed to compare the effect of a fixed dose tablet consisting of a combination of benazepril and spironolactone to that of benazepril alone have been reported. Canine patients that had developed stage C mitral valve disease were randomized to receive either combination therapy or benazepril alone. All patients received furosemide. The combination was superior to benazepril alone and is now licensed for use in North America. This formulation might have advantages with regard to client compliance as "quadruple therapy" can be carried out through administration of three, rather than four, tablets.

Therapy of Stage D by Advanced Mitral Valve Regurgitation

- In the most recent Consensus Statement, stage D is defined by diuretic dose; those patients that require more than 8 mg/kg/day furosemide—or the equivalent dose of torsemide—to relieve signs caused by congestion when receiving standard therapy are considered to be in stage D heart failure.
- In stage D, torsemide often is substituted for furosemide. Alternatively, the use of triple diuretic therapy—the combination of **furosemide** with a **thiazide** and a potassium-sparing diuretic such as **spironolactone**—can be considered for patients that require high doses of furosemide to remain free of congestive signs. The use of three different diuretic agents interferes with nephron function at anatomically and functionally distinct sites; together, the drugs may have synergistic effects, allowing the use of lower doses of the individual agents. In addition, the use of a potassium-sparing agent such as spironolactone serves to limit some of the adverse effects that are associated with the use of high doses of loop diuretics such as furosemide.
- Despite proved efficacy in the management of HF caused by MVD, the ACE inhibitors are not potent vasodilators. In some patients with HF caused by severe MVD, the use of the vasoselective calcium channel blocker **amlodipine**, in addition to an ACE inhibitor, may be helpful. When vasodilators are used in addition to ACE inhibitors, the initial dose should be low; ideally, the dose is titrated to effect based on serial blood pressure determinations.

SUMMARY OF THERAPEUTIC APPROACH

Stage A

Diagnostic screening of predisposed breeds and genetic counseling are the only management strategies relevant to this stage of the disease.

Stage B1

Medical therapy is not indicated for patients in stage B1.

Stage B2

The inodilator, pimobendan, delays the onset of clinical signs in asymptomatic canine patients that have cardiac chamber enlargement resulting from MVD. The evidence to support this contention is the result of a randomized, placebo-controlled and blinded clinical trial; relative to a placebo, administration of pimobendan delayed the onset of pulmonary edema. The inclusion criteria for that trial identify patients that, on average, benefit from administration of pimobendan. They are:

- Grade 3/6 (or louder) murmur caused by MVD
- VHS > 10.5

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- Echocardiographic evidence of left atrial and left ventricular enlargement
  - LA:Ao > 1.6
  - nLVDD > 1.7
    - nLVDD = LVIDD/[body weight (kg)]$^{0.294}$

Pimobendan is administered at a daily dose of 0.5 mg/kg PO, divided into two not necessarily equal doses. In some patients with stage B2 MVD, there might be a role for ACE inhibition. Home monitoring of resting or sleeping respiratory rates might allow early identification of decompensation.

Stage C

Patients with stage C have, or in the past have had, clinical signs. In practical terms, the onset of stage C generally is defined by the development of cardiogenic pulmonary edema. In patients with stage C MVD, recommended therapy consists of:

- Diuretic therapy—generally, furosemide
- ACE inhibition—most often, enalapril or benazepril
- Pimobendan
- Spironolactone

Home monitoring of resting or sleeping respiratory rates might allow early identification of clinical deterioration and can usefully guide diuretic therapy.

Stage D

Patients with stage D have medically refractory heart failure which, based on the ACVIM Guidelines, is identified when a furosemide dose in excess of 8 mg/kg is required to prevent congestive signs in patients receiving standard therapy for stage C. Therapeutic strategies used in this stage of disease include intensification of diuretic therapy—"triple diuretic therapy" or substitution of furosemide with torsemide—higher, off-label doses of pimobendan and, in some cases, additional vasodilators such as amlodipine. The evidence to support these interventions generally is weak.

COMPLICATIONS OF MITRAL VALVE DISEASE AND ITS TREATMENT

Cardiorenal Syndrome

- The cardiorenal syndrome has been defined in different ways, but the term is generally used to describe worsening renal function in patients with cardiac disease. A decrease in stroke volume related to declining cardiac performance likely contributes to the development of azotemia in the setting of HF. However, the relationship between cardiac dysfunction and worsening renal function is complex; invasively acquired data from humans demonstrate that increases in cardiac output do not prevent worsening of renal function or necessarily improve long-term outcome.
- Monitoring of renal function is particularly important for patients with stage C or D HF that generally are receiving diuretics and ACE inhibitors. ACE inhibitors are not generally thought to be directly nephrotoxic; however, ACE inhibition results in relatively selective dilation of the efferent arteriole of the nephron, a hemodynamic effect that can predispose to the development of prerenal azotemia. Those patients with preexisting renal disease and those receiving overly aggressive doses of diuretics seem most likely to have azotemia. In addition, patients with severe cardiac dysfunction that are critically dependent on the effects of angiotensin II to maintain glomerular filtration fraction may also have azotemia when ACE inhibitors are administered.
- There are few circumstances under which diuretic therapy can increase stroke volume and renal blood flow; in general, diuretics decrease filling pressures and potentially decrease cardiac performance. In contrast, judicious vasodilation in the setting of MR may increase stroke volume. Thus the development of azotemia in patients that do not at the time have pulmonary edema is usually managed first by a cautious reduction in the diuretic dose. Should creatinine values fail to decrease, the diuretic can be discontinued, but the patient's respiratory rate and character must be carefully monitored. If clinical signs are associated with azotemia while patients are receiving the minimum diuretic dose shown to eliminate congestive signs, euthanasia is a consideration.

Rupture of Chordae Tendineae

- Rupture of chordae tendineae is a relatively common complication of MVD. When a primary chorda ruptures, the attached mitral leaflet becomes suddenly flail (Fig. 6.14), potentially resulting in catastrophic MR, marked elevations in ventricular filling pressures, and fulminant pulmonary edema. Rupture of minor chordae may result in less impressive clinical signs or be subclinical.
- Published echocardiographic data suggest that rupture of chordae tendineae in patients with preexisting MR and cardiac enlargement is relatively common. It can result in clinical decompensation of varying degrees of severity; echocardiographic evidence of past chordal rupture in patients that do not have a history of acute clinical deterioration is fairly often identified. In contrast, the development of acute pulmonary edema resulting from chordal rupture in patients that have normal cardiac dimensions is uncommon.
- Acute CHF caused by chordal rupture is treated similarly to acute or decompensated HF caused by other disorders; however, there may be a particular role for the intravenous administration of nitroprusside in addition to parenteral diuretic administration.
- Prognosis depends on numerous factors; the most important is probably response to therapy.

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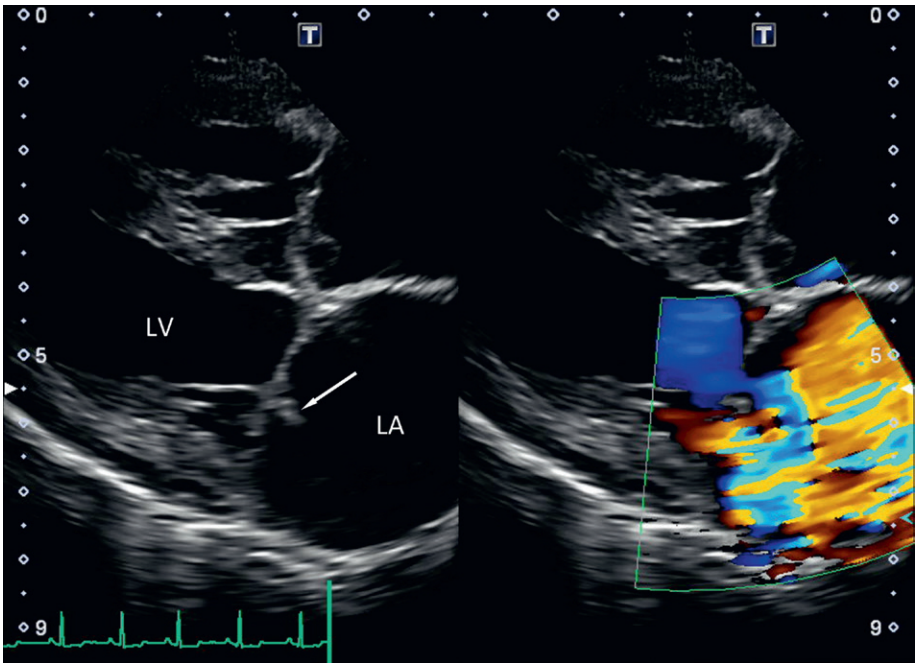


Fig. 6.14 Right parasternal long-axis echocardiographic images obtained from a 14-year-old male castrated Chinese Crested dog with (*right*) and without (*left*) superimposed color Doppler map. The anterior mitral valve leaflet (*arrow*) is partially flail—a segment of the leaflet is perpendicular to the plane of the mitral annulus—because of past rupture of a chordae tendineae. *LA*, Left atrium; *LV*, left ventricle.

Left Atrial Rupture

- Left atrial rupture is an uncommon complication of advanced MVD that generally is associated with marked left atrial dilation. Cocker Spaniels and Dachshunds may be predisposed to left atrial rupture. Unsurprisingly, a complete and full-thickness tear of the atrial wall can be lethal. However, compressive pericardial effusions and structures in the pericardial space assumed to represent thrombi are occasionally evident when patients with severe MR are subject to echocardiographic examination. These findings provide indirect evidence of past partial left atrial rupture.
- The clinical consequences of left atrial rupture are variable; marked clinical deterioration or sudden death can be observed, but a few patients seemingly have a subclinical left atrial rupture. A recent publication described a series of 11 dogs with presumed left atrial rupture for which the median survival was 203 days. Rupture of the interatrial septum resulting in an atrial septal defect also has been reported as a complication of MVD.

Pulmonary Hypertension

- Pulmonary hypertension (PH) occasionally complicates the clinical presentation of MVD in the dog. Doppler studies can provide noninvasive estimates of pulmonary artery pressure. Doppler echocardiographic evidence of tricuspid valve regurgitation (TR) is commonly observed in patients with MVD. The velocity of the TR jet is related to the systolic pressure difference between the right atrium and the right ventricle by the simplified Bernoulli equation ($\Delta P = 4v^2$, where ΔP is the pressure difference and v is the velocity of the regurgitant jet). Provided pulmonary stenosis is excluded by Doppler evaluation of the right ventricular outflow tract, right ventricular and pulmonary artery pressures are equal during systole. Thus measurement of the velocity of the TR jet can provide noninvasive estimates of systolic pulmonary artery pressure (Fig. 6.15). While this method is often used, studies in both humans and dogs have called its accuracy into question. As a result, the ACVIM Consensus Panel that published guidelines for the management of pulmonary hypertension advocated a "probabilistic" approach to the diagnosis of pulmonary hypertension. The velocity of TR is considered together with echocardiographic variables and a low, intermediate, or high echocardiographic probability that there is pulmonary hypertension is assigned.
- The cause of PH associated with MVD is probably multifactorial. On a pathophysiologic basis, PH can be described as pre- or postcapillary. The primary function of the right ventricle is to propel the stroke volume through the pulmonary vascular tree to the left atrium. Elevations in left atrial pressure cause commensurate increases in right ventricular systolic pressure; sometimes known as "passive" PH, elevations in pulmonary artery pressures that result from this mechanical effect characterize postcapillary PH. The tendency for left atrial hypertension

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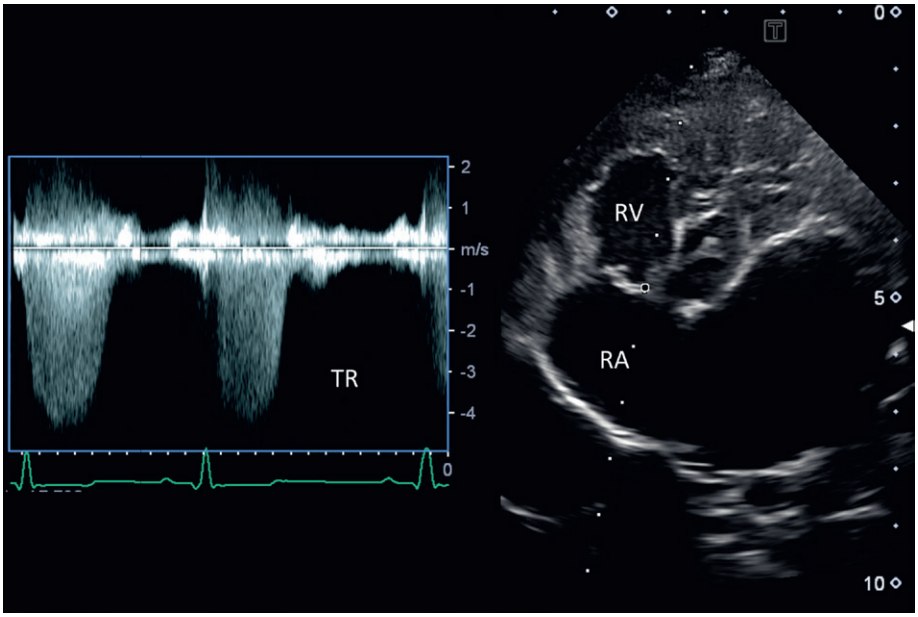


Fig. 6.15 Continuous-wave Doppler study performed with the Doppler cursor through the right atrium of a 14-year-old male castrated Chinese Crested dog with severe mitral regurgitation (MR) caused by valvular degeneration. There is tricuspid valve regurgitation (*TR*); the peak velocity of the TR jet exceeds 4 m/s and the estimated systolic pulmonary artery pressure is therefore greater than 64 mm Hg. *RA*, Right atrium; *RV*; right ventricle.

to cause postcapillary PH is probably the primary explanation for high pulmonary artery pressures in most cases; however, in some patients, the increase in estimated pulmonary artery pressure seems to be disproportionate to any credible estimate of left atrial pressure. Identification of "combined pulmonary hypertension" in which there are etiologic contributions of both postcapillary PH and precapillary pulmonary arteriolar constriction, requires invasive studies. In some cases, pulmonary hypertension might be also related to the presence of concurrent pulmonary or airway disease, which can cause precapillary PH through hypoxic vasoconstriction.

- There is no established therapy for patients with pulmonary hypertension associated with MVD.
- As initial therapy, interventions that reduce left atrial pressure are reasonable. Diuretic therapy should be tailored to rid the patient of radiographic evidence of pulmonary congestion or edema. Alveolar hypoxia is a stimulus for constriction of the pulmonary arterioles. If bronchoconstriction associated with primary respiratory disease has contributed to the development of pulmonary hypertension, bronchodilators may be helpful.
- When pulmonary congestion is effectively controlled and clinical signs such as episodic weakness or ascites can plausibly be related to pulmonary hypertension, the use of sildenafil can be considered. Sildenafil is an inhibitor of phosphodiesterase-5 that has a relatively selective effect on pulmonary arterioles (see Chapter 15).
- Caution is justified; a decrease in pulmonary vascular resistance caused by administration of sildenafil can increase pulmonary venous return, raise filling pressures, and precipitate the development of pulmonary edema.

The prognosis associated with the development of MVD depends on numerous factors. Most patients with MR caused by MVD succumb to noncardiac disease. In fact, in two retrospective investigations in which observation periods were generally longer than 2 years, it was not possible to calculate median survival of patients with subclinical MVD because so few patients died during follow-up. Clinical trial data suggest that the median time for progression from stage B2 to stage C MVD is approximately 2 years. Stage C MVD is generally terminal, and the median survival of patients receiving furosemide and pimobendan is close to 9 months. Echocardiographically determined left atrial enlargement and increased levels of BNP and troponin I are associated with poor outcome.

INFECTIVE ENDOCARDITIS

Infective endocarditis (IE) occurs occasionally in dogs and rarely in cats. The prognosis is generally grave and this emphasizes the importance of detecting IE, a disease that sometimes poses a considerable diagnostic challenge.

PREVALENCE AND INCIDENCE

- IE is a relatively uncommon disease that is observed occasionally in dogs but rarely in cats. Mural endocarditis and infection of the tricuspid valve are observed occasionally, as is infection of endocardial

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pacing leads; however, bacterial infection of the aortic or mitral valve leaflets is most common.
- Male, middle-aged, large dog breeds, including German Shepherds and Boxers, are affected most often.
- In humans, the presence of a congenital cardiac malformation is a risk factor for the development of IE. An association between subvalvular aortic stenosis and IE of the aortic valve has been demonstrated, and it is likely that subvalvular aortic stenosis and, perhaps, other congenital malformations are important factors in the epidemiology of IE in dogs.

ETIOPATHOGENESIS

- On the basis of experimental studies, it is likely that the following factors are important in the pathogenesis of infective valvular endocarditis:
  - Endocardial damage (which may result from valvular insufficiency, stenosis, or a shunting lesion)
  - Activation of clotting factors
  - Bacteremia and colonization of a noninfective thrombus
- Experimentally, the development of a noninfective thrombus precedes valvular infection. Episodes of bacteremia can result in infection of the thrombus and in the initiation of a variably aggressive inflammatory process that results in distortion and destruction of the valve leaflets and their associated structures.
- In clinical cases of canine IE, a congenital cardiac malformation may represent a predisposition for the development of the disease. Prostatitis, pyelonephritis, or even dental disease can be a source for the development of bacteremia; often, however, the site of bacterial entry into the bloodstream remains undiscovered.
- Degenerative valvular disease has no known association with IE. Despite the prevalence of dental disease in patients with MR caused by MVD, IE is extremely uncommon in this group. Indeed, the possibility that there is an association between canine periodontal disease and IE has been systematically investigated. A large retrospective cohort study identified a relationship between IE and periodontal disease. However, the absence of echocardiographic corroboration of diagnoses, together with the improbable incidence data reported for specific cardiac diseases, makes the conclusion that periodontal disease predisposes to IE dubious. A smaller case-control study in which cases were rigorously defined and documented found no such relationship.
- Gram-positive bacteria such as streptococci and staphylococci are most often implicated in the development of IE. Valvular infection with gram-negative organisms such as *Escherichia coli* is less common.
- With regard to the etiology of IE, there has been interest in the fastidious, intracellular organisms of the genus *Bartonella*. Four species of *Bartonella* have been documented to cause canine IE. Although geographic distribution of *Bartonella* IE may not be uniform, *Bartonella* is an important cause of IE in northern California and possibly elsewhere. Retrospective evaluation of case data from canine patients that had IE in the United Kingdom did not disclose *Bartonella* as a cause. Patients with *Bartonella* infection are often concomitantly seropositive for tick-borne diseases, and *Bartonella* itself may be arthropod borne. *Bartonella* species are difficult to culture with standard microbiological techniques. The diagnosis depends on documentation of serum antibodies to *Bartonella*, or, more specifically, demonstration of *Bartonella* antigens through polymerase chain reaction testing performed on bacterial isolates or valve tissue.

PATHOPHYSIOLOGY

- The clinical signs of IE relate to sepsis, thromboembolism, and cardiac dysfunction. IE results in intermittent shedding of bacterial organisms into the bloodstream, resulting in episodes of bacteremia. Signs of sepsis, including pyrexia and rarely circulatory collapse, may be observed.
- Sequelae of sepsis related to chronic antigenic stimulation and consequent development of immune complex disease are observed fairly commonly in IE. Polyarthritis is observed often, and glomerulonephritis can also develop.
- In canine IE, it is often the destruction of the valve leaflets and associated structures that is of greatest clinical importance. The development of infected thrombi results in failure of the valve leaflets to coapt. Occasionally, perforation of the valve leaflets contributes to valvular incompetence. The hemodynamic consequences of MR have been discussed previously.
- IE of the aortic valve typically results in aortic valve incompetence, which is a potentially catastrophic hemodynamic lesion. When the aortic valve becomes incompetent, the left ventricle is filled during diastole by the pulmonary venous return and by the blood that enters through the regurgitant orifice. When severe, the increase in ventricular filling pressures (end-diastolic ventricular pressures) is reflected back on the pulmonary venous circulation, resulting in pulmonary congestion and edema. In contrast to MR, aortic valve insufficiency (AI) causes a substantial increase in left ventricular afterload and therefore myocardial oxygen demand. As a result, myocardial dysfunction (cardiomyopathy of overload) is observed commonly and early in the course of aortic valve IE.
- Embolization of fragments from the endocarditis lesion occurs commonly. Sites where infected thrombi lodge include the spleen, the kidney, and occasionally the brain. Most often, embolization of

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the spleen is clinically silent; infarction of the kidneys or central nervous system can be catastrophic, resulting in renal failure and nervous system signs such as head tilt. Embolization of joints, resulting in bacterial arthritis, can also occur.

CLINICAL PRESENTATION

History

- IE in dogs is observed most commonly in a subacute form. In these cases, historical evidence of prior illness or infectious disease may be lacking. A congenital cardiac murmur may or may not have been detected. IE is also observed in an acute form.
- Clinical signs in subacute IE are often vague; lameness, inappetence, dyspnea, syncope, and exercise intolerance are observed commonly. The lameness is often mild and may be difficult to localize. The embolization of infected thrombi to joints contributes to lameness, although immune complex arthropathy may be of equal etiologic importance.
- Clinical signs of sepsis may be more prominent in patients that have acute IE. The sudden onset of fever and the development of a new cardiac murmur in the critically ill suggest the presence of IE.

Physical Findings

- Fever is a common but not necessarily consistent finding in patients with IE. Published cases series of canine IE reflect the experience of referral centers and are therefore biased. At some point in the natural history of the disease, it is likely that most patients are pyrexic; however, fever associated with IE can be intermittent and may resolve before clinical presentation.
- The pulse rate is often elevated because some degree of cardiac dysfunction is commonly present at the time of presentation.
- The respiratory rate is elevated, and respiratory distress is usually apparent in patients that have pulmonary edema.
- A cardiac murmur is present in most patients with established valvular infection. MR results in a systolic murmur that is most easily heard over the left cardiac apex. Aortic valve IE usually results in a diastolic decrescendo murmur that is typically heard most easily over the left cardiac base. A concurrent systolic murmur related to the increase in left ventricular stroke volume associated with AI results in a murmur known as a "to-and-fro" or "bellows" murmur.
- Diastolic murmurs are uncommon in veterinary patients and are usually soft; consequently they may escape detection. Despite this, they are of considerable diagnostic and prognostic importance; acquired diastolic murmurs in dogs most often result from aortic valve IE.
- When moderate or severe AI is present, the arterial pulses are hyperkinetic or bounding; this physical finding should prompt consideration of IE whenever signalment and other clinical findings are suggestive.

DIAGNOSTIC FINDINGS

Thoracic Radiography

- Radiographic findings in IE are variable. Cardiac enlargement is apparent when valvular lesions have imposed a chronic volume overload on the heart. Pulmonary congestion or edema is commonly observed (Fig. 6.16). Occasionally, IE results in severe and acute AI; this is one of the few clinical scenarios in which cardiogenic pulmonary edema is observed in association with a normal or minimally enlarged cardiac silhouette.

Electrocardiography

- There are no electrocardiographic findings that are diagnostic of IE. However, all manner of cardiac arrhythmias can be observed in association with this disease. Ventricular tachyarrhythmias, including ventricular tachycardia, are relatively common, and supraventricular tachyarrhythmias, including atrial fibrillation, are also observed.
- The association of third-degree atrioventricular block with IE deserves mention. Presumably, an aggressive aortic lesion can invade the interventricular septum or, alternatively, embolize the nodal coronary artery, resulting in destruction of the atrioventricular node or bundle. This catastrophic complication is uncommon, but lesser degrees of atrioventricular block or intraventricular conduction delays, such as left bundle branch block, are observed fairly often.
- Electrocardiographic evidence of cardiac chamber enlargement is observed in patients that survive long enough for the volume overload associated with IE to result in chamber dilation and hypertrophy.

Echocardiographic Findings

- The term *vegetative endocarditis* is generally used to refer to cases in which there is a macroscopic infected thrombus associated with a valve leaflet.
- The echocardiographic findings in vegetative IE are distinctive. In most cases, nodular distortion of the valve leaflets is readily apparent (Fig. 6.17). Often the valvular abnormality is discrete, and the lesion may oscillate independently of valve motion.
- When these abnormalities are associated with the aortic valve leaflets, the diagnosis is usually assured.
- It can be difficult to distinguish severe MVD from infection of the mitral valve leaflets (Fig. 6.18). However, nodular freely oscillating structures attached to valve leaflets or chords are likely to have high diagnostic specificity for the identification of

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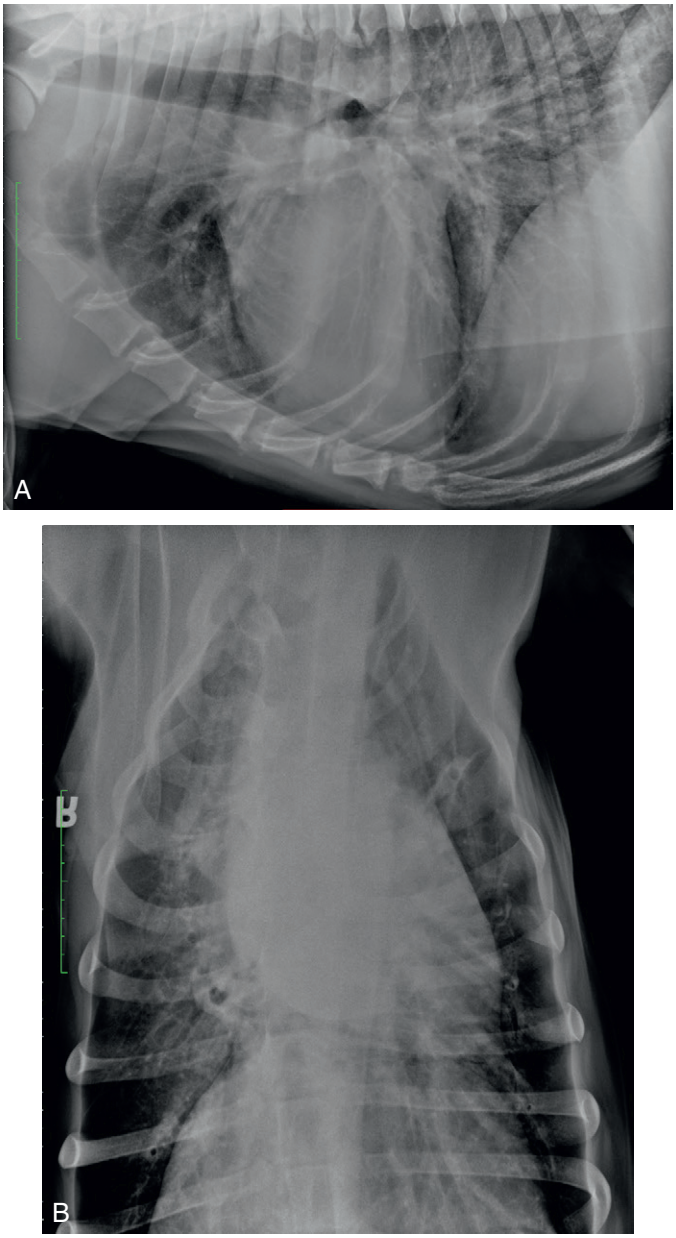


Fig. 6.16 Lateral (**A**) and ventrodorsal (**B**) thoracic radiographs obtained from a 7-year-old male castrated Great Dane with mitral valve endocarditis. The cardiac silhouette is mildly enlarged, and there is evidence of left atrial enlargement. Pulmonary opacities indicate the presence of pulmonary edema.

IE. As stated previously, IE is very uncommon in elderly small dog breeds, which are of course the patients most likely to have severe MVD.

- The interpretation of subtle valvular abnormalities detected by echocardiography poses a difficult clinical problem, and other, ancillary clinical data, including the results of blood cultures, must be considered.
- The absence of a readily detectable valvular nodule on echocardiographic examination does not eliminate IE from the differential diagnosis.
- When vegetative IE of the aortic or mitral valve is present, there is often echocardiographic evidence of left atrial and left ventricular enlargement. Premature diastolic closure of the mitral valve and diastolic flutter of the anterior mitral valve leaflet indicate severe AI (Fig. 6.19). Some degree of systolic myocardial dysfunction is typically present when cardiac enlargement results from AI. Doppler echocardiography is used to confirm the presence of valvular incompetence.

Laboratory Data

- In many cases of IE, the hemogram reveals leukocytosis, but this finding is not consistently present. In subacute cases of IE, the leukocytosis is relatively mild, and evidence of acute inflammation may or may not be present.
- Abnormalities in the serum chemistries are not specific and, when present, are secondary to the disease

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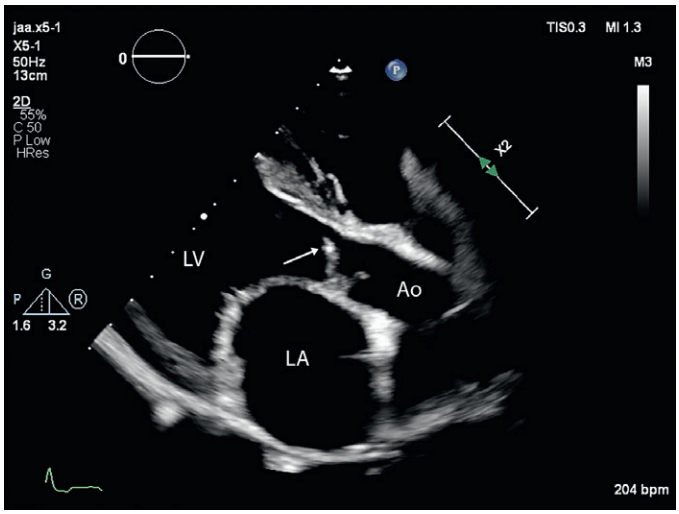


Fig. 6.17 Two-dimensional echocardiographic image obtained from a 6-year-old mixed-breed dog with aortic valve endocarditis. This right parasternal long-axis view demonstrates the presence of a large, highly echogenic nodule attached to one of the aortic valve leaflets. *Ao*, Aorta; *LA*, left atrium; *LV*, left ventricle.

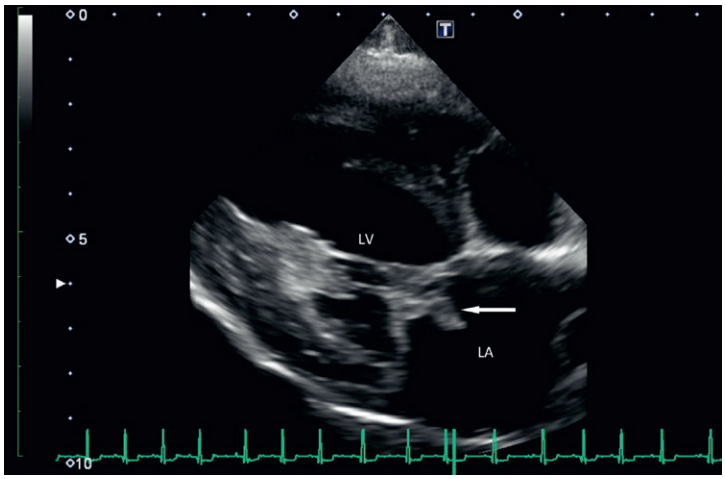


Fig. 6.18 Two-dimensional echocardiographic short-axis image obtained from a 5-year-old female-spayed Doberman Pinscher with mitral valve endocarditis. There is a discrete nodule attached to the anterior mitral valve leaflet (*arrow*). *LA*, left atrium; *LV*, left ventricle.

process. Azotemia is relatively common and can be prerenal, as a result of poor renal perfusion, or it can result from renal infarction. Hypoalbuminemia, hyperglobulinemia, and elevations in serum alkaline phosphatase may also be observed.

- Bacteria are cultivated from whole-blood samples in approximately 40% to 60% of IE cases. When possible, three whole-blood samples are obtained over a 12- to 24-hour period from a central vein after aseptic preparation of the overlying skin. When clinical circumstances allow, blood samples are obtained before the institution of antibacterial therapy, and the results are used to guide treatment. Taking samples over a shorter time period, say 1 to 3 hours, is reasonable when the clinical findings suggest that initiation of antibiotic therapy should not be delayed. Recent evidence suggests that a polymerase chain reaction test for bacterial nucleic acid may be a useful diagnostic adjunct to conventional blood cultures.

THERAPY

- Therapy of IE is directed toward control of sepsis, prevention of thromboembolism, and management of cardiac dysfunction that results from valvular incompetence.
- Antibacterial therapy is best guided by the results of bacterial culture and sensitivity testing of whole-blood samples. When culture results are unavailable or when attempts to isolate bacteria from blood are unsuccessful, antibiotics are chosen on an empiric basis.
- In most published case series, when a bacterium is isolated, gram-positive organisms predominate,

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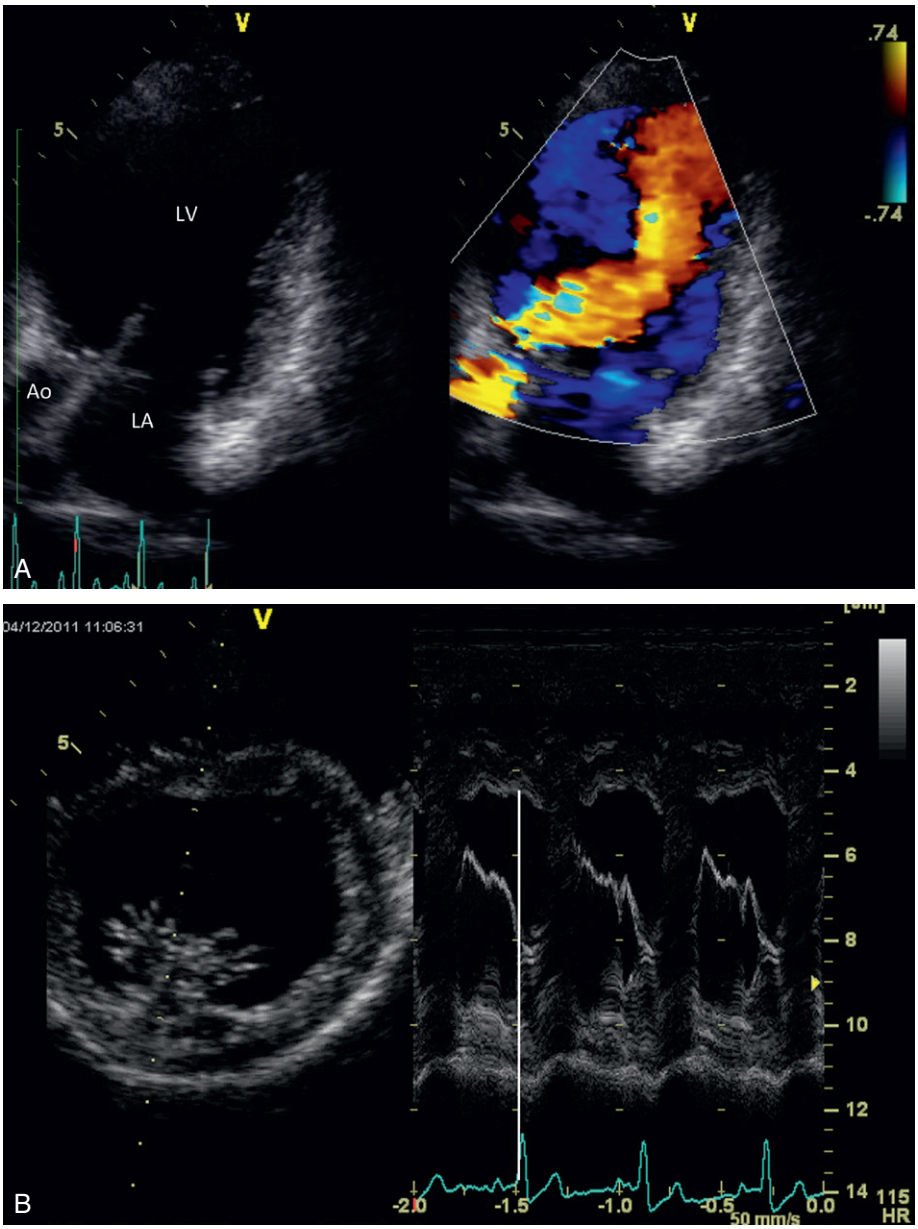


Fig. 6.19 Echocardiographic images that provide evidence of severe aortic valve incompetence caused by bacterial endocarditis. **(A)** A diastolic right parasternal image with (*right*) and without (*left*) superimposed color Doppler map; severe aortic valve regurgitation streams through the aortic valve (*arrow*). **(B)** A short-axis image of the mitral valve and accompanying M-mode image from a Boxer dog with severe aortic valve regurgitation caused by endocarditis. There is diastolic flutter of the anterior mitral valve leaflet and premature closure of the mitral valve (*line*)—indirect echocardiographic evidence of severe aortic valve incompetence. *Ao*, Aorta; *LA*, left atrium; *LV*, left ventricle.

although a greater number of gram-negative organisms was recently identified during a retrospective investigation conducted in the United Kingdom. Agents such as the clavulanate-potentiated penicillins or the cephalosporins are generally appropriate for empirical therapy. **Azithromycin** may have a role in the management of IE caused by *Bartonella* species. In general, bactericidal agents are preferred. A long course of therapy, 6 to 8 weeks, is generally recommended. There may be some advantages to initiating antibiotic therapy with the intravenous route because high serum drug levels are achieved quickly and with certainty.

- When clinical signs of sepsis are prominent and blood culture results are negative, initial therapy consisting of intravenous administered broad-spectrum antibiotics is justified. When patients are free of gastrointestinal signs such as vomiting or diarrhea, oral antibiotic therapy is probably appropriate.
- Cardioactive agents are chosen on the basis of the results of radiographic and echocardiographic examinations. It is probably reasonable to apply the current guidelines for management of myxomatous valvular disease to patients with cardiac dysfunction related to IE, although an evidentiary basis for this approach is lacking.

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Monitoring of Therapy

- As with other patients with HF, it is prudent to monitor serum electrolytes and renal function. Further monitoring of patients with IE may include follow-up echocardiograms.
- It is tempting to think that appropriate antibiotic therapy will result in resolution of echocardiographically detected valvular lesions. The valvular lesions of dogs in which the presentation is subacute and there is echocardiographic evidence of severe valvular incompetence, do not generally resolve. Serial echocardiograms in these cases usually demonstrate progressive valvular incompetence and myocardial dysfunction.

Prophylaxis of Infective Endocarditis

It is accepted that bacteremia is a requisite for the development of IE. However, although bacteremia results from dental procedures, transient bacteremia occurs in humans associated with normal day-to-day activities. Furthermore, the efficacy of antibiotic prophylaxis is uncertain. Accordingly, guidelines for antibiotic prophylaxis in humans have evolved, and antibiotics are no longer recommended before dental procedures for human patients with MVD.

Relevant data that relate to canine patients are not available. It is reasonable to provide antibiotic prophylaxis for patients with congenital cardiac disease when they will undergo procedures in which bacteremia can be anticipated. Antibiotic administration intended to treat oral infection is also reasonable, but the value of antibiotic prophylaxis for patients with MVD subject to dental procedures is unknown. It is reasonable to perform dental procedures to improve dental health, but prevention of IE is not a justification with evidentiary basis.

PROGNOSIS

The long-term prognosis for patients with echocardiographic evidence of IE is generally poor. Reported survival times vary widely perhaps because of regional differences in causative organisms and because almost all of the findings were retrospectively described. In most published case series, survival after diagnosis is brief; a median survival time of 72 days was calculated from the case records of 104 canine patients. By the time the disease is detected, many cases of IE have progressed to the extent that there is irreversible damage to the valve. Failing surgical valve replacement, which is rarely practical in veterinary patients, the clinical picture is often of progressive cardiac dysfunction, although patients with mitral valve involvement generally fare better than those with aortic valve disease. Causes of death include renal failure related to renal infarction or sequelae of embolic events and medically refractory HF.

Frequently Asked Questions

There is current interest in the use of spironolactone as adjunctive therapy for HF in dogs. What are the indications for spironolactone in MVD?

Spironolactone is an antagonist of the mineralocorticoid aldosterone. It is not a potent diuretic, and interest in its use primarily relates to its presumed extrarenal effects. Cardiac dysfunction is associated with neuroendocrine responses that result in supraphysiologic aldosterone activity. Aldosterone is a ligand for specific renal receptors that mediate retention of sodium and excretion of potassium. In addition, there are data that support a role for aldosterone in the development of myocardial fibrosis in animals with experimentally induced cardiovascular disease. Inhibition of aldosterone activity by spironolactone reduced mortality in humans with severe HF, and this effect was evident at a dose that did not have a diuretic effect. Although invasive studies may disclose systolic myocardial dysfunction in dogs with mild but experimentally induced MR, contractility is apparently preserved until late in the natural history of MVD.

A double-blind, placebo-controlled clinical trial intended to test the hypothesis that administration of spironolactone would slow progression of stage B2 to stage C MVD did not demonstrate superiority of spironolactone. Current guidelines include the recommendation to administer spironolactone to patients in stage C.

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Chapter

7

Canine Cardiomyopathy

Joanna Dukes-McEwan

Cardiomyopathy is a primary heart muscle disease. In dogs, dilated cardiomyopathy (DCM) is by far the most common cardiomyopathy, but rarely, hypertrophic cardiomyopathy (HCM) is diagnosed and certain breeds may present with arrhythmogenic right ventricular cardiomyopathy (ARVC) (Boxers, English bulldogs). Since certain conditions may result in a similar echocardiographic appearance to DCM (and HCM), initially it is better to describe the condition as a DCM (or HCM) phenotype prior to actively excluding primary conditions, which can result in a secondary cardiomyopathy. ARVC may present with no significant echocardiographic abnormalities but can also be manifest as a DCM phenotype (see Arrhythmogenic Right Ventricular Cardiomyopathy section). Potential causes of a secondary DCM phenotype include tachyarrhythmia, leading to a tachycardia-induced cardiomyopathy (TICM), nutritional deficiencies, toxins, endocrine disease, or a consequence of myocarditis. Conditions that may result in a secondary HCM phenotype include systemic hypertension (high afterload resulting in left ventricular hypertrophy), volume depletion (pseudohypertrophy), and myocardial infiltrative conditions. Ventricular arrhythmias may occur with primary cardiac disease such as ARVC and other cardiomyopathies (e.g., DCM in Dobermanns), which do not have to be of right ventricular origin. Better terminology may be arrhythmogenic cardiomyopathy (AC), with ventricular arrhythmias often preceding any echocardiographic changes. These may lead to syncopal episodes or sudden death. Arrhythmias can occur with systemic conditions, which must be actively excluded before presuming the presence of a primary AC.

DILATED CARDIOMYOPATHY

Primary idiopathic dilated cardiomyopathy is a myocardial disease characterized by impaired left ventricular (LV) systolic function, leading to progressive dilation of the LV without appropriate eccentric hypertrophy, so LV walls are proportionately thin. The remodeling leads to a change in LV geometry, so it appears rounded rather than elliptical. The left atrium is affected at a more advanced stage, and as left-sided filling pressures increase, left-sided congestive heart failure (CHF; cardiogenic pulmonary edema) may be the reason for the presentation. The right heart may or may not be affected, depending on breed and the stage of presentation, which may result in right-sided CHF signs (ascites, possibly pleural effusion).

There are two phases that may be recognized for canine DCM (Box 7.1):

1. Clinical DCM with the CHF signs described above.
2. Preclinical (occult) DCM in asymptomatic dogs, which may be identified when proactively screening for the condition by echocardiography (or for arrhythmias by ambulatory ECG monitoring). Such screening is prompted by high prevalence in certain breeds or if familial disease is reported. The preclinical phase can be very prolonged (over years).

PREVALENCE AND SIGNALMENT

Surveys indicate that between two and six dogs are diagnosed with DCM per 600 case referrals. In certain breeds, the prevalence of DCM is remarkably high. Approximately 58% of Dobermanns, 36% Great Danes, 24% of Irish Wolfhounds, 22% of Scottish Deerhounds, and 17% Newfoundlands may develop DCM shown by prospective, serial screening. Some of these cases may never develop clinical DCM, as the preclinical phase can be prolonged; the prevalence of clinical DCM is usually much lower. Screening is problematic in young breeding dogs, since normal results do not preclude the possibility of developing DCM in the future; serial screening is indicated (e.g., every 1–2 years).

DCM is an acquired disease which typically occurs in middle-age, but young to old dogs may manifest clinical disease. Other predisposed breeds include other giant breeds such as St. Bernards, Leonbergers, and Dogues de Bordeaux, large-breed dogs including Golden retrievers, Weimaraners and spaniel breeds including American and English cocker spaniels, English Springer spaniels. In some breeds (especially cocker spaniels, Golden retrievers and Newfoundlands), taurine deficiency should be excluded as a nutritional DCM is relatively common in these breeds. DCM is rare in crossbred dogs or small breeds; secondary causes of the phenotype need to be actively excluded.

There does not seem to be a strong sex predisposition in prospective screening studies. However, male

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Box 7.1 Preclinical and Clinical Dilated Cardiomyopathy: Phases of Progression

ASYMPTOMATIC PRECLINICAL (OCCULT) PHASE

- No clinical signs are evident; however, myocardial or electrical abnormalities are present and may include the following:
  - Increased left ventricular (LV) and atrial dimensions and volumes
  - Rounded LV with relatively thin LV walls
  - Decreased myocardial systolic function (hypokinesis)
  - Ventricular premature complexes (ECG or 24-hour ambulatory ECG [Holter] monitoring)
- The duration of the occult phase is highly variable and may possibly last for months to several years.
- During this phase, progressive heart enlargement and worsening arrhythmias occur.
- The occult phase ends with the appearance of the first clinical signs of disease.
  - Approximately 40% of Doberman Pinschers experience sudden cardiac death as the first clinical sign.

OVERT CLINICAL PHASE

- Clinical signs develop:
  - Congestive heart failure (CHF); left-sided, right-sided, or biventricular CHF
  - Syncope
  - Exercise and activity intolerance
- Arrhythmias may develop in the form of ventricular premature complexes or more complex ventricular ectopy, including ventricular tachycardia (which may lead to syncopal episodes), and atrial fibrillation.
- Death is due either to advanced CHF that is refractory to medical therapy or sudden death.
  - A total of 30% to 50% of Doberman Pinschers die suddenly.
  - Many dogs with advanced heart failure are euthanized because of chronic respiratory distress, severe activity intolerance, anorexia, and weight loss (usually with cardiac cachexia; severe muscle wasting).

dogs may be more likely to present with clinical DCM with CHF. In some studies, Dobermann females were more likely than males to have ventricular arrhythmias.

ETIOLOGY AND NATURAL HISTORY

Primary DCM is usually idiopathic; the cause is unknown. However, there is an increasing body of evidence supporting a genetic basis to DCM, including identification of familial disease within breeds, pedigree analysis investigating mode of inheritance and most recently, loci or genes implicated in DCM (Dobermanns, Irish Wolfhounds, standard Schnauzer, Welsh Springer spaniel). Although initial pedigree analyses in many breeds suggested an autosomal dominant mode of inheritance with variable penetrance, it has become increasingly clear that the genetic basis of DCM is more complex and may be multigenic. Even in a single breed such as the Dobermann, three different genes/loci have been implicated (with two commercially available genetic tests: PDK4 and Titin).

As in human DCM, the primary issue in many cases of DCM is likely to be a defect in the cardiomyocyte cytoskeleton. The wear and tear of constantly beating may lead to progressive loss in function and this eventually results in the echocardiographic phenotype we identify. Defects in energy metabolism with cardiomyocytes may result in a single phenotype. There may be various pathways that lead to the final phenotype we call DCM.

As mentioned previously, there is a long preclinical phase prior to development of clinical DCM (see Box 7.1). Ventricular dilatation with relatively thin walls and altered geometry may lead to high wall stress (La Place relationship), which may result in arrhythmias. Atrial dilatation and stretch, especially in large or giant breeds, may result in atrial fibrillation. In some breeds (especially the Irish Wolfhound), atrial fibrillation may precede conclusive echocardiographic abnormalities. In such cases, it is important to exclude a TICM, but "lone" atrial fibrillation is usually not at an abnormal heart rate.

On histopathology, there may be few changes. In some cases, the classical DCM phenotype, attenuated wavy fibers may be seen, separated by myocardial edema. In other cases, with loss of cardiomyocytes, there may be an increase in adipocytes and fibrosis (fibrofatty infiltration). This is likely to be a substrate for the ventricular arrhythmias seen in some DCM or arrhythmogenic cardiomyopathy cases.

HISTORY AND PHYSICAL EXAMINATION FINDINGS

Asymptomatic Preclinical (Occult) Phase

The history is usually unremarkable, since the dog is asymptomatic. It is worth asking if any familial DCM is known, and to check diet history, travel history, prior illnesses or medication, parasitic control, etc. These factors are important in excluding secondary causes of a DCM phenotype.

- Common findings on physical examination include the following:
  - There may be no abnormalities detected (echocardiography required).
  - Soft systolic heart murmur. This is usually due to mitral regurgitation, due to stretch of the mitral annulus with the altered LV geometry.
  - Irregular heart rhythm with pulse deficits. Atrial fibrillation can occur in some breeds (especially Irish Wolfhounds) without clinical signs, usually not with tachycardia. Premature beats may also be detected in the dog with frequent ventricular premature complexes.
- Occasional findings include the following:
  - Diastolic gallops (usually audible S3 sounds with a stiff, poorly compliant LV, especially if LA pressure is increased).

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- Decreased intensity of heart sounds
- Weak femoral pulse quality
- Jugular vein distension or pulses (rare in preclinical DCM).

Overt Clinical Phase

History may include lethargy, exercise intolerance, syncope, anorexia, difficulty breathing, coughing, and abdominal distension.

- Common findings on physical examination include the following:
  - Soft or moderate-intensity systolic heart murmur (stretch of atrioventricular annuli and secondary mitral and/or tricuspid regurgitation).
  - Irregular heart rhythm with pulse deficits (atrial fibrillation or ventricular arrhythmia).
  - Increased respiratory rate and effort; restrictive breathing pattern.
  - Increased bronchovesicular sounds; occasionally audible crackles if cardiogenic pulmonary edema.
  - Decreased intensity of heart sounds (reduced systolic function, but may also reflect presence of pleural effusion).
  - Weakness.
- Occasional findings include the following:
  - Jugular vein distension or pulsation
  - Hepatomegaly
  - Ascites
  - Pale mucous membranes with sluggish capillary refill
  - Hypothermia
  - Pulmonary crackles
  - Depressed mentation

MAKING THE DIAGNOSIS

Diagnostic investigations for all phases of DCM will include echocardiography, as this is considered to be the gold standard to confirm the diagnosis of DCM, after active exclusion of other conditions that may result in a DCM phenotype. An electrocardiogram (ECG) is indicated in any case with an arrhythmia, often with an ambulatory 24-hour (or longer; optimally 4 days) ECG (Holter monitoring) to screen for arrhythmias, especially in breeds such as the Dobermann. In dogs with coughing or respiratory signs, thoracic radiographs are indicated to confirm the presence of cardiogenic pulmonary edema and to exclude respiratory disease contributing to these signs. A thoracic point-of-care ultrasound will quickly identify pleural effusions and B-lines in "wet lung," which may be cardiogenic pulmonary edema especially if the left atrium is enlarged. Blood pressure monitoring is important, to identify those hypotensive dogs who may present with cardiogenic shock; inotropic support should be promptly provided to these cases (see Treatment section). Blood tests include hematology and biochemistry, to check for significant comorbidities and to monitor renal function and electrolytes in dogs requiring diuretic treatment. Urinalysis should also be considered. Hypothyroidism should be identified if present, as many DCM breeds are also predisposed to this endocrine condition. The cardiac biomarkers may be used for diagnostic and prognostic information and have been described as a way of prescreening at-risk breeds prior to echocardiographic screening. Genetic testing is indicated in breeds where tests are commercially available, especially breeding animals.

Echocardiography

Echocardiography is widely used to quantify heart chamber enlargement and systolic function. Routine echocardiography is not particularly sensitive in detecting early changes in occult disease and it is common to find "equivocal findings," especially of systolic function when screening. However, in the genuine preclinical DCM case, progression is expected over years so serial screening can eventually confirm the diagnosis. The utility of echocardiography increases as the disease moves from the occult to the overt clinical phase. In arrhythmogenic cardiomyopathy, especially in Dobermanns, the echocardiogram can be normal, despite significant ventricular arrhythmias. Echocardiographic values from any individual dog are compared with breed reference ranges where available or are scaled to body size with allometric techniques. Allometric techniques (Chapter 4; Appendix 3) are being increasingly used as they account for the nonlinear relationship between echocardiographic dimensions and volumes, such as the left ventricular internal diameter in diastole (LVIDd) or LV end-systolic volume and body weight.

- Echocardiographic criteria are used to help diagnose occult DCM. The greater number of indices that are abnormal, the higher the likelihood that occult DCM is present.
- The typical echocardiographic changes that can be seen in a dog with DCM are listed in Box 7.2 and shown in Figs. 7.1, 7.2, and 7.3.
- M-mode LV diameters are most commonly used in DCM screening. There are some reference ranges for different breeds, and criteria for abnormal results, given in Table 7.1. Note that the single dimensional assessment of systolic function with fractional shortening is not a reliable indicator of systolic function, especially in deep-chested breeds such as the Dobermann, as it does not include longitudinal or global left ventricular function, or in athletic breeds (e.g., Springer spaniel). Fractional shortening is also not informative if there is asynchronous LV motion, common in DCM.
- To account for changes in global LV function, rather than using the single dimension of M-mode, it is better to determine LV systolic volumes in diastole and systole, using 2D methods that are free from geometric assumptions of ventricular shape, such as

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Simpson's method of discs (Fig. 7.4). These volumes may be normalized for body surface area (m^2; meters squared) which is acceptable in a single breed such as the Dobermann (see Table 7.1). With the diversity of breeds and when reference ranges are not available, it is better to normalize the volumes by body weight (in kilogram). Increased LV systolic volume index is >1.35 mLs/kg, and increased diastolic volume index is >3.0 mLs/kg. From the actual volumes, LV ejection fraction can be calculated.

Box 7.2 Typical Echocardiographic Features Identified in Canine Dilated Cardiomyopathy

- Moderate to severe LV and atrial enlargement (Fig. 7.1).
- Globally reduced LV systolic function (Figs. 7.1 and 7.2).
- Reduced systolic motion of the LV wall and interventricular septum (see Fig. 7.2).
- Decreased systolic thickening of the LV wall and interventricular septum.
- LV wall thickness proportionately thin compared with chamber diameter (but absolute measurements within reference ranges) (see Figs. 7.1 and 7.2)
- Rounded LV chamber (can be assessed by sphericity index—ratio of LV length to width; however, this index does not appear to have value as a screening tool) (see Fig. 7.1)
- Mild to moderate mitral regurgitation secondary to mitral annulus dilation (Fig. 7.3). Continuous wave Doppler may show lower-than-expected MR peak velocity.
- Incomplete systolic opening of the aortic valves or premature closure of the aortic valves (seen on aortic valve M-mode).
- Decreased aortic blood flow velocity (spectral Doppler).
- Increased M-mode mitral valve E-point to septal separation (normal < 7 mm) (see Fig. 7.2)
- The right heart may be subjectively normal, or dilated.
- Note that in arrhythmogenic cardiomyopathies (especially Dobermanns), ventricular arrhythmias may precede any echocardiographic abnormalities.

- Impaired LV systolic function is better reflected by documenting increased LV systolic diameter or volume. Fractional shortening or ejection fraction may be "higher" than intrinsic systolic function if there is significant mitral regurgitation (low afterload) or "lower" than intrinsic with wall motion abnormalities or in athletic breeds.
- Serial echocardiographic studies can document progression of DCM or response to treatment (see later).
- As the disease progresses, the left atrial (LA) size also increases. This can be documented by looking at the systolic right parasternal long-axis LA maximum diameter/aortic annulus ratio (>2.5), or the diastolic short-axis LA/aortic ratio (>1.6).
- The right heart may also become dilated, with reduced RV function (documented as the tricuspid annular plane systolic excursion [TAPSE]) on M-mode.

It can be seen that the echocardiographic diagnosis of DCM is based on documentation of increased LV dimensions or volumes and documenting impaired LV systolic function. Color flow and spectral Doppler echocardiography are indicated to exclude other structural cardiac conditions that may result in chamber dilatation. However, it is common to see secondary mitral and tricuspid regurgitation if there is stretch of the respective atrioventricular annulus (see Fig. 7.3). Mitral regurgitation may be at lower-than-expected velocity (e.g., <5 m/s, with MR velocity of <4 m/s associated with poor prognosis in view of low LV systolic pressure and high LA pressure, reducing the pressure gradient between the LV and LA). Furthermore, lower-than-expected aortic velocity may reflect the impaired LV systolic function. It is possible that the newer ultrasound modalities, such as speckle tracking in 2D strain imaging or Doppler tissue imaging, reflect the intrinsic impairment of LV systolic function prior to changes in chamber sizes, but more data are required before this imaging can be recommended.

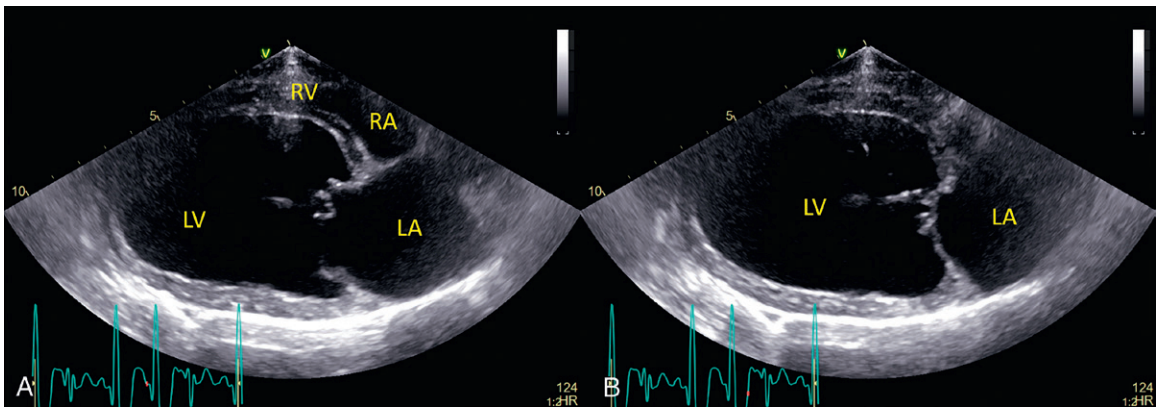


Fig. 7.1 Right parasternal long-axis four-chamber views from a Vizsla with dilated cardiomyopathy (DCM). The left ventricle is dilated, rounded, and relatively thin walled. Frames during diastole (A) and systole (B) demonstrate the hypokinetic left ventricle, with little difference in LV area (and volume) between the frames. An arrhythmia is noted on the simultaneously acquired ECG (sinus with ventricular and supraventricular premature complexes). *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

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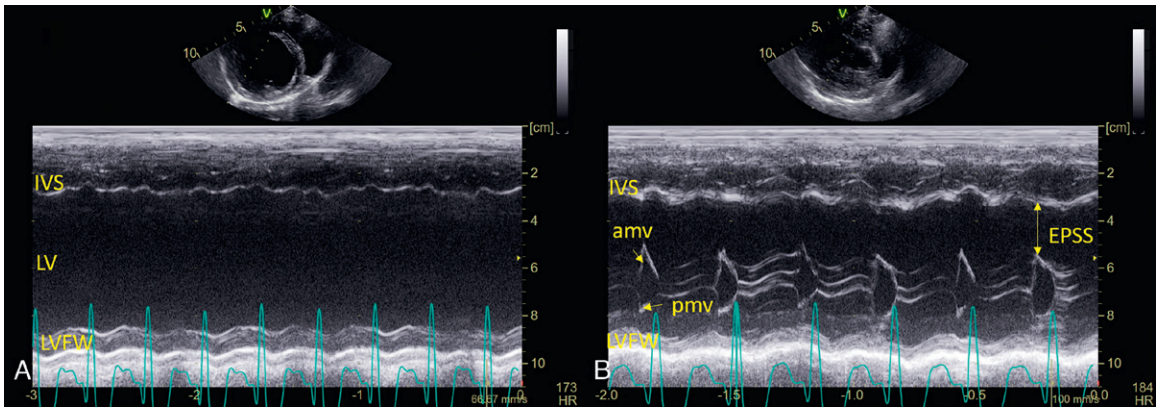


Fig. 7.2 M-modes obtained from obtained right parasternal short-axis views: (A) At chordae tendinae level to give the left ventricular M-mode, with the M-mode cursor bisecting the LV cavity to show the movement of the interventricular septum and left ventricular free wall during diastole and systole; evidence of hypokinesis. (B) At mitral valve level to show the increased distance between the mitral valve anterior leaflet during early diastole (E peak) and the interventricular septum (increased E-point to septal separation; EPSS; measurement shown by double-headed arrow). This increased EPSS is a consequence of both the rounded geometry and the reduced stroke volume, so mitral valve leaflets do not open completely. This dog is in sinus rhythm during these M-modes but at a high heart rate, so separate E and A peaks are not seen. *amv*, Anterior mitral valve leaflet; *EPSS*, mitral E-point to septal separation; *IVS*, interventricular septum; *LV*, left ventricular chamber; *LVFW*, left ventricular free wall; *pmv*, posterior mitral valve leaflet.

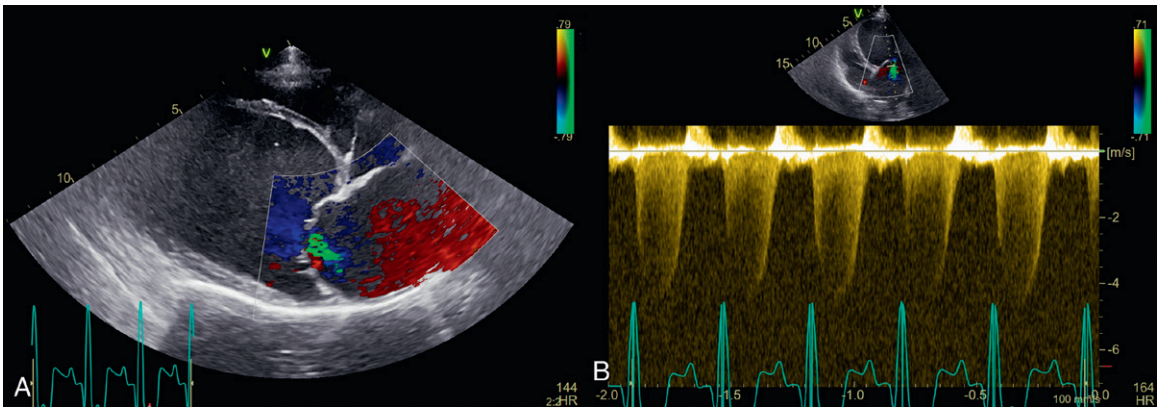


Fig. 7.3 Mitral regurgitation is common in dilated cardiomyopathy, secondary to stretch of the mitral annulus. (A) Right parasternal long-axis four-chamber view, systolic frame, showing color flow Doppler evidence of a small mitral regurgitant jet. (B) Left apical four-chamber view, aligned with transmitral flow and mitral regurgitation, showing continuous wave spectral Doppler of the mitral regurgitation. This velocity is lower than expected (ref. 5–6 m/s), due to reduced LV systolic pressure generation and/or increased left atrial pressure.

Electrocardiography and Holter Monitoring

If an arrhythmia is identified from physical examination, an electrocardiogram (ECG) is considered mandatory to make the diagnosis of the arrhythmia, which is a prerequisite to safely treating hemodynamically significant arrhythmias. If a dog presents with a clinical sign that may be due to a transient arrhythmia (such as syncope), the ECG may be informative but does not confirm the cause of the syncope. Also, an ECG, even if over 3 to 5 minutes, is not as informative as a 24-hour ambulatory ECG recording (Holter), which can document changes in heart rate and rhythm during the dog's daily activities (including any syncopal event). In Dobermanns, documenting any ventricular premature complexes (VPCs) during a 5-minute ECG is supportive of significant ventricular arrhythmia (Fig. 7.5) and suggestive of an arrhythmogenic cardiomyopathy (diagnosed based on Holter monitoring criteria [Table 7.2]). The standard six-lead ECG may provide evidence of specific chamber enlargement, but it is not sufficiently sensitive to be used as a screening tool. A normal ECG does not exclude DCM, which may be apparent on echocardiography.

- Arrhythmias are often the first indication of disease in some breeds, especially VPCs. Holter monitoring as well as echocardiographic screening is recommended in breeds at high risk for DCM.
- Detection of the following ECG signs is associated with a high index of suspicion for occult cardiomyopathy:

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Table 7.1 Echocardiographic Criteria of Normality and Abnormality During Examination for Dilated Cardiomyopathy in Certain Predisposed Dog Breeds

	DOBERMANNS	IRISH WOLFHOUNDS	GREAT DANES	NEWFOUNDLANDS	SCOTTISH DEERHOUNDS
LV M-mode values					
LVIDd (mm)	<47 mm *Abnormal >48 mm (male) or >46 mm (female)*	42.7–65.5 *Abnormal >61 mm*	46.7–57.7 (male) 42.7–56.1 (female) *Abnormal >59 mm*	37.29–53.41 *Abnormal >55 mm (male)* *>50 mm (female)*	47.08–65.78 (male) 44.26–58.47 (female) *Abnormal >61.8 (male) or >58 (female)*
LVIDs (mm)	<38 mm *Abnormal >36 mm (males or females)*	25.4–41.5 *Abnormal >41 mm*	33.7–42.5 (male) 28.8–41.9 (female) *Abnormal >45 mm*	28.31– 40.31 *Abnormal >42 mm (males and females)*	32.8–48.26 (male) 31.28–44.89 (female) *Abnormal >45.6 (male) or >45.1 (female)*
FS (%)	>20%	25–48 *Abnormal <25%*	20–37 *Abnormal <20*	18.05– 30.81 *Abnormal <18%*	18.79–34.87 *Abnormal <21.1*
LV volumes: 2D Simpson's (method of discs) values					
EF (%)	Not given	Not given	42.1–63.9 *Abnormal <40*	25.29–58.29	40.39–67.28 *Abnormal <42%*
EDVI (mLs/m²)	56–92 *Abnormal >95*	Not given	Not given	35.91–88.87	53.19–141.64 *Abnormal >140.2*
ESVI (mLs/m²)	24–53 *Abnormal >55*	Simpson's not reported. Teichholz from M-mode: 15.3–40.6	21.9–47 *Abnormal >47*	19.7–52.22 *Abnormal >50*	30.5–71 *Abnormal >71.9*
Main references	Wess G, Domenech O, Dukes-McEwan J, et al: European Society of Veterinary Cardiology screening guidelines for dilated cardiomyopathy in Doberman Pinschers, *J Vet Cardiol* 19: 405–415, 2017. Wess G, Mäurer J, Simak J, Hartmann K: Use of Simpson's method of disc to detect early echocardiographic changes in Doberman Pinschers with dilated cardiomyopathy, *J Vet Intern Med* 24: 1069–1076, 2010.	Vollmar AC: Use of echocardiography in the diagnosis of dilated cardiomyopathy in Irish wolfhounds, *J Am Anim Hosp Assoc* 35: 279–283, 1999.	Stephenson HM, Fonfara S, Lopez-Alvarez J, et al: Screening for dilated cardiomyopathy in Great Danes in the United Kingdom, *J Vet Intern Med* 26: 1140–1147, 2012.	Dukes-McEwan J: Echocardiographic/Doppler criteria of normality, the findings in cardiac disease and the genetics of familial dilated cardiomyopathy in Newfoundland dogs. PhD thesis. University of Edinburgh, 1999.	Dutton E, Cripps P, Helps SAF, et al.: Echocardiographic reference intervals in healthy UK deerhounds and prevalence of preclinical dilated cardiomyopathy: a prospective, longitudinal study, J Vet Cardiol 40: 142-155, 2022.
See also:	Chapter 4 and Appendix 3 in this text. Esser LC, Borkovec M, Bauer A, et al: Left ventricular M-mode prediction intervals in 7651 dogs: Population-wide and selected breed-specific values, *J Vet Intern Med* 34: 2242–2252, 2020. Visser LC, Ciccozzi MM, Sintov DJ, Sharpe AN: Echocardiographic quantitation of left heart size and function in 122 healthy dogs: A prospective study proposing reference intervals and assessing repeatability, *J Vet Intern Med* 33: 1909–1920, 2019. Wess G, Bauer A, Kopp A, et al: Echocardiographic reference intervals for volumetric measurements of the left ventricle using the Simpson's method of discs in 1331 dogs, *J Vet Intern Med* 35: 724–738, 2021.				

EDVI, LV end-diastolic volume index; *EF*, ejection fraction; *ESVI*, LV end-systolic volume index; *FS*, fractional shortening; *LVIDd*, left ventricular internal diameter in diastole; *LVIDs*, left ventricular internal diameter in systole.

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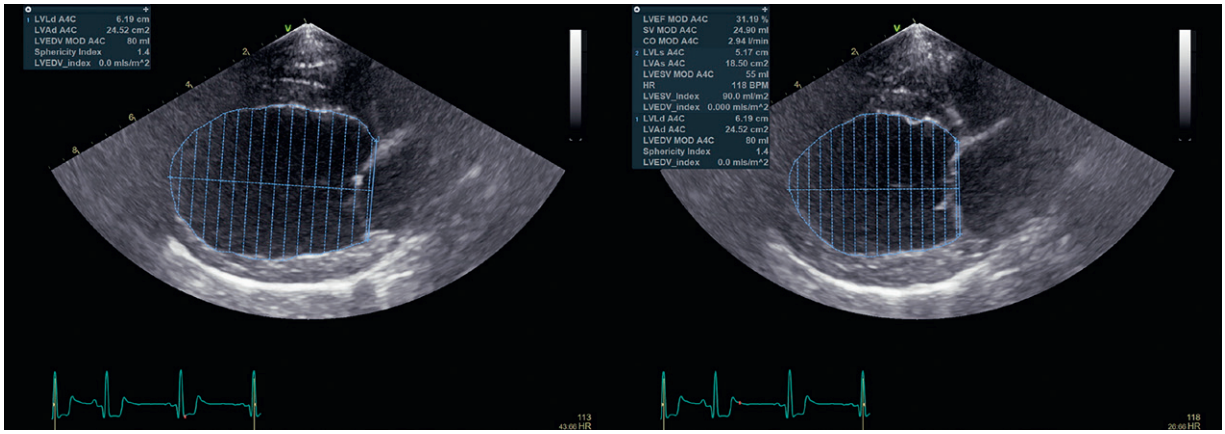


Fig. 7.4 Simpson's method of discs to determine ventricular volumes. Right parasternal long-axis four-chamber views in end-diastole (*left*) (frame before mitral valve closes) and end-systole (*right*) (frame before mitral valve opens), after optimizing the LV length and area. Simpson's method of discs is used during end-diastole (*left*) to determine the LV diastolic volume (*left*) and during systole to determine the LV systolic volume (*right*). Note that these volumes are best normalized by indexing to body weight (in mLs/kg) rather than body surface area. NOTE: Simpson's method of discs can also be applied to the left apical four-chamber view, but whichever view optimizes left ventricular length and area should be used; the anatomical left ventricular apex is often not visible in the left apical views unless it is a deep, narrow-chested breed (like the Dobermann).

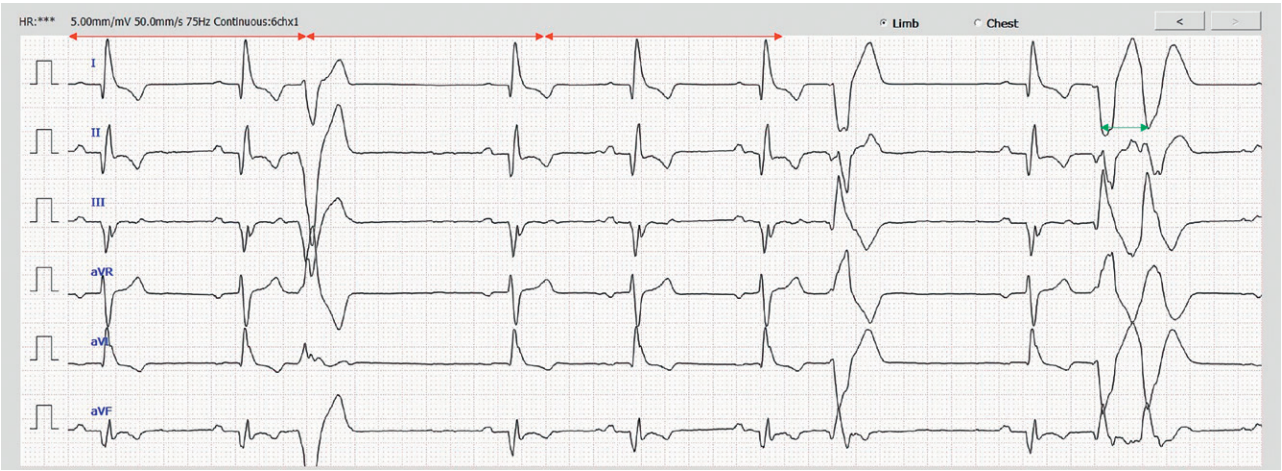


Fig. 7.5 Six-lead ECG in a Dobermann with preclinical dilated cardiomyopathy. Three 1-second marks are shown (*red arrows*) (50 mm/s paper speed). From the left, the 3rd, and 7th complexes are single ventricular premature complexes (VPCs) and the last two complexes are a couplet of VPCs. The instantaneous heart rate (*green arrow*) for the couplet can be calculated as 3000 divided by the number of paper grid small boxes between the ectopics. So here, 3000/10 = 300 bpm. The ventricular ectopics are monomorphic in this trace. Underlying rhythm is sinus, but there is a left-axis deviation (lead I R wave > lead II, negative QRS in lead III), common in the breed.

- One or more VPCs in a Dobermann (see Fig. 7.5) (or Boxer with ARVC).
- Criteria for LV (QRS duration >0.06 seconds, R-wave amplitude >3.0 mV) or atrial enlargement (P-wave duration >0.04 seconds)
- In Irish Wolfhounds and sometimes Great Danes and other giant breeds, atrial fibrillation may be an early sign of disease in contrast to other breeds, where atrial fibrillation is associated with advanced stages of disease. Initially, this can appear to be "lone" atrial fibrillation with minimal echocardiographic abnormalities (see Fig. 7.6). It is often at an acceptable rather than tachycardic heart rate. Holter monitoring is indicated to identify the mean heart rate/24 hours and maximum heart rate, to assess whether rate control medication (or cardioversion) is indicated.
- Supraventricular (atrial) premature complexes are common and may reflect atrial stretch and risk of future development of atrial fibrillation. However, in Dobermanns, they do not seem to be predictive of dogs likely to develop DCM.
- Holter monitoring detects arrhythmias with greater sensitivity and is recommended in dogs at high risk (i.e., dogs with a familial history of disease). The Holter criteria for DCM screening in Dobermanns are shown in Table 7.2. More serious than single ventricular premature complexes are presence of couplets, triplets, or runs of ventricular tachycardia, especially if at a fast instantaneous rate (>250 bpm)

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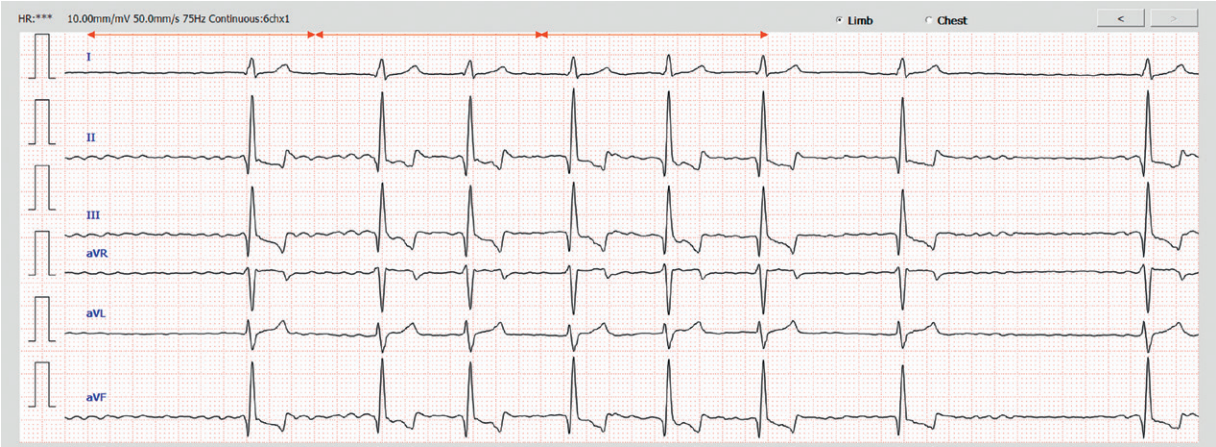


Fig. 7.6 Atrial fibrillation without tachycardia in a Great Dane with preclinical dilated cardiomyopathy. One-second marks are shown with each red arrow (50 mm/s speed). The heart rate is approximately 120 bpm, with instantaneous rates between beats during this section of between 55 and 158 bpm. This Great Dane had impaired left ventricular systolic function but no ventricular dilatation. Mild biatrial dilatation was noted (possible consequence of the atrial fibrillation rather than the cause).

Table 7.2 Holter Criteria to Diagnose Arrhythmogenic Cardiomyopathy (DCM in Dobermanns or ARVC in Boxers) Associated with Ventricular Ectopy (VPCs)

NUMBER OF VENTRICULAR PREMATURE COMPLEXES/24 OURS	DOBERMANN	BOXER
Normal	• <50 VPCs/24 hours • Any ectopy a concern especially if complexity (couplets/triplets or runs) especially if IHR is > 250 bpm.	< 20 VPCs/24 hours
Equivocal	• 50–300 VPCs/24 hours highly suggestive of DCM • More likely to be affected if also evident on repeat Holter within 6–12 months. • More likely to be affected if complexity and fast IHR > 250 bpm.	20–100 VPCs/24 hours Suspicious: 100–300 VPCs/24 hours: suspicious, repeat Holter in 3–6 months
Abnormal	> 300 VPCs/24 hours. Complexity, couplets, triplets, and runs of ventricular tachycardia.	Likely affected: • 100–300 VPCs/24 hours with complexity (couplets, triplets, runs) • 300–1000 VPCs/24 hours Affected: • > 1000 VPCs/24 hours (consider treatment)
Key references	Wess G, Domenech O, Dukes-McEwan J, et al: European Society of Veterinary Cardiology screening guidelines for dilated cardiomyopathy in Doberman Pinschers, *J Vet Cardiol* 19: 405–415, 2017.	Cunningham SM, Dos Santos L: Arrhythmogenic right ventricular cardiomyopathy in dogs, *J Vet Cardiol* 40: 156–169, 2022.

ARVC; Arrhythmogenic right ventricular cardiomyopathy; *bpm*, beats per minute; *DCM*, dilated cardiomyopathy; *IHR*, instantaneous heart rate (determined as 60,000/time in milliseconds between two VPCs); *VPCs*, ventricular premature complexes.

(Fig. 7.7). Note that there can be day-to-day variability in the frequency of arrhythmia, which may produce false-negative results. If the initial Holter examination is inconclusive, multiple Holter examinations may be indicated, especially in dogs with a family history of disease or in those that have experienced syncope.

- During the overt clinical phase, the following ECG abnormalities may be detected:
  - Atrial fibrillation (Fig. 7.8) (usually fast rate and associated with significant echocardiographic abnormalities and CHF)
  - Occasional to frequent ventricular or supraventricular premature complexes (Fig. 7.9)
  - Ventricular tachycardia (see Figs. 7.5 and 7.7).
  - Criteria for LV or atrial enlargement (see previous section).
  - Left bundle branch block (see Fig. 7.9).

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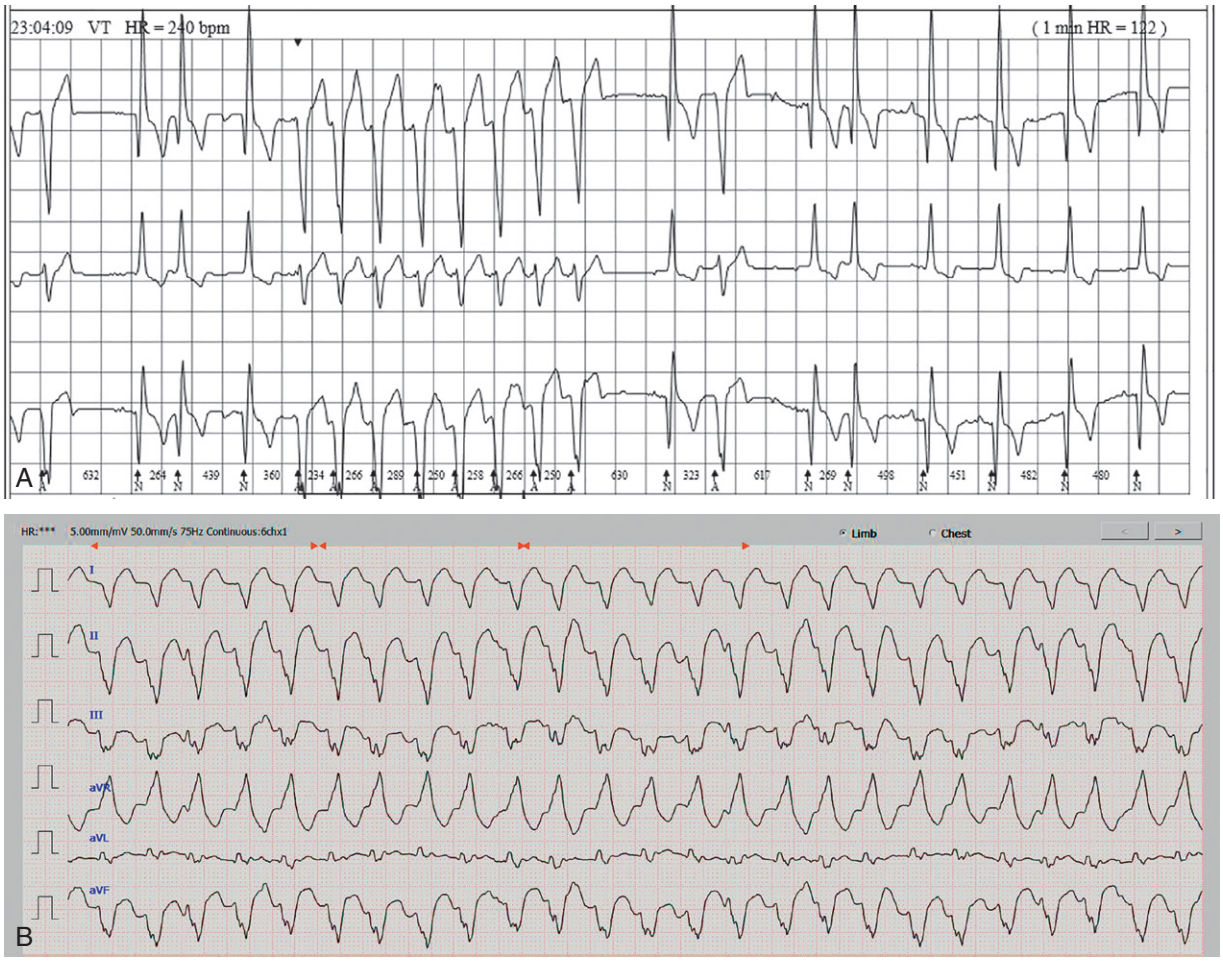


Fig. 7.7 Dobermanns with ventricular tachycardia due to dilated cardiomyopathy (DCM). (A) Extract from Holter recording from a Dobermann with preclinical DCM showing a paroxysm of ventricular tachycardia at a fast instantaneous rate. Make sure the Holter software detects each ventricular ectopic (*arrows*). The instantaneous heart rate can be calculated by 60,000 divided by the time shown between ectopics (in milliseconds). (B) Six-lead ECG showing sustained ventricular tachycardia, following a syncopal event. One-second marks are shown with each red arrow. The instantaneous heart rate during this episode was 300 beats a minute (15 ectopics in 3 seconds).

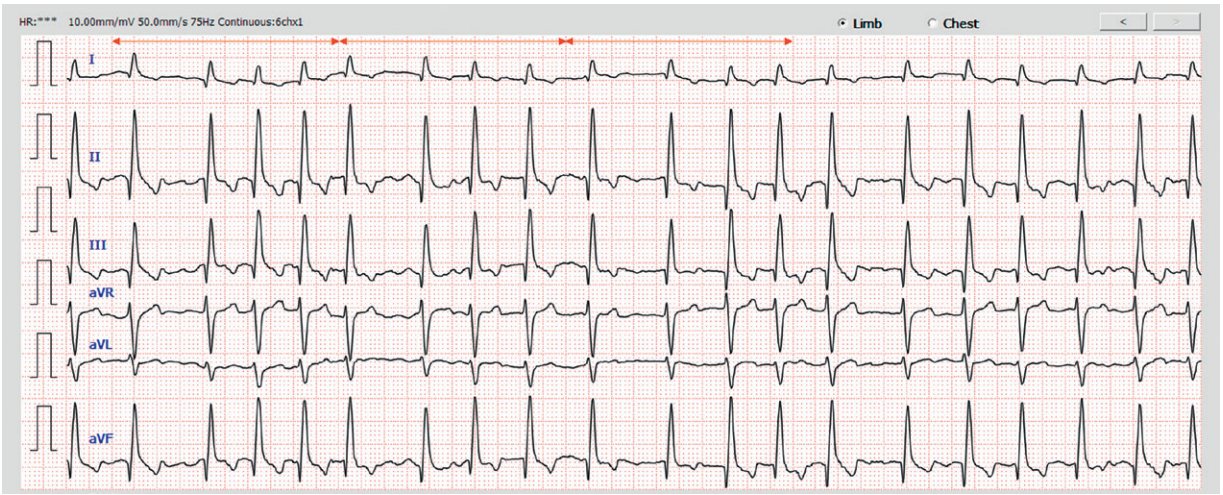


Fig. 7.8 Fast atrial fibrillation in a Weimaraner with dilated cardiomyopathy and biventricular congestive heart failure. One-second marks are shown with each red arrow. The heart rate is approximately 240 bpm, with instantaneous rates (beat to beat) in this section varying between 176 and 300 bpm. It is very important to achieve ventricular response rate control quickly.

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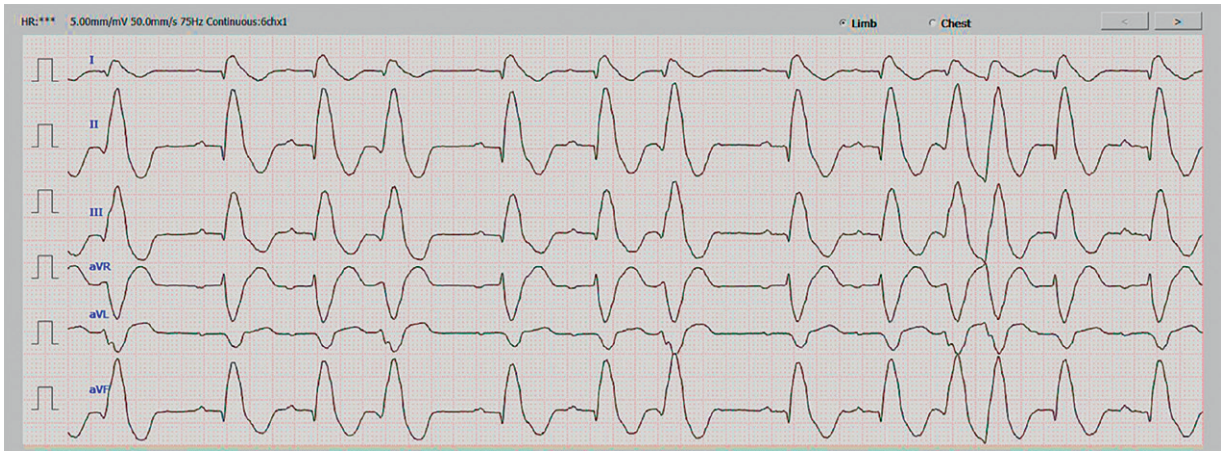


Fig. 7.9 Boxer with dilated cardiomyopathy phenotype and congestive heart failure showing a left bundle branch block and supraventricular premature complexes. In sinus rhythm (e.g., for QRS complexes 2, 3, 5, and 6), the P waves preceding the QRS complexes can be appreciated. However, the QRS complex is significantly prolonged in duration (0.10 second) with an oppositely directed T wave and normal mean electrical axis. This is consistent with a left bundle branch block. Similar morphology QRS complexes in all leads, occurring prematurely without obvious P' waves are consistent with supraventricular premature complexes (e.g., QRS complexes 4, 7) with a couplet of supraventricular premature complexes 10 and 11. Histopathology would be required to confirm that this was due to arrhythmogenic right ventricular cardiomyopathy, but given the breed, this diagnosis is likely.

Key Point

Holter monitoring should be considered the gold standard for detecting preclinical DCM in Dobermanns, especially since arrhythmias can precede any echocardiographic abnormality. Holter monitoring is relatively easy to perform, and there are veterinary Holter companies, with equipment readily available to rent by the general practitioner with instructions for fitting it to obtain a diagnostic recording. These companies then carry out the analyses and send the report to the primary veterinarian. Many veterinary telemedicine or remote consulting practices lease Holter units and assist in result analysis.

Staging of Dilated Cardiomyopathy

The ABCD classification has been applied to canine DCM. See Table 7.3.

Table 7.3 Staging of Canine DCM or ARVC

STAGE	CRITERIA
A	Echocardiographically and electrically normal heart and no clinical signs. Dogs with breed or familial predisposition or a known genetic mutation associated with DCM/ARVC.
B1	Asymptomatic dogs. Arrhythmias associated with DCM/ARVC but no echocardiographic abnormalities. Includes ventricular arrhythmias in Dobermanns/Boxers or atrial fibrillation in giant breeds.
B2	Asymptomatic dogs. Left ventricular systolic dysfunction and chamber dilatation, i.e., echocardiographic abnormalities. May also have ECG/Holter abnormalities.
C	Current or previous (treated) congestive heart failure. Dogs symptomatic for arrhythmias, and receiving treatment for this arrhythmia.
D	End-stage DCM, usually refractory congestive heart failure, requiring individualized treatment optimization.

ARVC; Arrhythmogenic right ventricular cardiomyopathy; *DCM*, dilated cardiomyopathy.

Thoracic Radiography

Thoracic radiographs are still considered to be the "gold standard" for the diagnosis of cardiogenic pulmonary edema with left-sided heart disease including DCM. The triad of signs, including left atrial enlargement, pulmonary venous congestion, and an interstitial to alveolar lung infiltrate, which may be predominantly perihilar, are typical (Figs. 7.10, 7.11). Note that prior diuretic treatment may resolve the radiographic evidence of pulmonary venous congestion, however.

- Chest radiographs are relatively insensitive to mild increases in heart size. A single set performed in the asymptomatic occult phase contributes relatively little to the immediate diagnosis; however, serial radiographs are well suited to monitor progressive heart enlargement and progression of disease. In the overt clinical phase, radiographs are invaluable in helping to diagnose CHF and to monitor response to treatment (Fig. 7.10).
- The chest radiographs in Dobermanns (Fig. 7.11) are often misleading in that heart enlargement is less striking than in other breeds with similar clinical signs. In these instances, cardiac ultrasound helps to determine the magnitude of left-sided

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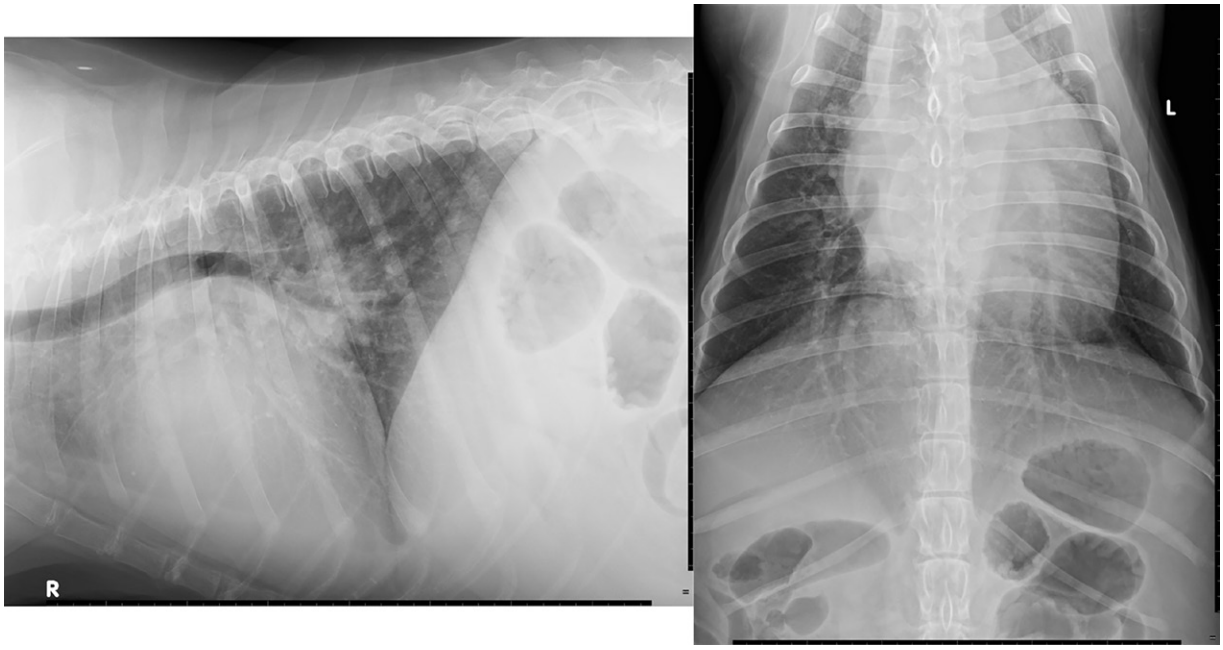


Fig. 7.10 Thoracic radiographs from a Newfoundland with dilated cardiomyopathy and biventricular congestive heart failure. Right lateral (*left*) and dorsoventral views. Note it was not possible to include the entire thorax on a single exposure, so selected exposures are shown. There is generalized cardiomegaly, with evidence of a small pleural effusion and loss of abdominal serosal detail due to ascites. There is a mild bronchointerstitial pattern, which is predominantly perihilar.

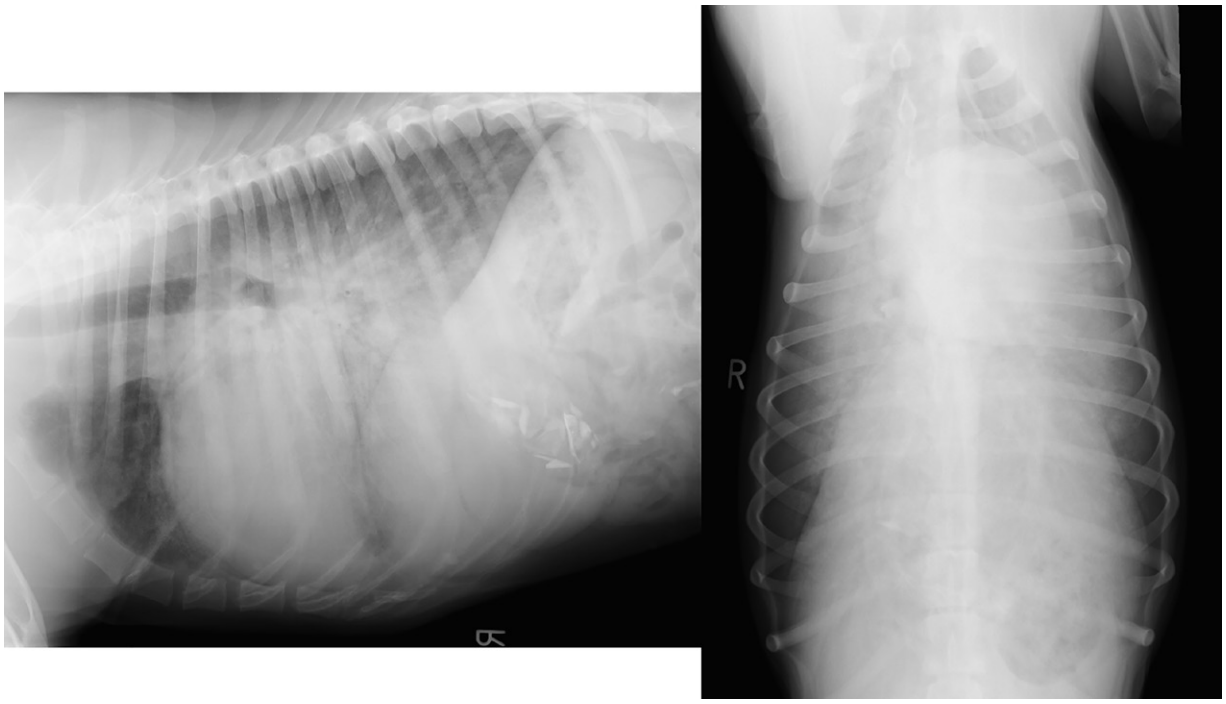


Fig. 7.11 Radiographs from a Dobermann with dilated cardiomyopathy (DCM) and fulminant cardiogenic pulmonary edema. Right lateral (*left*) and dorsoventral views of the thorax. There is a mixed but predominantly alveolar pattern, effacing the cardiac silhouette, especially on the dorsoventral view (*right*). Subjectively, in Dobermann DCM, there often does not appear to be massive generalized cardiomegaly on radiographs, but the left atrial enlargement is prominent. In this Dobermann, estimated vertebral heart sum was 12.3, so significantly increased. The deep chest of this breed often masks the severity of the cardiomegaly.

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heart enlargement and systolic dysfunction. However, once significant heart enlargement is present, in most cases, the radiographs appropriately identify it and also confirm the presence of CHF, if suspected.
- The chest radiographs in dogs with arrhythmogenic cardiomyopathy, including Boxers with ARVC, are unremarkable if there are no echocardiographic abnormalities.

Clinical Pathology

Routine hematology and biochemistry should be carried out in dogs presenting with clinical signs, to exclude primary disease or other comorbidities and also to assess the consequence of DCM, such as reduced perfusion or organ congestion and the consequences of medication (especially azotemia with diuretic therapy).

- Biochemistry is most important. The following abnormalities may be identified:
  - Prerenal azotemia (urea elevated to greater extent than creatinine) which may reflect impaired renal perfusion or prior diuretic medication, renal venous congestion (with right-sided CHF). Concurrent chronic kidney disease should be excluded. Ideally, urine analysis should be carried out prior to diuretic therapy to assess concentrating ability (urine specific gravity). Azotemia will reduce clearance of certain drugs, which is especially important for digoxin due to its narrow therapeutic index.
  - Hypokalemia; this is usually as a consequence of diuretic therapy. Potassium should be supplemented if severe hypokalemia is identified (e.g., <3.0 mmol/L), as it may contribute to weakness, increases risk for arrhythmias and decreases efficacy of some antiarrhythmic drugs. It may also predispose to digoxin toxicity.
  - Other electrolyte abnormalities include hypochloremia (expected consequence of aggressive treatment with loop diuretics, which block the chloride cotransporter in the ascending limb of the loop of Henle). Hyponatremia (and hypochloremia) may be dilutional, reflecting the intense neurohormonal activation in CHF, with release of vasopressin (antidiuretic hormone; ADH), which results in distal renal tubular absorption of free water. Hyponatremia (e.g., <130 mmol/L) is a poor prognostic indicator, unless it was provoked by initial aggressive diuresis, which may resolve with dose reduction as clinical signs improve.
  - Increased liver enzymes, especially ALT, due to hepatic venous congestion. Increased liver enzymes and bilirubin are of poor prognostic significance.
  - Mildly reduced total protein and albumin, especially if there is loss into ascitic fluid with right-sided CHF (congested hepatic sinusoids leak plasma proteins).
  - Note that dogs with preclinical DCM are likely to have normal biochemistry results unless there is a concurrent condition.
- Hematology is generally unremarkable in DCM, but the following findings of minor importance may be identified:
  - Mild nonregenerative anemia, which may be dilutional due to fluid retention as a consequence of activation of the renin-angiotensin-aldosterone (RAAS) system, or reflect chronic disease.
  - An inflammatory or stress leucogram may be evident, with mild neutrophilia monocytosis and lymphopenia/eosinopenia, but this is not specific for cardiac disease.
- Thyroid function.
  - Many breeds at risk of DCM also have breed predispositions for hypothyroidism, including Dobermanns. Therefore thyroid function should be assessed.
  - No actual association has been reported between DCM and hypothyroidism.
  - There have been occasional case reports of impaired LV systolic function in hypothyroid dogs improving with thyroid supplementation.
- Cardiac biomarkers. These are proteins that are elevated in the presence of cardiac disease, but they are not specific for DCM.
  - *N*-terminal proBNP (NT-proBNP) assay is increased when there is increased myocardial wall stress. The species-specific canine assay must be used. The current second-generation assay is more stable than the original assays, and degradation during transport to the lab is less likely.
    - It is therefore reliably increased in DCM, with levels reflecting the severity of the condition and presence of CHF, so they are of prognostic value.
    - Increase in NT-proBNP correlates with increasing left ventricular and left atrial size on echocardiography.
    - NT-proBNP has been described as a method of "prescreening" dogs in predisposed breeds for preclinical DCM, especially Dobermanns, to focus echocardiography on dogs with abnormal results. Various cutoffs have been proposed, ranging from 548 to 735 pmol/L, but sensitivity and specificity are not perfect. Note that these cutoffs are less than the normal range cited of <900 pmol/L.
    - NT-proBNP is not sensitive at detecting the arrhythmogenic forms of cardiomyopathy.
    - It is important to be aware that some breeds have higher NT-proBNP values, even though healthy (e.g., Labradors). In addition, there can be considerable day-to-day variability of levels.
    - NT-proBNP may be falsely increased in presence of azotemia.

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- Cardiac troponin I (troponin I) is cardiac specific and leakage is from injured cardiomyocytes, so elevations are common in all forms of cardiomyopathy.
  - There are many available commercial troponin I assays, which are not species specific. Some have high sensitivity or ultrasensitivity.
  - Higher levels imply worse prognostic significance.
  - In predisposed breeds, troponin I (especially the high or ultrasensitive assays) can be used as method of prescreening for DCM (especially Dobermanns), to focus echocardiography/Holter monitoring on those dogs with abnormal values. Sensitivity and specificity are not ideal.
  - Troponin I may also be increased with secondary myocardial injury due to systemic disease
  - Very high troponin I concentration (e.g., >1.5 ng/mL) may indicate myocarditis, especially if the patient has malignant ventricular arrhythmias or atrioventricular block. It should prompt a search for possible infectious disease.

Identifying or Excluding Nutritional Deficiencies

- Taurine deficiency has been described in some breeds in association with DCM. Breeds include American and English cocker spaniels, Golden retrievers and Newfoundlands. If deficient, the DCM could be considered a secondary (nutritional) DCM (see later in this chapter), but supplementation does not completely reverse the DCM in most patients. This response may reflect an interaction between genetic susceptibility to DCM and the environment (diet).
  - The cause of taurine deficiency is not understood, since most dogs are on an appropriate diet. There may be impaired absorption or metabolism.
  - Taurine can be assayed on plasma (usually heparinized) samples, with commercial assays available at significant cost. The plasma must be rapidly separated after centrifugation from the cells/platelets (which are rich in taurine). The sample should not be hemolyzed. A plasma concentration of >50 nmol/L is considered normal, and values of <50 nmol/L are deficient (most taurine-deficient dogs have significantly lower values).
  - Whole blood taurine is considered superior, but in most countries, the laboratory analysis is not commercially available. Whole blood levels of >200 nmol/L are considered normal. Whole blood levels are less influenced by recent diet or food intake.
- L-carnitine deficiency cannot be diagnosed on circulating levels with a simple blood sample. The deficiency is myocardial and has been historically described in a family of Boxer dogs with a DCM phenotype that improved following L-carnitine supplementation. Endomyocardial biopsy to determine myocardial L-carnitine tissue concentration is rarely performed.
  - In American Cocker Spaniels with taurine deficiency, improvement was seen with both taurine and L-carnitine supplementation.
  - There is little evidence of L-carnitine deficiency in other breeds.

Genetic Testing

Historically, pedigree analyses in breeds with familial DCM suggested that DCM may be a single-gene condition in most breeds, compatible with an autosomal dominant transmission, with incomplete and age-associated reduced penetrance. Progress has been slow identifying loci or genes implicated in canine DCM, with most progress in Dobermanns. It is most likely that DCM is multigenic or multifactorial.

- In Dobermanns, genetic mutations have been identified in genes encoding:
  - PDK4 (pyruvate dehydrogenase kinase, isoenzyme 4), a mitochondrial protein that is nuclear encoded.
  - Titin (TTN), the largest cytoskeletal protein in cells.
  - In a European/UK population of Dobermanns, a locus was identified on chromosome 5, but the causal gene has not been identified.
  - Some Dobermanns with DCM do not have these mutations, implying there are additional genetic causes or other causes of the phenotype (phenocopies).
  - Penetrance is reduced, not just at a younger age; some Dobermanns with one of the mutations may never develop DCM.
  - Genetic testing is recommended in breeding Dobermanns (PDK4, TTN) to attempt to reduce the frequency of these causal mutations in the breed and subsequently prevalence of DCM in this breed.
- In the Irish Wolfhound, genome-wide association studies have identified six different loci with variants associated with DCM.
- In the standard Schnauzer, with an autosomal recessive form of DCM in young adults, a mutation in the RBM20 gene (RNA-binding motif protein 20). The mutation is also reported in giant Schnauzers.
- A single Welsh Springer Spaniel family of young-adult onset DCM has been reported. An association with a mutation in an exon of the phospholamban gene was recognized. It is not known how common DCM or this mutation are in the breed. Transmission is autosomal dominant with reduced penetrance. The genetic test is commercially available.
- Portuguese water dogs with a juvenile-onset, autosomal recessive form of DCM and have had a locus

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mapped to chromosome 8, although the causal gene is yet to be identified.
- In other breeds (e.g., Great Danes, Newfoundlands), despite evidence of familial DCM (most likely autosomal dominant transmission with reduced and age-related penetrance), confirmation of loci or associated genetic variants has proved elusive to date.

TREATMENT

Treatment of DCM depends on the phase of the disease (preclinical or overt), the presence of chamber dilatation, the presence of CHF, and the presence of arrhythmias such as atrial fibrillation or significant ventricular arrhythmia. Ideally, treatment of DCM follows an evidence base, which shows a particular treatment can improve the outcome or survival time, maintain or improve the quality of life, slow the progression of preclinical disease to overt DCM, and reduce the risk of sudden cardiac death.

Dilated Cardiomyopathy With Congestive Heart Failure

It should be acknowledged that the dog with overt DCM and CHF is the "tip of the iceberg"; the patient will have had preclinical disease for months or usually years before the condition results in clinical signs.
- Dogs may show forward heart failure signs (lethargy, weak femoral pulses, quiet heart sounds, pallor with sluggish capillary refill, etc.). Blood pressure may be low (cardiogenic shock).
- Most commonly, they show signs of CHF (backwards), which can be left- or right sided.
  - Left-sided CHF (cardiogenic pulmonary edema) may be life-threatening dyspnea or more chronic (tachypnea) with increased respiratory effort and a restrictive breathing pattern.
  - Right-sided CHF: ascites, hepatic venous congestion, possible pleural effusion, etc.
- Dogs presenting severely decompensated with acute or chronic CHF, especially fulminant pulmonary edema, require oxygen supplementation, anxiolysis (e.g., butorphanol), and aggressive diuresis for cardiogenic pulmonary edema. If significant pleural or abdominal effusion compromising respiration, these should be drained. In patients with cardiogenic shock (hypotensive), positive inotropic support is indicated. Stabilization is required before pursuing diagnostic investigations.

Diuretics

Diuretics are mandatory in any patient with CHF.
- Furosemide is most commonly used. This is a ceiling loop diuretic that blocks the chloride cotransporter in the ascending limb of the loop of Henle, blocking reabsorption of chloride, sodium, potassium, and water.
- In the acutely dyspneic patient with pulmonary edema, furosemide is given as 1 to 2 mg/kg IV boluses, repeated every 1 to 2 hours, until the respiratory rate declines to <40 rpm, when the interval between doses can increase.
- Renal function and electrolytes need to be monitored prior to and during diuresis.
- The patient is transitioned to oral furosemide, with the dose depending on the individual patient, often 2 mg/kg BID or TID. The dose should be gradually downtitrated to the minimum required to maintain a normal sleeping or resting respiratory rate (e.g., <25 or 30 breaths a minute).
- Right-sided CHF with ascites is rarely an emergency, but ascites can become very tense and compromise diaphragmatic function. A significant pleural effusion requires thoracocentesis. IV furosemide is effective in the initial stages, but body cavity fluid that compromises respiration should be drained emergently.
- Furosemide may have variable gastrointestinal absorption and bioavailability, especially if there is right-sided CHF or bowel edema.
- Making a transition to torasemide, which is 10 to 20 times more potent loop diuretic than furosemide, can be helpful in right-sided CHF or refractory left-sided failure. Close monitoring of renal function and electrolytes is recommended. Torasemide dose is usually selected as 1/10th or 1/20th of the total daily furosemide dose when making the transition, or 0.1 to 0.6 mg/kg/day.
- In right-sided CHF, sequential nephron blockade can be helpful; this is the addition of another diuretic(s) acting in a different part of the nephron. Hydrochlorothiazide (alone or with amiloride) is often selected as an additional diuretic(s). Dose of hydrochlorothiazide is usually 1 to 2 mg/kg SID or BID. Electrolytes and renal function should be monitored.
- Spironolactone may also be added (see later); it is a mild diuretic that is potassium sparing.
- Importantly, diuretics should not be used on their own; they will further activate the RAAS. For chronic use, they should be used with antagonists of the RAAS such as ACE inhibitors (see later) or drugs such as inodilators that improve hemodynamic status.

Inodilators or positive inotropes and vasodilators

Positive inotropes may be indicated when there is such poor systolic function that the blood pressure is not maintained, even though this is a homeostatic priority; low blood pressure in this situation is called *cardiogenic shock* and positive inotropic support is indicated. Fluid therapy is contraindicated in a patient with CHF.

Vasodilators may be balanced or be predominantly an arteriodilator or venodilator. Arteriodilators will reduce afterload and therefore the workload of the failing heart (and thereby also reduce myocardial oxygen

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consumption). Venodilators reduce preload, reducing filling pressures within the heart, and increasing capacitance of the systemic circulation, so diverting blood away from the cardiopulmonary circuit, helping to alleviate pulmonary edema.

Pimobendan has both positive inotropic and vasodilator effects (called an *inodilator*) and this drug is certainly indicated to treat DCM.

- It is a calcium sensitizer, which increases the cardiomyocyte contractile apparatus response via troponin C to respond to calcium and increase contractility.
- It is a phosphodiesterase 3 (PDE3) inhibitor, which increases cyclic AMP in both cardiomyocytes (which increases intracellular calcium and contractility) and vascular smooth muscle cells (which inhibits myosin light chain kinase and therefore results in vasodilation).
- Theoretically, the PDE3 inhibition, by increasing intracellular calcium in cardiomyocytes, may be proarrhythmic. However, this does not appear to be a clinical problem at recommended doses even in breeds like Dobermanns at high risk of ventricular arrhythmias. Pimobendan is safe and effective.
- Studies show pimobendan increases survival and quality of life in dogs with clinical DCM and Dobermanns with preclinical DCM (PROTECT study) (data pending in non-Dobermann breeds).
- The oral dose is 0.1 to 0.3 mg/kg BID. Some formulations may be better absorbed on an empty stomach.
- It is available as an IV preparation at a dose of 0.15 mg/kg. This route is useful when immediate positive inotropic support is required, while also reducing afterload and preload, so myocardial oxygen consumption is not increased.

Dobutamine may also be used as a constant-rate infusion if positive inotropic support is indicated in the emergency situation of the patient with cardiogenic shock. However, as a synthetic beta-agonist that increases contractility, it will increase myocardial oxygen consumption and can be proarrhythmic. Furthermore, it will increase the heart rate, which is detrimental in uncontrolled tachyarrhythmias such as atrial fibrillation. This is only appropriate to use in an ICU setting with continuous ECG monitoring.

Other **vasodilators** may be prescribed to treat DCM, including ACE inhibitors (see later).

- Nitroprusside as a constant-rate infusion can be considered if rapid afterload reduction is required (e.g., fulminant pulmonary edema), provided the dog is not hypotensive.
- The venodilator, nitroglycerine (glyceryl trinitrate), is topically applied (patches or ointment) and can also theoretically help in controlling pulmonary edema by increasing systemic venous capacitance. However, there is limited evidence regarding absorption or efficacy.

Counteract the neuroendocrine activation in congestive heart failure

The neuroendocrine activation, especially due to the RAAS, is well recognized in patients with advancing heart disease and particularly CHF. In the chronic setting, this exacerbates fluid retention and vasoconstriction, as well as remodeling of the heart and vascular smooth muscle cells, so RAAS activation is detrimental. Historically important studies have shown that counteracting RAAS with angiotensin converting enzyme inhibitors (ACE-I) improves survival time and quality of life in dogs with CHF due to DCM. As mentioned previously, furosemide will also provoke further activation of RAAS. See also Chapter 15.

- **ACE inhibitors** are licensed to treat dogs with CHF. There are a number of ACE-I available, but currently, market leaders are enalapril and benazepril.
  - ACE-I are not emergency drugs; they are for long-term oral therapy. They should not be prescribed if the patient is azotaemic or hypotensive, which may be a consequence of DCM or aggressive diuresis. Blood pressure, electrolytes, and renal function should be monitored before and after starting these drugs.
  - Recent evidence suggests that it is important to optimize the dose given, rather than remaining on the low end of the dose rate, possibly by giving it twice a day. For benazepril and enalapril, the aim should be >0.5 mg/kg/24 h.
- **Spironolactone** directly competes with aldosterone for the mineralocorticoid receptor and blocks many of its effects. Spironolactone therefore has a mild potassium sparing diuretic effect, by blocking reabsorption of sodium and water from the distal convoluted tubules. It has an antifibrotic effect in the myocardium and helps prevent the maladaptive remodeling provoked by aldosterone. The dose is 2 to 4 mg/kg SID. One prospective clinical trial did not show improvement in survival time with the addition of spironolactone to conventional CHF therapy in Dobermanns with DCM, although they were less likely to develop atrial fibrillation.

Standard quadruple therapy of DCM and CHF for chronic therapy usually includes furosemide (or torasemide), an ACE-I such as benazepril or enalapril, pimobendan, and spironolactone.

Antiarrhythmic drugs

Arrhythmias commonly occur in dogs with DCM and treatment therefore often also requires antiarrhythmic drugs.

- **Atrial fibrillation**. If there is significant structural and functional heart disease with atrial fibrillation and a fast ventricular response rate, rate control medication is indicated.
  - Digoxin slows conduction through the atrioventricular (AV) node via its vagomimetic effect. Due to a narrow therapeutic index, it is important to

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be cautious with the dose (e.g., 0.22 mg/m^2 per day, divided BID) and monitor the digoxin serum concentration after 1 week (should be 0.5–0.9 ng/mL at trough level, 8 hours post-pill).
- Diltiazem slows conduction through the AV node via its calcium channel blockade. Although a mild negative inotrope, this characteristic is rarely a clinical problem, since it is offset by rate control. The dose is 1 to 3 mg/kg TID of modified release, or BID for sustained-release diltiazem.
- The combination of digoxin and diltiazem provides better rate control than either drug alone; the rate control should be assessed by a Holter monitor to determine the mean and maximum heart rate (HR) over 24 hours. The aim would be to have mean HR/24 hours <125 bpm (ideal) or <140 bpm (if severe disease, where maintaining cardiac output requires a faster heart rate).
- Sotalol can also offer rate control plus efficacy if there are concurrent ventricular arrhythmias. This is also negatively inotropic, but rarely a clinical problem. It may be an alternative to digoxin/diltiazem.
- Amiodarone can also reduce ventricular response to atrial fibrillation but it is not likely to result in cardioversion of atrial fibrillation given significant structural heart disease and atrial stretch. It is also effective if there are concurrent ventricular arrhythmias. However, side effects are common (e.g., hepatotoxicity, gastrointestinal, thyroid dysfunction) and Dobermanns may be particularly predisposed to these. It should be only used if antiarrhythmic efficacy is not achieved with other medications.

- **Ventricular arrhythmias**. Hemodynamically significant (i.e., result in collapse) or malignant ventricular arrhythmias (e.g., fast instantaneous rate ventricular tachycardia, R on T phenomenon or polymorphic ectopy) will require antiarrhythmic therapy.
  - In the patient presenting with ventricular tachycardia resulting in clinical signs, lidocaine is the drug of choice and is effective in >80% of cases. A bolus of 2 mg/kg IV is given, and this may be repeated if the arrhythmia does not improve in a few minutes, up to total dose of 8 mg/kg. If successful, maintain the patient on a lidocaine constant-rate infusion (e.g., 50 µg/kg/min).
  - If conversion is not successful with lidocaine, the following IV drugs may be considered, if available: IV Procainamide, IV Esmolol, or IV Sotalol. Amiodarone can be given IV. In the original formulation with polysorbate 80, anaphylaxis can occur, so it is better given after premedication with antihistamines and with close monitoring, and as a CRI that can be stopped, rather than a bolus. New, safer formulations of this drug are now available.
  - For oral management of ventricular arrhythmias, Sotalol or Mexiletine are most commonly prescribed.
  - Holter monitoring may indicate that the ventricular arrhythmia is worse during excitement or exercise, at fast heart rates, suggesting an influence of catecholomines (Sotalol, with weak beta-blocker effect, may be useful). Sotalol is dosed at 0.5 to 2.5 mg/kg PO BID.
  - Conversely, if ventricular arrhythmias are most likely during a slow heart rate or during sleep, sotalol may be better to be avoided. Mexiletine, if available, is effective at a dose of 5 to 8 mg/kg PO TID.
  - After prescribing an antiarrhythmic drug, it is important to ensure efficacy and to exclude pro-arrhythmia (worsening the arrhythmia). This is best achieved by Holter monitoring. Reduction of complexity, especially runs and rate of ventricular tachycardia is the aim, rather than reduction of total count of VPCs; there is a significant day-to-day variability in numbers of VPCs/24 hours (up to 80%).
  - Some refractory ventricular arrhythmias respond well to a combination of sotalol and mexiletine.
  - Oral amiodarone can be used with refractory ventricular arrhythmias. As mentioned above, there is a risk of side effects and close monitoring is required. As there is a long half-life, oral loading doses are used, with a dose reduction over a few weeks:
    - Week 1: 10 to 15 mg/kg BID
    - Week 2: 5 to 7.5 mg/kg BID
    - Week 3 onwards: 5 to 7.5 mg SID
    - A serum drug level of amiodarone and its metabolite (desethylamiodarone) should be taken after 1 month, and liver enzymes and thyroid function monitored at that time and every 3 months.
  - Note: No antiarrhythmic drug regimen has been proven to reduce the risk of sudden death, even though they can improve Holter results and alleviate clinical signs.
  - Beta-blockers in humans with DCM and other cardiac diseases and chronic heart failure have been shown to improve survival time and quality of life. Beta-blockers are considered standard of care in humans (with ACE inhibitors). However, they are negatively inotropic and are contraindicated in uncontrolled CHF, where patients are maintaining their cardiac output with high sympathetic drive. It can take 6 months in a human to see a benefit, but in dogs with DCM, this is likely to be a significant portion of their remaining life. Many humans and dogs fail to tolerate a beta-blocker. It may just be specific beta-blockers showing a survival benefit in humans with chronic heart failure (e.g., carvedilol, bisoprolol, metoprolol) rather

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than hydrophilic ones like atenolol. So far, beta-blocker studies have not shown a similar reduction in mortality or morbidity in dogs with DCM to that shown in humans, so further studies are required. Beta-blockers would counteract some of the neuroendocrine activation and remodeling associated with high catecholamine drive. Beta-blockers also slow conduction through the AV node so could be considered an additional drug to control ventricular response to atrial fibrillation, if tolerated. If beta-blockers are used, the initial dose should start low, and be slowly uptitrated over weeks to reduce side effects such as lethargy or weakness. They should not be suddenly stopped, but slowly downtitrated.

Preclinical Dilated Cardiomyopathy Based on Echocardiographic Diagnosis

Preclinical DCM diagnosed on echocardiography is characterized by left ventricular dilatation in diastole and systole, with impaired LV systolic function. There is good evidence based on the PROTECT study in Dobermanns that **Pimobendan** will slow progression and prolong time to onset of CHF or death. These results have been extrapolated to other breeds with preclinical DCM, but studies to confirm that pimobendan has a similar effect in these dogs are not yet published.

ACE inhibitors such as enalapril or benazepril may also slow the rate of progression of preclinical DCM in Dobermanns, similar to their effect in humans with left ventricular dysfunction. Whether pimobendan in combination with an ACE inhibitor is superior to either drug alone is not known.

The role of beta-blockers in preclinical DCM (extrapolating from human recommendations) is not known. Beta-blockers may be better tolerated in asymptomatic dogs.

Arrhythmias in Preclinical Dilated Cardiomyopathy

- **Atrial fibrillation (AF)** is not common in preclinical DCM, but it can occur in giant breed dogs such as the Irish Wolfhound. The rate is usually not excessive, and rate control may not be required (should be checked by Holter monitoring to obtain a mean HR/24 hours).
  - If rate control medication is indicated, as noted above, digoxin and/or Diltiazem are most commonly prescribed.
  - In a dog with minimal structural or functional cardiac disease evident on echo, often described as "lone" AF, cardioversion can be considered. This is more likely to be successful if it is known that AF is recent onset. Amiodarone may result in pharmacological cardioversion, or pretreatment may make it more likely AF is converted back to sinus rhythm by DC cardioversion. However, it is highly likely that AF will recur sometime after the cardioversion.
- **Ventricular arrhythmia** is a hallmark of dilated cardiomyopathy in certain breeds, especially the Dobermann. It may occur prior to or without any echocardiographic abnormalities. In asymptomatic dogs, it is diagnosed usually during Holter screening or monitoring once an arrhythmia has been identified.
  - If the Holter shows malignant ventricular arrythmias, treatment as described above (usually Sotalol and/or Mexiletine).
  - Follow-up Holters are indicated to confirm efficacy and exclude proarrhythmia.
  - No antiarrhythmic drug regime is proven to reduce the risk of sudden cardiac death.

Supplements and Nutraceuticals

In dogs with a proven or suspected deficiency, supplementation may well result in some improvement of DCM, if it was associated with that deficiency.

- Taurine deficiency in American and English Cocker Spaniels, Golden Retrievers, or Newfoundlands should respond to taurine supplementation, reducing the dose or requirement of other conventional cardiac drugs. Note, however, this is not usually a completely reversible DCM. Some American Cocker Spaniels also require L-carnitine supplementation.
- A family of Boxer dogs with a DCM phenotype confirmed to have myocardial L-carnitine deficiency responded to L-carnitine supplementation. These supplements are not likely to cause harm and may be of benefit.
- There is much less evidence to support a vitamin deficiency or response to supplementation. Vitamins E, A, and C have antioxidant properties but the benefits of adding them are unproven in dogs with DCM. Dogs with CHF, including those with DCM, have a reduced vitamin D status compared with healthy dogs, but the role of supplementation of vitamin D in CHF is not known. Oversupplementation of vitamin D and other vitamins should be avoided.

CHF is an inflammatory condition, associated with elevated circulating levels of proinflammatory cytokines such as tumour necrosis factor alpha (TNFα), interleukin (IL-)1β, and IL-6. These have systemic effects such as reduced appetite, muscle wasting (cachexia), and weight loss.

- Fish oil supplementation (27 mg/kg/day Eicosopentanoic acid (EPA) and 18 mg/kg/day Docosahexaenoic acid (DHA); high omega-3) reduces circulating levels of inflammatory cytokines (IL-1β).
- Fish oil supplementation may also have an antiarrhythmic effect, reducing ventricular arrhythmias.

There are also changes in cardiomyocyte energy production and metabolism.

- Coenzyme Q_{10} (Ubiquinone) may improve mitochondrial energy production (oxidative phosphorylation; electron carrier), but evidence of efficacy in DCM is not available. It also has antioxidant properties and stabilizes cell membranes.

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PROGNOSIS

The time course from occult to symptomatic DCM is highly variable and can be years. During this phase, serial echocardiograms and ECGs are recommended. Sudden death can occur during the occult phase, especially in Dobermanns. Once clinical signs such as CHF develop, long-term prognosis is poor. Survival times derived from clinical studies are difficult to assess because of nonstandardized treatment, lack of ACE inhibitor use, and statistical issues surrounding euthanasia. Once CHF is present, median survival time is reported to be 3 to 4 months in Dobermanns and 5 to 6 months in other breeds. Dogs that survive more than 7 months may do well for an extended period of time. Dogs that show a good response to treatment, including reverse remodeling (reduction in severity of LV dilatation), may have better survival. There is enormous individual variation. One-year survival is approximately 10% to 15%. Atrial fibrillation (in Dobermanns), biventricular CHF, and young age at time of presentation (<5 years) are associated with worse prognosis.

SECONDARY CARDIOMYOPATHIES RESULTING IN THE DILATED CARDIOMYOPATHY PHENOTYPE

NUTRITIONAL CARDIOMYOPATHY

Nutritional cardiomyopathies can include taurine deficiency (American and English Cocker Spaniels, Golden Retrievers, and Newfoundlands) and L-carnitine deficiency (Boxers with DCM phenotype, possibly some American Cocker Spaniels) and should be considered as secondary DCM, even though supplementation is not completely reversible and a genetic predisposition may also be present.

In recent years, an association between grain-free diets or novel diets with high levels of peas, legumes, lentils, or potatoes and DCM has been described and investigated by the FDA. The reason for the association has not yet been elucidated. Only some of the affected dogs are taurine deficient (mainly Golden Retrievers). Therefore there may be some diet interaction or impaired absorption of another nutrient, or altered metabolism. Because it is nutritional, DCM may occur in atypical breeds or cross-breed dogs or the typical large-breed dogs. It shows that it is very important to obtain a thorough history from owners about their dog's diet. Change of diet or supplementation (if deficient) can result in significant improvement, and dogs may be able to stop diuretics and conventional cardiac medications (Fig. 7.12).

MYOCARDITIS/POSTMYOCARDITIS

Myocarditis needs to be excluded prior to coming to a diagnosis of idiopathic DCM. However, unless in the acute phase, characterized often by malignant ventricular arrhythmias or atrioventricular blocks and associated with marked elevation in troponin I, it is often not suspected or diagnosed. Unless histopathology is carried out, such as at postmortem examination or if endomyocardial biopsies are performed, the diagnosis of myocarditis is missed. It is possible that some DCM cases may have had prior myocarditis. Potential causes include viral (e.g., Parvovirus in young dogs), bacterial, tick-borne conditions, and *Bartonella* species. Myocarditis and DCM could also potentially be immune mediated following an inflammatory process exposing cardiomyocyte antigens to the immune system.

TOXINS AND DRUGS

The anthracycline chemotherapy drugs such as doxorubicin and epirubicin are cardiotoxic, especially with high total cumulative doses. If the total cumulative dose is exceeded (e.g., >240 mg/m^2), there is significant risk for development of a DCM phenotype, including arrhythmias and CHF. Echocardiographic monitoring +/− troponin I monitoring are recommended prior to and during chemotherapy treatment cycles.

ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY

ARVC is well described in Boxers and English Bulldogs; it is familial in these breeds. In the Boxer, it is most consistent with autosomal dominant transmission, with variable penetrance. Sporadically, other breeds are reported with ARVC. Although some dogs may present with a DCM phenotype (see later), the histopathology findings in ARVC are strikingly different from the relatively subtle histological abnormalities in DCM. There is loss of cardiomyocytes and with fatty or fibrofatty replacement. These changes are predominantly or initially in the right ventricle, but they can affect all the myocardium, including the atria.

Affected dogs usually present with malignant ventricular arrhythmias, which can result in syncopal episodes or sudden death. The ventricular ectopics typically show right ventricular origin with a left bundle block pattern on ECG (Fig. 7.13). Echocardiography may be unremarkable. There may be evidence of right heart dilatation (Fig. 7.14), which can be focal (e.g., right ventricular outflow tract, especially in the Bulldog), with impaired right ventricular systolic function (reduced TAPSE). Other affected dogs may show four-chamber dilation with impaired LV systolic function, and an echocardiography shows a DCM phenotype. In these cases, there may be left-sided or biventricular CHF.

In the 1980s, Harpster defined three categories of Boxer dogs with cardiomyopathy, which still apply to the clinical presentations today, and are applicable to Bulldogs:

- Category 1 are asymptomatic dogs with ventricular arrhythmias (e.g., detected on Holter monitoring).

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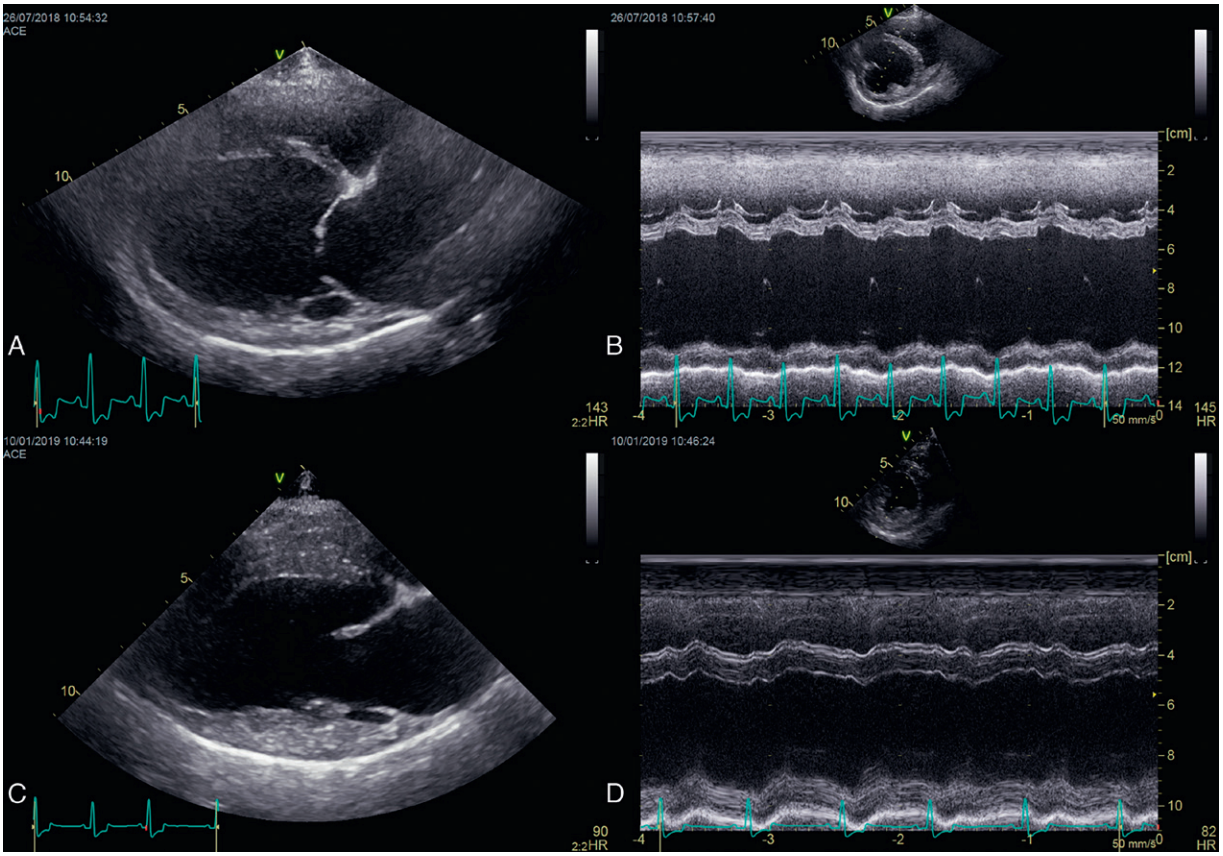


Fig. 7.12 Response to cardiac medication and taurine supplementation in a Golden Retriever. The dog had been on a grain-free diet, when he presented in congestive heart failure in July 2018. Images (A) and (B) at presentation. Images (C) and (D) in January 2019, 6 months later. The dog had been weaned off furosemide but continued on pimobendan and taurine supplementation. (A) Right parasternal long-axis four-chamber view showing marked left dilatation and a dilated, rounded relatively thin-walled left ventricle (LV) at presentation. (B) M-mode confirming severe systolic dysfunction at presentation. (C) Some reverse remodeling with treatment and taurine supplementation. The left atrium was normal in size, and the LV is less dilated and rounded, 6 months later. (D) LV M-mode confirming improved but not normal LV systolic function, 6 months later (note depth of M-mode is less than in the initial M-mode).

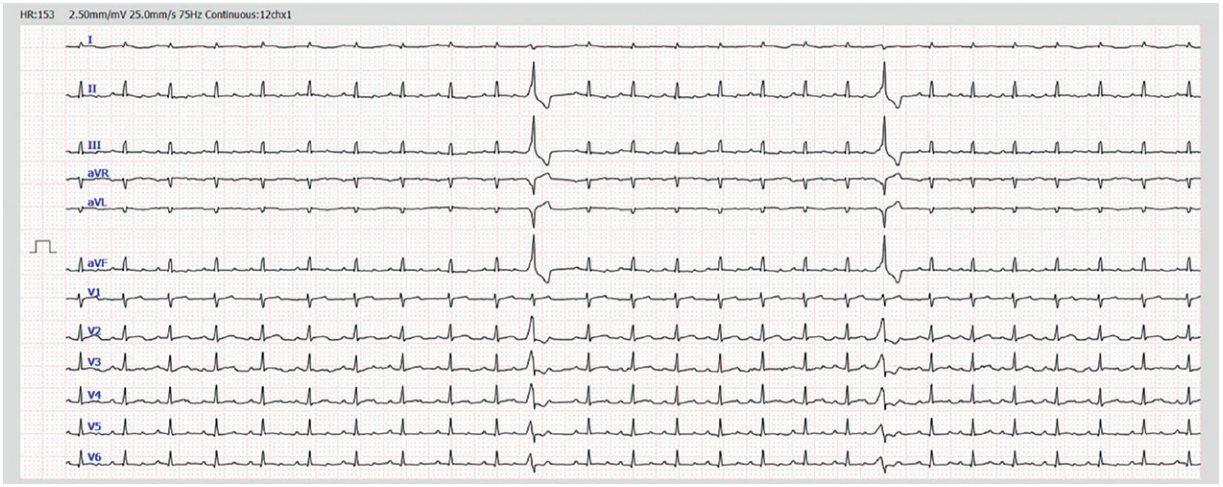


Fig. 7.13 Boxer with arrhythmogenic right ventricular cardiomyopathy (ARVC) showing ventricular premature complexes (VPCs) (12-lead ECG). This Boxer had preclinical ARVC and occasional single VPCs are noted during this 12-lead ECG. In lead II, III, and aVF, the complexes are positive, consistent with right ventricular origin with conduction traveling cell to cell towards the left ventricle apex, so a left bundle block branch pattern. The ventricular ectopics are QRS complexes 11 and 19 from the left. The dog otherwise has an underlying sinus rhythm.

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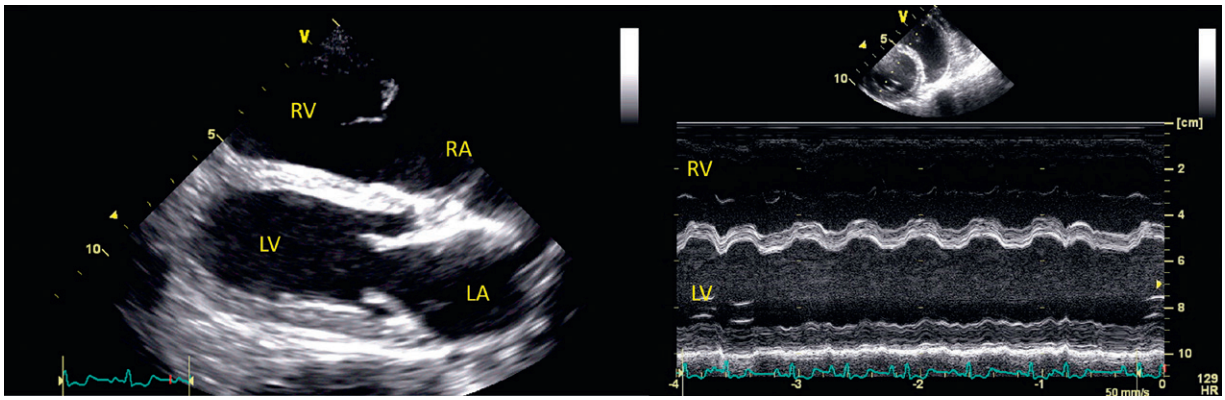


Fig. 7.14 Boxer with arrhythmogenic right ventricular cardiomyopathy showing the dilated cardiomyopathy phenotype. Right parasternal long-axis four-chamber view (*left*) and left ventricular M-mode (*right*). There is marked dilatation of the right atrium and right ventricle and this dog was in right-sided congestive heart failure. However, the left ventricle is also dilated and hypokinetic. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

- Category 2 Boxers may have clinical signs such as syncope, due to ventricular tachycardia, and either mild echocardiographic abnormalities (right heart dilatation, impaired RV function) or none.
- Category 3 Boxers show the DCM phenotype on echocardiography (see Fig. 7.14) and usually present in CHF. Although the characteristic ventricular arrhythmias are present, they may not be the reason for the presentation (no syncope reported), although sudden death may occur.

These different categories apply to the presentation of the dogs, rather than representing a natural history of disease progression. The age of onset with clinical signs is typically in middle to older age. In some studies, Boxers presenting with the DCM phenotype are younger than those presenting with arrhythmias.

Bulldogs may also present similarly; however, supraventricular arrhythmias may be apparent in some individuals.

ETIOLOGY OF ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY AND GENETIC TESTS

Familial disease, best documented in the Boxer dog, indicates a genetic basis to the disease. In human familial ARVC, a number of genetic mutations are known, most of which affect the desmosome. The gene encoding the desmosomal protein, Striatin, was identified to be associated with Boxer dog ARVC. Boxers with the DCM phenotype also had the striatin mutation, and they were more likely to be homozygous for this (genetic test is commercially available). However, the mutation is not present in all cases of familial ARVC in Boxers suggesting other genetic mutations are pending discovery. Furthermore, autoantibodies to desmosomal proteins have been reported in human ARVC cases in which genetic etiology has not been identified, especially to desmoglein-2. Similar autoantibodies have also been identified in Boxer dogs. A recent report, however, has not confirmed the association or presence of these autoantibodies and cardiac disease, although the autoantibodies did correlate with complexity of arrhythmia. The role of autoantibodies in ARVC and DCM in general still needs to be clarified. The cause of ARVC in English Bulldogs is not known, but striatin mutations have not been reported. American Bulldogs may also be affected.

MAKING THE DIAGNOSIS OF ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY

- The characteristic right ventricular origin of the ventricular ectopics in ARVC (left bundle branch block morphology: positive in lead II) is typical of ARVC, so this finding in a predisposed breed with this morphology of ventricular ectopics, especially with a history of syncope or compatible clinical signs is highly suspicious for the diagnosis (see Fig. 7.13).
- Holter monitoring is the main screening tool to identify affected dogs, or for preclinical screening. Holter is indicated to assess the significance, frequency, and complexity of the ventricular arrhythmias, and whether antiarrhythmic medication is indicated. It is also important to assess the response to antiarrhythmic medication and to exclude proarrhythmia.
- The total number of ventricular premature complexes/24 hours on Holter monitoring is usually used to make the diagnosis. The actual cutoff of what is normal or abnormal varies significantly in different studies, but recent criteria have been proposed (see Table 7.2).
- Since there is considerable day-to-day variability of the total number of ventricular ectopics/24 hours, it is difficult to be certain on a single 24-hour Holter screening as to the status of the dog. Breeding dogs (especially Boxers) should have two normal Holter results. The presence of complexity characteristics makes it more likely the dog is affected. However, it must be remembered that the presence of systemic

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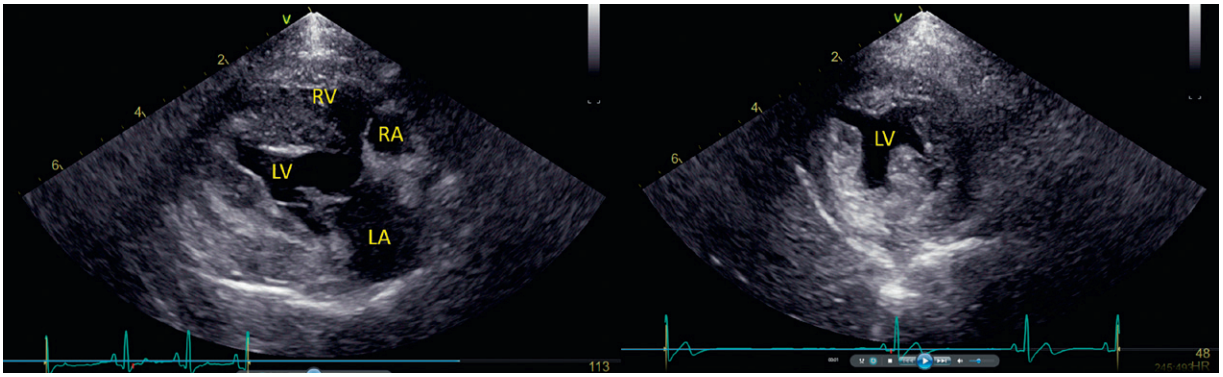


Fig. 7.15 Terrier cross dog with hypertrophic cardiomyopathy. Right parasternal long-axis four-chamber view (*left*) and short-axis view at the level of papillary muscles (*right*). There is generalized concentric hypertrophy of the left ventricle, with prominent, apparently hypertrophied papillary muscles. *LA*, Left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

conditions may result in secondary arrhythmias, and these may need to be excluded before determining cardiac status. ARVC cases may also have supraventricular arrhythmias, so Holter monitoring is also useful to detect this abnormality.

- Echocardiography is less useful as a screening test, since affected dogs may have a completely normal echo study. However, it is important to identify significant structural heart disease as this will prompt appropriate treatment, especially if the DCM phenotype is detected or if the dog is in CHF.
- Other tests are similar to that previously described for DCM, including radiology, clinical pathology including cardiac biomarkers, etc.

HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy is a rare primary myocardial disease in dogs, but it must be considered in dogs with left ventricular concentric hypertrophy, in which conditions that can cause this hypertrophy are excluded, such as systemic hypertension. In a recent multicentered case series, Shih Tzus and Terriers/Terrier crosses were overrepresented and the condition predominantly affects small-breed dogs. Initial reports suggested a male overrepresentation, but this was not confirmed in the recent survey. Typically these dogs can be identified over a wide age range, but mostly middle aged. As in cats with HCM, there may be generalized symmetrical concentric left ventricular hypertrophy (Fig. 7.15), or occasionally, it may be asymmetric. Focal basal septal hypertrophy has not been described in the dog.

- Affected dogs may have a heart murmur, due to systolic anterior motion (SAM) of the anterior mitral valve leaflet, resulting in dynamic left ventricular outflow tract obstruction (DLVOTO). The severity of this obstruction can be alleviated with beta blockade (e.g., Atenolol 0.5–1.0 mg/kg BID).
- In very young dogs, especially terrier breeds, primary abnormalities of the mitral valve resulting in SAM, DLVOTO, and secondary hypertrophy should be excluded. Mitral dysplasia may be suspected on echocardiography, including an elongated anterior leaflet. These very young dogs respond to beta blockade, which reduces the SAM, DLVOTO, and often hypertrophy resolves. It may resolve in other cases without treatment. These cases, despite mimicking HCM with LV outflow tract obstruction, should not be considered primary HCM.
- Diastolic dysfunction is common, with evidence of impaired relaxation associated with the hypertrophy.
- Affected dogs are susceptible to arrhythmias, which may result in syncopal episodes, or sudden death.
- Less commonly, CHF occurs, which needs to be managed accordingly.
- Many dogs remain asymptomatic for years.

Frequently Asked Questions

WHAT CAUSES DILATED CARDIOMYOPATHY?

The etiology of primary dilated cardiomyopathy (DCM) is unknown. DCM is a description of the heart's response to injury (i.e., dilation and systolic dysfunction) and as such may be the final common pathway and end result of multiple causes. Possible causes include genetic/familial, immune mediated or autoimmune, infectious, toxic, or nutritional. Known primary causes of dilated heart chambers with impaired LV systolic function and/or arrhythmias are actively excluded before the diagnosis of idiopathic or primary DCM. There is some evidence of genetic associations with DCM in certain breeds, but even within a single breed such as the Dobermann, it does not appear to be a single-gene disorder with Mendelian inheritance.

WHY SHOULD I SCREEN ASYMPTOMATIC DOGS FOR PRECLINICAL DILATED CARDIOMYOPATHY?

In breeding dogs with familial or breed associated prevalence of DCM, where there is a known or likely breed predisposition to DCM, identifying affected dogs and removing them from the breeding pool may reduce the prevalence of DCM in the breed. Also, pregnancy and lactation with the associated increased volume load may be harmful in dogs with preclinical DCM.

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Dogs with preclinical DCM are at increased risk of general anesthesia, especially if prolonged procedures are indicated. Such dogs may decompensate sometime after their anesthesia. Therefore screening and treatment with pimobendan of dogs with echocardiographic DCM and special monitoring of the higher-risk patient should improve safety of the anesthesia and subsequent status.

There is an advantage to the individual dog if preclinical DCM is identified, particularly those with echocardiographic changes. The PROTECT study in Dobermanns showed that treatment with pimobendan increased the asymptomatic period before development of clinical signs or sudden death by about 9 months. Screening therefore should not just be restricted to breeding animals. Although there is no current published evidence at this time of a benefit of pimobendan in non-Dobermanns, there is no reason to think that they would be different.

There is also no evidence available that antiarrhythmic medication makes a difference to outcome or survival in dogs with the arrhythmogenic forms of cardiomyopathy.

WHY DO I NEED TO ASK ABOUT DIET HISTORY IN DOGS WITH DILATED CARDIOMYOPATHY?

Although the breed associations with taurine and/or L-carnitine deficiency have been known about for a long time, there is recent emerging evidence of a nutritional DCM that is not at this time known to be associated with any known particular nutritional or amino acid deficiency. Grain-free diets, which are currently extremely popular, are implicated, but it may be the substitutes for grain, especially peas, other legumes, pulses, lentils, and potatoes, including sweet potatoes, present in the diet somehow exerting this effect (perhaps by altering absorption or metabolism of key nutrients). In the Golden Retriever, there is often an associated taurine deficiency, but not in all cases. If DCM occurs in atypical breeds, including small-breed dogs, a possible nutritional association should be investigated. Even in a typical DCM breed, this association may make it more likely that DCM is manifest. If the nutritional association is suspected, there may be a dramatic improvement with a diet change to a conventional, good-quality diet.

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Chapter 8

Feline Cardiomyopathy

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The term *cardiomyopathy* literally means heart muscle disease and designates a disorder of the heart that primarily affects the cardiac muscle (myocardium). *Primary* indicates that the myocardial disease is not secondary to valvular disease, pericardial disease, coronary vascular disease, systemic or pulmonary hypertension, congenital abnormalities, or systemic disease. Most primary cardiomyopathies are of unknown etiology (idiopathic). However in human medicine, recent advances in genetic testing have identified causative mutations in many cardiomyopathies previously categorized as idiopathic. A secondary cardiomyopathy is a disease that affects the myocardium secondary to infectious, toxic, metabolic, or other disease processes that affect multiple body systems. In 1995 the World Health Organization categorized the types of cardiomyopathies according to the dominant pathophysiologic features produced by the myocardial disease, which has since then been revised by the American Heart Association in 2006. Veterinary medicine has adopted much of the human categorization of cardiomyopathies because they seem to clinically fit the diseases we see in feline cardiology.

FELINE CARDIOMYOPATHIES

GENERAL COMMENTS

- Cardiomyopathies are classified into functional pathophysiologic categories on the basis of echocardiographic evaluation, including hypertrophic cardiomyopathy (HCM; i.e., left ventricular concentric hypertrophy), dilated cardiomyopathy (DCM; i.e., systolic myocardial failure), restrictive cardiomyopathy (RCM; i.e., marked diastolic dysfunction in the face of a relatively normal left ventricle [LV]), arrhythmogenic right ventricular cardiomyopathy (ARVC), and nonspecific or unclassified cardiomyopathy (UCM; i.e., atrial dilation without other functional myocardial abnormalities).
- Most cardiomyopathies diagnosed in cats are idiopathic (primary). HCM often has a heritable basis and may be caused in some cats within the Maine Coon and Ragdoll breeds by sarcomeric mutations of the individual contractile unit of the heart muscle. The Sphynx breed develops a genetically heritable form of HCM due to a mutation in the ALMS1 gene. The only nutritional cause of a cardiomyopathy in cats is taurine deficiency causing DCM. Although ARVC, DCM, and RCM are currently defined as idiopathic in veterinary medicine, there may be genetic causes that have yet to be identified in cats, just as there have been in people.
- Nonspecific cardiomyopathy (NCM) is a poorly defined clinical entity in cats, with presumed primary myocardial disease that do not meet the criteria for making a diagnosis of HCM, RCM, or DCM.
- Echocardiography is the essential diagnostic test to diagnose the specific cardiomyopathies in cats and is necessary for early and accurate morphologic classification of the cardiomyopathy. It is helpful to guide treatment recommendations and assess risk for adverse events such as arterial thromboembolism or congestive heart failure.

Key Point

Cardiomyopathies represent the overwhelming majority of heart diseases in cats. Other diseases such as valvular disease or pericardial disease are very uncommon. In one study, 62% of 408 cats presenting for cardiology workup were diagnosed with a primary cardiomyopathy. The most common feline cardiomyopathy is HCM, which accounts for approximately 58% to 68% of feline cardiomyopathy cases. The other cardiomyopathies are less common, including RCM (5%–21%), NCM (10%), DCM (10%), and ARVC (<1% of cases).

CLASSIFICATION

- Cardiomyopathies are classified according to their morphologic appearance, typically based on echocardiography (Table 8.1). Within each classification, a wide range of morphologic and clinical presentations may be seen. Primary cardiomyopathies (genetic, nongenetic, and acquired) are confined to the heart muscle. Conversely, secondary cardiomyopathies show myocardial involvement as part of more widespread multiorgan systemic disorders, and were previously referred to as "specific" cardiomyopathies.

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Table 8.1 Echocardiographic Abnormalities in Feline Cardiomyopathy

CARDIOMYOPATHY	ECHOCARDIOGRAPHIC ABNORMALITIES	HEART FAILURE
HCM HOCM	LV wall or septal wall thickness >6 mm, papillary muscle hypertrophy, normal systolic function. Left atrial dilation may or may not be present. HCM with SAM of the mitral valve present, creating mild-to-severe LVOT obstruction and MR.	Diastolic heart failure Left heart failure Pulmonary edema or pleural effusion
DCM	Systolic myocardial failure (FS < 26%, ESD > 11 mm, EPSS > 4), compensatory LV eccentric hypertrophy (EDD > 20 mm), LA +/– RA dilation, +/– RV eccentric hypertrophy and myocardial failure.	Systolic heart failure Left-sided + right-sided heart failure Pulmonary edema, pleural effusion, +/– ascites
RCM Myocardial form EMF form	Normal LV wall thickness, normal systolic function. LA or biatrial dilation. Restrictive filling pattern of mitral inflow (E/A > 2, short deceleration time). Large fibrotic bridging band or scar of the LV, severe LA +/– RA dilation.	Diastolic heart failureLeft-sided +/– right-sided heart failure; pulmonary edema or pleural effusion
NCM	Normal to equivocal LV wall thickness (<6 mm), normal to mild systolic myocardial failure. LA +/– RA dilation; +/– diastolic dysfunction.	Probable diastolic heart failure Left-sided +/– right-sided heart failure Pulmonary edema or pleural effusion
ARVC	Severe RV eccentric hypertrophy and myocardial failure, severe RA dilation, normal to mild LA dilation.	Systolic heart failure Right-sided heart failure; pleural effusion and ascites

ARVC, Arrhythmogenic right ventricular cardiomyopathy; *DCM*, dilated cardiomyopathy; *E/A*, peak E to E-wave velocity ratio; *EDD*, end-diastolic diameter; *EMF*, endomyocardial fibrosis; *ESD*, end-systolic diameter; *EPSS*, E-point to septal separation; *FS*, fractional shortening; *HCM*, hypertrophic cardiomyopathy; *HOCM*, hypertrophic obstructive cardiomyopathy; *LA*, left atrium; *LV*, left ventricle; *LVOT*, left ventricular outflow tract; *MR*, mitral regurgitation; *NCM*, nonspecific cardiomyopathy, *RA*, right atrium; *RCM*, restrictive cardiomyopathy; *RV*, right ventricle; *SAM*, systolic anterior motion of the mitral valve.

Primary Cardiomyopathies

- Hypertrophic cardiomyopathy (HCM)
- Idiopathic dilated cardiomyopathy (DCM)
- Restrictive cardiomyopathy (RCM)
- Nonspecific (previously unclassified) cardiomyopathies (NCM)
- Arrhythmogenic right ventricular cardiomyopathy (ARVC)

Key Point

A limitation in the functional classification of cardiomyopathies based on diastolic function, systolic function, and left ventricular hypertrophy is that there may be considerable overlap encountered between categories and the potential to shift between categories based on their subsequent echocardiographic appearance. Therefore although the cardiomyopathies are presented as distinct categories in this text, there may be overlap and changes in functional abnormalities over time. Although there are specific criteria used to diagnose restrictive diastolic-filling physiologic processes seen in some cardiomyopathies, there is not a clear clinical categorization or differentiation of RCM from NCM in cats, and the two categories are often merged together.

Staging of feline cardiomyopathies using the ACVIM heart disease classification system (ABCD) has been suggested, which may aid in risk assessment and management recommendations. Cats at risk for cardiomyopathy, with genetic predisposition for HCM, are a stage A. Cats with structural cardiac abnormality without clinical signs are a stage B, which is further divided into mild disease stage B1 and moderate-to-severe disease with moderate to severe atrial dilation stage B2. Cats with active or compensated congestive heart failure are a stage C, and cats with refractory heart failure are a stage D.

Secondary Cardiomyopathies

- Nutritional (taurine deficiency-induced DCM)
- Metabolic (hyperthyroidism, acromegaly)
- Infiltrative (neoplasia, amyloidosis)
- Inflammatory (toxins, immune reactions, infectious agents)
- Toxic (doxorubicin, heavy metals)

CLINICAL CLASSIFICATION AND PATHOPHYSIOLOGY

- Most feline cardiomyopathies (HCM, RCM, and NCM) are caused by abnormalities of diastolic function or the ability of the heart to actively relax and passively fill with blood. The cause of the diastolic function in these cardiomyopathies may differ, but the clinical effect of severe diastolic dysfunction is the same: elevated left ventricular pressure, variable elevation of right ventricular filling pressures (NCM and +/– RCM), and development of congestive heart failure (CHF) (see Table 8.1).
- Systolic myocardial failure is seen in DCM, ARVC, or possibly nonspecific cardiomyopathy. DCM is caused by a primary contractile abnormality of the ventricle whereby it cannot contract and eject blood out of the heart, leading to decreased stroke volume and cardiac output. This is seen on an echocardiogram as an increased left ventricular systolic dimension and decreased fractional shortening (FS) (see Table 8.1). Right ventricular myocardial failure also can be seen in these cardiomyopathies. Systolic myocardial failure also increases diastolic-filling pressure because excess blood remains in the

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ventricle after systole and because of expansion of the blood volume by renin-angiotensin-aldosterone system (RAAS) activation.

- Left-sided CHF (i.e., backward failure) is manifested by elevated left-sided heart filling pressure and increased pulmonary capillary wedge pressure >20 to 25 mm Hg, leading to the development of pulmonary edema and pleural effusion in cats. Right-sided CHF is caused by elevated right-sided heart filling pressures (>10 to 15 mm Hg), leads to systemic venous hypertension, and is manifested by pleural effusion, jugular venous distension, and often ascites in cats. Peripheral edema is not a typical characteristic of right-sided heart failure in cats. A combination of left-sided and right-sided heart failure is common in cats with biatrial dilation caused by various cardiomyopathies (DCM, NCM, and RCM).
- Low-output heart failure (i.e., forward failure) is caused by marked decrease in cardiac output and systemic arterial hypotension and is manifested by cold peripheral extremities, weak pulses, tachycardia, pale mucous membranes, weakness, or collapse. Low-output heart failure is typically seen in end-stage heart disease.
- High-output heart failure in cats is most commonly associated with thyrotoxicosis causing excessive blood flow through the capillary beds and is associated with tachycardia, a gallop heart sound, bounding pulses, and CHF (pulmonary edema or pleural effusion).
- Arterial thromboembolism (ATE) may occur in cats with cardiomyopathy and left atrial dilation of various severities. Left atrial and left auricular blood flow stasis can cause platelet aggregation and formation of a thrombus. Other potential contributing causes of ATE in cats with cardiomyopathy include left atrial/auricular endothelial cell damage, and/or a hypercoagulable state caused by increased platelet activation or increased blood coagulation proteins. An overwhelming majority of cats suffering from ATE have a cardiogenic origin.
- Heart disease is not synonymous with heart failure (congestive or low output). Many cats with heart disease, especially HCM, never have heart failure. Heart failure is the end result of severe heart disease with many causes. Often cats with DCM, RCM, or NCM miss early detection because a murmur may be absent on auscultation and are often only diagnosed at an end stage once severe symptoms of heart failure are present.

SIGNALMENT AND PRESENTING COMPLAINTS

Key Point

The clinical presentation, physical examination findings, radiographic findings, and electrocardiographic (ECG) findings are similar for all forms of myocardial disease and generally cannot be used to differentiate among them. Echocardiography is necessary to determine the specific disorder that is present.

- Presenting complaints in cats with cardiomyopathies are often referable to the presence of CHF or other sequelae such as ATE or, rarely, severe arrhythmias that causes syncope or collapse. Cardiomyopathies may be mild to severe, with a wide spectrum of potential clinical presentations ranging from asymptomatic to severely affected cats. The typical clinical presentation, physical examination findings, radiographic findings, and ECG findings are discussed in this section, but echocardiographic findings are discussed within the specific cardiomyopathy category.
- Common presenting complaints in cats with severe cardiomyopathy may include the following:
  - Dyspnea/tachypnea
  - Weight loss, weakness, lethargy
  - Anorexia
  - Acute paresis or paralysis of pelvic limbs or a thoracic limb consistent with ATE
- Uncommon complaints may include syncope, collapse, coughing, or abdominal distension.
- A precipitating event is common in cats that have heart failure. Recent fluid administration, anesthesia or surgery, or repositol corticosteroid administrations are among the most common antecedent events.
- Cats may be symptom free for cardiomyopathy and have no presenting complaint referable to heart disease but may be examined for other systemic disease or for a wellness examination.

PHYSICAL EXAMINATION

- Early detection of disease should be a primary goal, before signs of fulminant heart failure or ATE occur. A thorough physical examination, with careful attention to auscultation, should be performed.

Cardiac auscultation abnormalities in cats with cardiomyopathy commonly include the following:

- Systolic murmur (commonly heard along the left sternal border). Murmur intensity does not relate to severity of underlying disease. This murmur is often caused by mitral regurgitation or left ventricular outflow tract obstruction. Presence of a murmur does not definitively diagnose the cat with cardiomyopathy, but raises suspicion of underlying cardiomyopathy.
- Gallop sound: an additional heart sound heard in diastole either during early diastolic filling (S_3) or during atrial systole (S_4). Given the rapid heart rates (HRs) in cats, differentiation of S_3 and S_4 heart sounds is impossible and unnecessary. Presence of a gallop is abnormal and may occur when there is diastolic or systolic dysfunction or volume overload to the ventricle. It is more commonly auscultated in cats with heart failure than in symptom-free cats. Aside from cardiomyopathy, other important causes of gallop heart sounds include: anemia and hyperthyroidism.
- Arrhythmia: premature beats, runs of tachycardia, or irregularly irregular tachycardia (i.e., atrial fibrillation) may be present. Bradycardia (HR <150 beats/min)

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may be present if there is hyperkalemia associated with reperfusion syndrome in cats with ATE or in cats with third-degree atrioventricular (AV) block that have underlying cardiomyopathy.

Clinical signs often seen in cats with heart failure include the following:

- Respiratory abnormalities
  - Tachypnea, dyspnea, orthopnea
  - Muffled or harsh lung sounds
- Hypothermia
- Jugular pulses/distention: uncommon but present in cats with right-sided heart failure

Clinical signs associated with ATE

- Acute paresis of pelvic limbs or thoracic limb associated with pain
- Cold peripheral extremity
- Absent femoral arterial pulse of the affected pelvic limb
- Cyanotic toe pads
- Firm painful muscles of affected limb
- Neuropathy, typically with absent conscious proprioceptions

ANCILLARY TESTS

- Thoracic radiography and electrocardiography may direct or reinforce suspicion that a cardiac disorder is present. They may also further characterize the disorder and alert the clinician to secondary ramifications that may require attention; however, neither electrocardiography nor thoracic radiography provides adequate evidence for ruling out, confirming, or classifying feline cardiac disease. Echocardiography is required to confirm or rule out and categorize myocardial disease.
- Electrocardiography is indicated for evaluation of an arrhythmia identified on auscultation, or for evaluation of cats with episodic weakness, syncope, or collapse. However, an electrocardiogram should not be used as a method to screen for underlying cardiomyopathy.
- ECG abnormalities seen in cats with cardiomyopathy
  - Ventricular arrhythmias: ventricular premature complexes (VPCs) are common but typically mild and often do not warrant antiarrhythmic therapy. Ventricular tachycardia or complex ventricular arrhythmias may be seen in cats with cardiomyopathy and warrant antiarrhythmic therapy.
  - Atrial tachyarrhythmias: atrial premature complexes are less common than VPCs and do not warrant treatment. Supraventricular tachycardia with high sustained rates >300 beats/min may be seen in cats with cardiomyopathy and warrants treatment. Atrial fibrillation may be present in cats with severe cardiomegaly and left atrial dilation and warrants negative chronotropic therapy.
  - Left-axis deviation or a left anterior fascicular block pattern may be present in some cats with HCM, although either may also be present in some normal cats or hyperthyroid cats.
- Increased amplitude of R waves may be present in cats with left ventricular enlargement or concentric hypertrophy and is insensitive for diagnosis of cardiomyopathy.
- Thoracic radiography is most useful for detecting gross cardiac enlargement and CHF (e.g., pulmonary venous distension, pulmonary edema, pleural effusion, or dilated caudal vena cava) (Figs. 8.1 and 8.2). Restraint for radiographic procedures can be life threatening to dyspneic cats. Extreme caution should be taken before proceeding with radiography. Severely dyspneic cats may require emergency stabilization before radiographs are obtained.
- While the cat is in sternal recumbency, a brief "triage" thoracic ultrasound is a less stressful initial diagnostic tool than radiography for detection of severe pleural effusion and also directs the optimal location for thoracocentesis. Pulmonary ultrasound scanning (Vet BLUE method) for presence of B lines may increase suspicion of cardiogenic pulmonary edema in the unstable patient and be safer than radiographs.
- Systemic blood pressure measurement is also a necessary test to evaluate for systemic hypertension or hypotension and helps direct acute and long-term medical therapy. Systemic hypertension can create mild left ventricular concentric hypertrophy, which may mimic HCM on echocardiography.

Key Points

- A thoracic radiograph with normal findings does not preclude the diagnosis of a cardiomyopathy. Many symptom-free cats with mild changes and normal LA size will have a thoracic radiograph with normal findings.
- Echocardiography, including Doppler echocardiography, is essential for noninvasive determination of a functional and anatomic diagnosis. Before a diagnosis of cardiomyopathy is assigned solely on the basis of morphologic/functional appearance, a concerted effort should be made to rule out cardiac and extracardiac diseases that might mimic the echocardiographic appearance of primary myocardial diseases.
- Other diagnostic tests do not usually contribute to the diagnosis of myocardial disease but are important for determining the overall status of the patient, identifying concomitant disorders, and assessing the efficacy or untoward effects of therapy. When possible, routine biochemistries, urinalysis, thyroxine level, and a complete blood count should be performed before pharmacologic intervention to establish baseline values for the patient and to rule out concurrent, secondary metabolic, or hematologic disturbances.
- Pleural effusion fluid analysis, including chemical and cytologic evaluation, can help determine whether heart failure may be causing pleural effusion accumulation. Cardiogenic pleural effusion may be characterized as a transudate, modified transudate, pseudochylous effusion, or true chylous effusion but is not a hemorrhagic effusion or an exudate.

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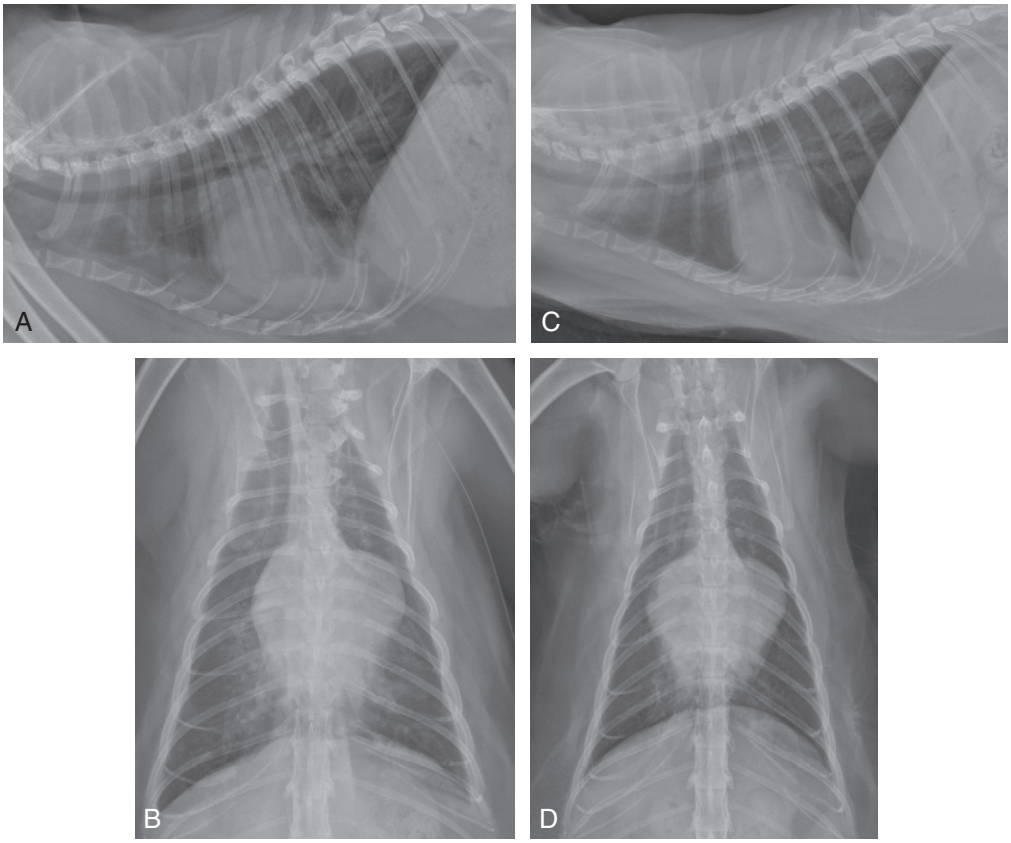


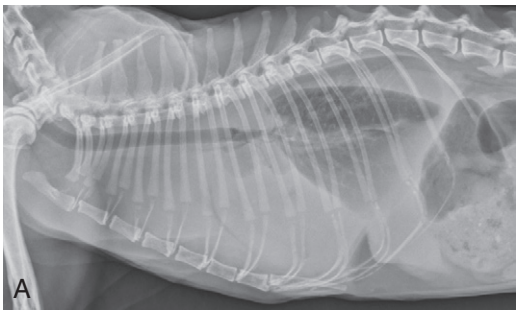
Fig. 8.1 Thoracic radiographs of a cat with severe hypertrophic cardiomyopathy and left heart failure. There is severe cardiomegaly and left atrial dilation seen on lateral **(A)** and ventrodorsal (VD) **(B)** views. The lateral view (A) depicts patchy interstitial infiltrates, especially of the caudal lung lobes and pulmonary venous distension, which are consistent with left-sided heart failure. Recheck radiographs **(C** and **D)** post furosemide therapy show resolution of the pulmonary edema and pulmonary venous distension but persistent cardiomegaly.

- Plasma and whole blood taurine concentrations should be measured in all cats with echocardiographically documented myocardial failure (see the section on taurine deficiency-induced myocardial failure).
- N-terminal pro-B-type natriuretic peptide (NT-proBNP) is a sensitive and accurate blood screening test for detection of heart failure in cats with cardiomyopathy and may be used to help distinguish whether the dyspnea is caused by heart failure or primary respiratory disease. Currently a quantitative ELISA (enzyme-linked immunosorbent assay) and point-of-care assay SNAP proBNP are commercially available (see Chapter 6). In a large-scale study of 425 cats diagnosed with mild-to-end-stage cardiomyopathy, NT-proBNP correlated with stage of cardiac disease severity and survival time, and levels >270 pmol/L were suggestive of clinically significant disease. There are variable results in studies in which symptom-free cats were evaluated. The NT-proBNP test does not replace the need for further diagnostic testing with an echocardiogram or radiographs but helps assess the likelihood of underlying cardiac disease.

THERAPY

- Therapy should be based on the clinical and functional classification of the disease process in the individual patient and not by following a standard approach based solely on the diagnosis.
- With only two exceptions, the indications for, and benefits of, therapeutic intervention in symptom-free cats with myocardial disease are controversial. These exceptions are the following:
  - Myocardial failure secondary to taurine deficiency
  - Thyrotoxic heart disease
- Dyspneic cats are easily stressed and may acutely deteriorate and die if stressful diagnostic or therapeutic interventions are initiated too early. Sedation with butorphanol can be given to lessen stress and aid in further diagnostic assessment. A triage-level thoracic ultrasound should be done if available in the severely dyspneic cat to evaluate for severe pleural effusion. If an ultrasound is unavailable and the cat has life-threatening dyspnea and is too unstable to safely obtain radiographs, thoracocentesis may be attempted at the right caudal thorax. The severely dyspneic cat can then receive oxygen and be given a parenteral dose of furosemide to stabilize before radiographs. If blood collection is possible, the bedside SNAP proBNP test can be run to place heart failure as likely (positive) or unlikely (negative). If physical abnormalities and history are suggestive of lower airway disease (asthma), the cat may be given steroids and bronchodilators parenterally and receive oxygen.

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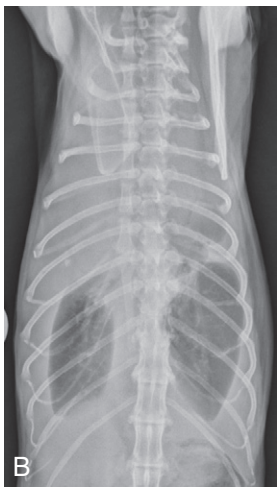


Fig. 8.2 **(A** and **B)** Thoracic radiographs from a cat with severe dilated cardiomyopathy (DCM) and biventricular heart failure. Severe pleural effusion is present in this cat with severe DCM and biventricular heart failure, which obscures visualization of the heart, pulmonary vasculature, and pulmonary parenchyma. Repeat thoracic radiographs after palliative thoracocentesis may allow visualization of the intrathoracic structures. An echocardiogram is needed to definitively diagnosis the heart failure causing the pleural effusion and to determine the origin of the cardiac disease.

- All patients with evidence of significant and life-threatening CHF (pulmonary edema, pleural effusion) require immediate therapy (i.e., appropriate combinations of thoracocentesis, parenteral furosemide, and oxygen). Cats with significant pleural effusion will benefit most from immediate thoracocentesis.
  - **Furosemide** is the diuretic of choice in cats. Furosemide should be administered intravenously (2–4 mg/kg q12-4h then rapidly tapered to 1–2 mg/kg q6-8h on the basis of resting respiratory rate and effort) or intramuscularly (2–4 mg/kg q2h until an intravenous catheter can be placed) depending on the stress level of the cat. Dosing must be dramatically reduced once the respiratory rate begins to decrease. In general, aggressive diuretic therapy is continued until the respiratory rate is below 40 breaths per minute.
  - Enriched fraction of inspired oxygen (FiO_2 50%) can be administered with an oxygen cage, which controls temperature and humidity and removes excess carbon dioxide.
  - Transdermal nitroglycerin (⅛- to ¼-in. QID ≤ 2 days) is a venodilator whose efficacy is unknown in cats. It may lessen pulmonary venous hypertension, thereby decreasing the formation of pulmonary edema.
  - Sedation with a low-dose opioid such as butorphanol or buprenorphine may be administered to calm distressed patients.
- Chronic heart failure therapy
- Maintenance therapy is generally aimed at minimizing clinical signs (e.g., dyspnea, tachypnea) and preventing an acute respiratory crisis. Oral furosemide may be uptitrated according to the severity of heart failure. The oral doses range from 1 to 3 mg/kg q24h BID, and may uptitrate to TID in refractory heart failure to a maximum in cats that can tolerate high furosemide doses, of 4 mg/kg TID for severe refractory heart failure. Often a typical oral dose of furosemide is 6.25 mg BID. Recheck radiographs are helpful to assess the presence and severity of pulmonary edema or pleural effusion after heart failure treatment and aid in furosemide dose titration (see Fig. 8.1C and D).
- Torsemide, a potent loop diuretic, may be used to treat progressive heart failure or in cats where frequent medication administration of BID to TID is not possible.
- Angiotensin-converting enzyme (ACE) inhibitors (enalapril 0.5 mg/kg PO q12–24h, ramipril 0.5 mg/kg PO q24h, and benazepril 0.5 mg/kg PO q12–24h) may provide adjunctive benefit for treatment of chronic heart failure.
- Antiarrhythmic drugs may be indicated to control significant arrhythmias such as atrial fibrillation, ventricular tachycardia, or supraventricular tachycardia.
- Positive inotropic agents such as pimobendan (0.25 mg/kg PO BID or 1.25 mg per cat PO BID) may be indicated in patients with myocardial failure identified by echocardiography or low-output heart failure (i.e., hypotension and signs of poor perfusion). It can be considered in RCM or NCM, and may be considered in heart failure cats with progressive nonobstructive HCM.
- All cats with myocardial failure identified by echocardiography should be supplemented with **taurine** (250 mg PO BID; see the section on taurine deficiency-induced myocardial failure) until proven to have a normal taurine blood concentration or a lack of response after several months of supplementation on the basis of a recheck echocardiogram.

Arterial Thromboembolism Therapy

- Prophylactic therapy may be instituted in cats with a high risk for having ATE, including those cats that have recovered from an ATE event, cats that have spontaneous echocardiographic contrast or

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thrombus in the atrium on echocardiography, or cats that have moderate or severe atrial dilation.

- Clopidogrel (18.75 mg PO q24h) is a potent platelet receptor antagonist that is currently favored for prophylaxis of ATE and was superior to aspirin in the FATCAT (Feline Arterial Thromboembolism: Clopidogrel vs. Aspirin Trial) clinical study for time to development of recurrent ATE in cats recovering from ATE.
- **Aspirin** (5–81 mg PO every 3 days) may be used for ATE prophylaxis, although it appears inferior to clopidogrel in cats that had ATE in the FATCAT clinical study. Recurrence of thromboembolic events in aspirin-treated cats were as high as 75% in another study. Aspirin may be added to clopidogrel in cats with refractory spontaneous contrast or thrombus seen on an echocardiogram.
- **Other anticoagulants:** Rivaroxaban (factor Xa inhibitor; 0.5–1 mg/kg/day or 2.5 mg/CAT PO q24h) or Enoxaparin (low-molecular-weight heparin; 1.5 mg/kg SC BID-TID), have shown promise in anecdotal clinical settings. Rivaroxaban was recently evaluated in a clinical trial of ATE cats and was not superior to clopidogrel in that study; however, combination therapy of clopidogrel and rivaroxaban or clopidogrel and enoxaparin may be considered in cats with refractory spontaneous contrast, refractory intracardiac thrombus, or in cats suffering from ATE since this approach combines antiplatelet- and anticoagulant-directed therapies.

Acute Therapy for Arterial Thromboembolism

- Left untreated, the outcome of arterial occlusion will depend on the extent of occlusion and time to spontaneous reperfusion, either through the primary vessel or the collateral circulation. Cats may lose the affected leg(s) because of ischemic necrosis, die of reperfusion syndrome or toxemia, remain paralyzed from peripheral nerve damage, or regain full or partial function of the leg. Overall response to presently available conservative or aggressive clinical intervention has been poor. Survival of cats receiving treatment ranges from 45% to 73%. Positive predictors for better survival include a single limb affected and presence of motor function at presentation. Negative predictors for survival include refractory heart failure, malignant arrhythmias, multiorgan embolization, presence of an intracardiac thrombus, progressive azotemia, disseminated intravascular coagulation, and unresponsive hypothermia. In cats recovering from ATE, the most common cause of death is CHF, followed by recurrent ATE.
- Invasive therapeutic options such as surgical removal of emboli, rheolytic embolectomy, or catheter embolectomy are not currently recommended because of similar survival rates to medical therapy.
- Medical therapies—most are untested and unproven
  - Anticoagulation with **heparin** (220 U/kg IV followed 3 hours later by a maintenance dose of 66–200 U/kg SQ four times a day) is used to prevent further thrombosis. Adjust the dose to maintain the activated partial thromboplastin time at or slightly above the upper limit of the normal reference range. **Enoxaparin** (1–1.5 mg/kg SC TID q6h) is another option that does not require coagulation panel monitoring because it affects factor X activity.
  - Thrombolytic therapy (e.g., tissue plasminogen activator, streptokinase, or urokinase) is rarely recommended because of expense of the drug, life-threatening complications, and lack of effective prevention of recurrent ATE.
  - Pain management: Analgesia is crucial for cats with ATE, particularly in the first 2 days after the ATE. Various opioids may be chosen, including fentanyl (2–5 µg/kg/h constant rate infusion [CRI] for 12–18 hours until fentanyl patch takes effect) or buprenorphine (0.005–0.015 mg/kg IV q6h).
  - Vasodilators such as acepromazine and hydralazine may not increase arterial blood flow to the affected limb but may lead to systemic hypotension and have therefore fallen out of clinical favor.
  - Clopidogrel (full 75-mg tablet orally once or 18.75 mg standard dose, then 18.75 mg PO q24h) should be given as soon as oral medication is feasible, since it lessens platelet release of vasoactive amines and proaggregation substances, which may aid in collateral circulation.
- Reperfusion syndrome: Cats may have acute reperfusion syndrome characterized by life-threatening hyperkalemia and acidosis caused by spontaneous autogenous or exogenous thrombus lysis. This may occur any time over the first several days to 1 week after ATE and must be treated aggressively and immediately. Continuous ECG monitoring may be useful to detect acute onset of bradycardia that occurs with life-threatening hyperkalemia. Electrolytes and pH should be measured at least daily to twice daily in hospitalized cats.
- Acute treatment
  - 10% calcium gluconate: 0.5 to 1.5 mL/kg IV slowly over 5 to 10 minutes
  - Sodium bicarbonate: 1 to 2 mEq/kg IV slowly over 15 minutes
  - 25% dextrose: 0.7 to 1 g/kg IV over 3 to 5 minutes
  - 25% dextrose with insulin: regular insulin 0.5 U/kg IV with dextrose 2 g/U of insulin administered
  - Atropine: 0.02 to 0.04 mg/kg IV if bradycardia present

PROGNOSIS

- Within the broad category of feline cardiomyopathy there is a wide range of diseases and severity, which makes it impossible to predict accurate prognosis.

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Although subclinical cats may live many years with cardiomyopathy, once heart failure occurs, prognosis is typically limited to months. Cats with ATE have the gravest prognosis, with a median survival time (MST) ranging from 4 to 8.4 months, with shortest survival in the presence of heart failure (MST 77 days).
- Prognosis for each form of cardiomyopathy is discussed in that respective section.

PATHOLOGY

- Gross examination of the heart provides useful anatomic information and should be performed when possible to confirm antemortem findings.
- Cats with severe cardiomyopathy often have gross evidence of left atrial enlargement and CHF. Edematous lungs are heavy and red and sink when placed in water or formalin. Pulmonary edema may be identified as a frothy fluid exuding from the cut surface of the lung or filling the trachea or smaller airways. Pleural effusion may be readily identified on opening the thorax.
- Increased heart weight or heart weight indexed to body weight (29–37 g [HCM] compared with <20 g [normal]; ≥6.4 ± 0.1 g heart weight per kilogram of body weight [HCM] compared with ≤4.8 ± 0.1 g/kg [normal]) is often seen in cats that die from HCM and may also be present in cats with enlarged hearts from other cardiomyopathies.
- In most cases, histopathologic evaluation adds little useful information, and unless readily available at low cost, it is not recommended unless specific indications are present (see the section on specific diseases). Hallmark histopathologic abnormalities of HCM include cardiomyocyte hypertrophy, myofiber disarray (in 30% of cases in one study, involving at least 5% of the septum), interstitial and replacement fibrosis, and small coronary arteriosclerosis.

PRIMARY CARDIOMYOPATHIES

HYPERTROPHIC CARDIOMYOPATHY

HCM is a disease of the ventricular (primarily left ventricular) myocardium characterized by mild-to-severe thickening (concentric hypertrophy) of the papillary muscles, interventricular septum, and LV free wall. The word *primary* in this context means that the hypertrophy results from an inherent problem in the myocardium and is not secondary to a pressure overload (systemic hypertension or aortic stenosis) or to hormonal stimulation (T4). Transient myocardial thickening is another cause of HCM phenotype that may reverse over several months, and is another differential for secondary causes of LV hypertrophy.

General Comments

- When any other disease process that may lead to concentric hypertrophy is present, the diagnosis of HCM is excluded. Secondary disorders typically produce symmetric concentric hypertrophy, usually only with a maximum increase in wall thickness of <50%, even with severe disease. If severe or asymmetric concentric hypertrophy is present in a patient with one of these disorders, then concomitant HCM should be considered. Likewise if heart failure is present, HCM should be suspected because secondary causes of left ventricular concentric hypertrophy do not usually independently cause heart failure. If left ventricular concentric hypertrophy does not regress after several months of effectively treating the underlying problem, such as hypertension or hyperthyroidism, HCM should also be suspected as being a concurrent disease.
- HCM is the most commonly diagnosed cardiac disease in cats, and its prevalence appears to be increasing (ranging from 15% to 30% of healthy cats); however, echocardiographic screening for the disease has also become more common during this time.
- The cause of HCM is idiopathic, yet heritable familial HCM is identified in several predilected breeds, including Maine Coon, Ragdoll, Persian, British Shorthair, American Shorthair, Norwegian Forest, Turkish Van, Sphynx, Bengal, and Scottish Fold cats.
- Genetic mutations causing HCM have been identified in Maine Coon, Ragdoll, and Sphynx cats. Two different missense mutations in cardiac myosin-binding protein C gene *(MYBPC3)* have been identified to cause HCM in some families of Maine Coon and Ragdoll cats, and a commercial assay is available for genetic screening (through the Cardiac Genetics Laboratory at the North Carolina State College of Veterinary Medicine). Heterozygous affected cats may not have phenotypic evidence of disease (i.e., no concentric ventricular hypertrophy on echocardiography) but may transmit the defect to progeny or develop HCM later in life. Homozygous affected cats often develop severe disease earlier in life. A mutation in the ALMS1 gene has been identified to cause HCM in Sphynx cats. Sixty percent of affected Sphynx cats have that mutation, meaning that other unidentified mutations are likely present. The ALMS1 gene regulates cell cycle proliferation in perinatal cardiomyocytes.

Clinical Classification and Pathophysiology

- HCM is characterized by increased left ventricular myocardial wall thickness in the absence of other secondary causes. Papillary muscle hypertrophy is subjectively assessed on echocardiogram and may range from mild to severe. Concentric hypertrophy may include the interventricular septum and the left ventricular free wall and may be either global (symmetric) or segmental (asymmetric). Hypertrophy may range from mild (6–6.5 mm) to severe (>7.5 mm). The concentric hypertrophy may also result in a decrease in afterload because of the

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increase in wall thickness, which may result in a decreased end-systolic volume, often to zero (end-systolic cavity obliteration).

- Impaired diastolic filling during early active relaxation or during passive ventricular filling caused by increased ventricular stiffness leads to diastolic dysfunction. Diastolic dysfunction in cats with HCM is caused by increased wall stiffness because of hypertrophy, myocyte disarray, myocardial fibrosis, and possible impaired calcium handling, causing impaired active diastolic relaxation. Reduced myocardial perfusion may lead to myocardial ischemia and myocardial necrosis, with subsequent replacement fibrosis of the myocardium. Small vessel coronary artery disease and activated neurohormonal systems such as RAAS may lead to interstitial myocardial fibrosis. Myocardial fibrosis may also cause ventricular arrhythmias and potentially sudden death.
- Systolic anterior motion (SAM) of the mitral valve is often (67% of cats with HCM in one study) but not always present in cats with HCM and may range from mild to severe. Cats with HCM and SAM are commonly said to have the obstructive type of HCM or to have hypertrophic obstructive cardiomyopathy (HOCM). It is typically the cause of the left parasternal systolic murmur in HCM. SAM of the mitral valve develops secondary to anterior ventrally displaced, hypertrophied papillary muscles that pull the mitral valve into the left ventricular outflow tract during systole. Other factors that may exacerbate or worsen SAM of the mitral valve include severe basilar septal concentric hypertrophy, increased contractility, and tachycardia.
- SAM of the mitral valve produces a dynamic subaortic stenosis that increases systolic intraventricular pressure in mid to late systole and is worsened with increased contractility. The dynamic subaortic stenosis increases the velocity of blood flow through the subaortic region and produces turbulence in the left ventricular outflow tract and aorta. Simultaneously, when the septal leaflet is pulled toward the interventricular septum, a gap is left in the mitral valve creating mitral regurgitation, which is typically only mild to moderate in severity. Moderate or severe SAM of the mitral valve greatly increases left ventricular systolic pressure, which increases the severity of concentric left ventricular hypertrophy and potentiates the vicious cycle of hypertrophy and the potential for worsened diastolic function.
- Diastolic heart failure is caused by severe diastolic dysfunction in cats with severe HCM. Severe diastolic dysfunction leads to elevated left ventricular filling pressure and left atrial dilation, ultimately leading to left-sided CHF when the pulmonary venous pressure exceeds 20 to 25 mm Hg. Approximately half of cats with CHF have concurrent pulmonary edema and pleural effusion. Pleural effusion is common in cats with heart failure. It can be classified as a modified transudate, pseudochylous, or true chylous effusion. The exact pathophysiologic processes of pleural effusion secondary to left heart failure is unknown in cats. The most likely possibility is that feline visceral pleural veins drain into the pulmonary veins such that elevated pulmonary venous pressure causes the formation of pleural effusion.
- Sudden death may occur in any cat and may be unrelated to disease severity. The incidence of sudden death in cats with HCM is unknown. Sudden death may be caused by severe ventricular arrhythmia that causes fibrillation or from massive ATE that occludes the proximal aorta.
- Left atrial or auricular thrombi may form in cats with HCM, typically when the left atrium (LA) is dilated. Left atrial thrombi commonly break loose (become emboli) and are carried by blood flow most commonly to the terminal aorta where they lodge. These arterial thromboemboli occlude aortic blood flow and elaborate vasoactive substances that constrict collateral vessels. The net result is cessation of blood flow to the caudal legs resulting in acute paresis/paralysis and pain. The incidence of ATE in cats with HCM is approximately 12% to 17% and is associated with varying degrees of left atrial enlargement. Thrombus formation may occur when there is an abnormality in one or more of the components of a Virchow triangle, which include hypercoagulability, endothelial disruption, and blood stasis. When the LA becomes moderately to severely dilated, the blood flow velocity is reduced, resulting in red cell aggregation, platelet activation, and subsequent thrombus formation. Blood flow stasis may be the most important factor for development of left atrial thrombi in cats with cardiac disease. Hypercoagulability has also been reported in cats with cardiomyopathy or ATE.

Signalment and Presenting Complaints

- There is a wide range of patient characteristics in feline HCM, with any age being possible for HCM to develop. Although the average age of cats diagnosed with HCM is reportedly 5.5 to 6.5 years, cats may range from several months of age to geriatric. With increased awareness of the disease and more frequent echocardiographic screening of predisposed breeds, cats are being diagnosed earlier in the disease course, often as young juveniles.
- Familial autosomal dominant heritable HCM occurs in Maine Coon and Ragdoll cats as a result of mutations in *MYBPC3* (see Chapter 6). In a family of Maine Coon cats with familial HCM caused by *MYBPC3* mutation, homozygous affected cats had phenotypic echocardiographic evidence of left ventricular hypertrophy between 4 and 6 months

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of age and severe left ventricular hypertrophy by 7 to 12 months of age. Heart failure usually developed by 20 months of age, and most of these cats died by 4 years of age. Male Maine Coon cats that are homozygous affected typically have more severe disease at an earlier age. Heterozygously affected Maine Coon cats may live many years with moderate left ventricular hypertrophy and atrial dilation without development of CHF or sudden death. Ragdoll cats can have a severe, early onset form of familial HCM, with a mean age of diagnosis of 15 months in one study (ages range from 6 months to 2 years). In a genetic study, Ragdoll cats that were homozygously affected with the *MYBPC3* mutation had HCM 18 months earlier than heterozygously affected cats.

- Other predisposed breeds include Persian, Himalayan, Birman, American Shorthair, British Shorthair, Sphynx, Bengal, Siberian, Norwegian Forest, and Scottish Fold cats. Despite certain breed predispositions, the domestic shorthair cat is the most commonly affected breed. The average age of onset and rate of progression in mixed breed cats is unknown.
- Although there is no sex-linked heritability pattern, approximately three-fourths of cats diagnosed with HCM are neutered males.
- Presenting complaints are variable, depending on the severity of HCM. Because cats are often symptom free, they may be seen by the veterinarian for other problems or for a routine health examination or preanesthetic evaluation. Other cats with severe HCM may have a complaint referable to heart failure, including difficulty breathing, anorexia, lethargy, or weight loss. A precipitating event was identified in approximately half of cats diagnosed with CHF in one study. The most common precipitating event was fluid administration (28%), followed by anesthesia/surgery (25%), and recent corticosteroid administration (21%) with methylprednisolone (70% of cats) or an injectable form of triamcinolone (30% of cats) given 1 to 2 weeks before diagnosis of heart failure.
- Sometimes the first presenting complaint is sudden death or acute paralysis caused by ATE.

Physical Examination

- Auscultation abnormalities: A systolic murmur is the most common physical examination abnormality in cats with HCM and has been reported in 31% to 72% of cats. Murmur grades range from soft (1) to loud (4), often depending on the presence and severity of SAM of the mitral valve. A gallop is less frequently auscultated (33%), and an arrhythmia is uncommon (7%). A small percentage (7%–20%) of cats have no abnormalities on auscultation. Please refer to the previous general section of cardiomyopathies for a complete discussion of physical examination abnormalities.

Echocardiography

- The defining abnormality seen on echocardiography in cats with HCM is an increased end-diastolic left ventricular wall thickness of 6 mm or greater, which may include the interventricular septum or the left ventricular free wall in the absence of other secondary causes of concentric hypertrophy. Papillary muscle hypertrophy is commonly present and is subjectively assessed (Fig. 8.3A). Sometimes papillary muscle hypertrophy is the only abnormality present. Cats may have generalized (global) concentric hypertrophy or asymmetric (segmental) concentric hypertrophy of the septum or free wall (Fig. 8.3A and C). Because of these various patterns of hypertrophy, HCM is a diagnosis that should be made by examining several two-dimensional echocardiographic views and measuring wall thicknesses in diastole from the thickest region or regions on the two-dimensional images. M-mode echocardiography may miss regional thickening unless it is guided by the two-dimensional view. Often the basilar interventricular septal hypertrophy is best assessed with the right parasternal long-axis view (Fig. 8.3C). The left ventricular end-diastolic or end-systolic dimension may be normal or decreased, and end-systolic cavity obliteration may occur. End-systolic cavity obliteration is seen as an obliterated end-systolic chamber lumen as a result of the marked increase in left ventricular hypertrophy and reduction in afterload (Fig. 8.3B).
- Left atrial dilation may be identified as a left atrial to aortic diameter ratio (measured at the first frame of aortic valve closure) with two-dimensional echo of >1.5 or left atrial diameter >16 mm. An enlarged LA indicates increased left ventricular end-diastolic pressure and risk of CHF. Typically left atrial dilation is moderate or severe (left atrial/aortic ratio >1.8) in cats with CHF. Occasionally a thrombus is identified in the LA or its appendage. Spontaneous red cell aggregation (i.e., smoke) should be carefully assessed within the LA and auricle in both right parasternal views and the left parasternal cranial view where the auricle is positioned close to the body wall.
- SAM of the mitral valve may be identified by two-dimensional echocardiography in the right parasternal long-axis left ventricular outflow tract view. Often the abnormality may be detected most easily by color flow Doppler interrogation of the left ventricular outflow tract and mitral valve. The characteristic color flow Doppler abnormality is a double jet of turbulence arising from a single point in the left ventricular outflow tract in mid to late systole, causing both mitral regurgitation and turbulent

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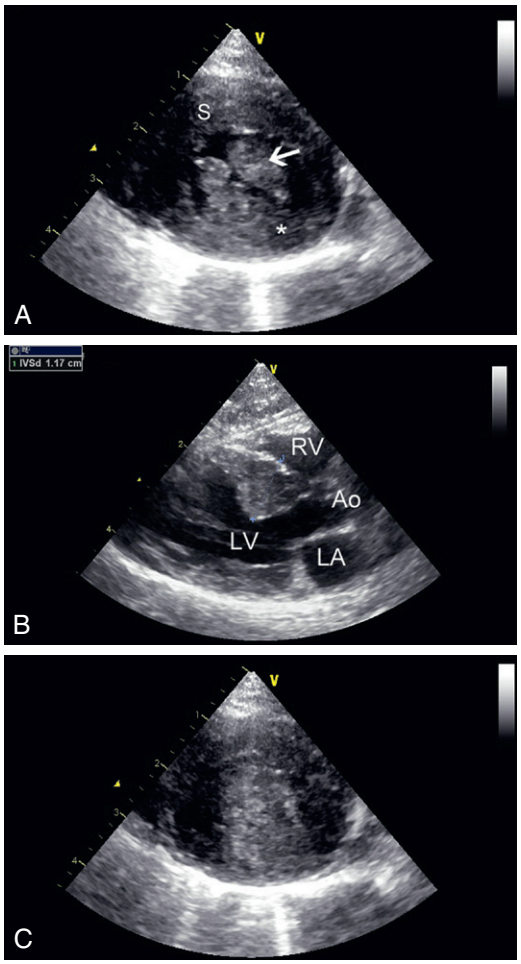


Fig. 8.3 Echocardiogram of the left ventricle (*LV*) of a cat with severe hypertrophic cardiomyopathy (HCM). HCM consists of concentric left ventricular hypertrophy (>6 mm) that may be global or asymmetric. Asymmetric hypertrophy may mainly include the papillary muscles (arrow) and left ventricular free wall **(A)** or the basilar interventricular septum **(B)**. There is often end-systolic cavity obliteration **(C)** where there is no chamber lumen remaining at end diastole because of marked concentric hypertrophy and reduced afterload. *Ao*, Aorta; *LA*, left atrium; *RV*, right ventricle.

blood flow in the left ventricular outflow tract out to the aorta (Fig. 8.4). With the left apical five-chamber view, continuous wave Doppler is used to measure the velocity of aortic blood flow and quantifies the severity of obstruction. The modified Bernoulli equation (pressure gradient = 4 × velocity^2) is used to calculate the LV to aortic pressure gradient and determine the severity of obstruction (mild 30–50 mm Hg, >50 mm Hg moderate to severe). The obstruction is dynamic, worsens through systole, and creates a characteristic shape of a "dagger" on continuous wave Doppler interrogation, with a peak velocity at end systole (see Fig. 8.4). The pressure gradient roughly correlates with the severity of the SAM, although it can be quite labile, changing with the cat's level of excitement. Provocation can be done to accentuate the dynamic obstruction.

- Diastolic dysfunction in cats with severe HCM has been documented with tissue Doppler imaging and measures of transmitral flow and relaxation time. Cats with severe HCM routinely have a decrease in early diastolic wall motion of the LV free wall and mitral valve annulus using tissue Doppler imaging. Pulsed wave (PW) Doppler measurement of mitral inflow velocity from the left apical four-chamber view often shows a delayed relaxation pattern (early diastolic-filling wave [E] to atrial systole [A] wave ratio of <1, prolonged isovolumic relaxation time, and prolonged deceleration time) in cats with HCM. Delayed relaxation is the first level of diastolic dysfunction when there is impaired early diastolic filling but normal left atrial pressures. As diastolic dysfunction worsens, left atrial pressure increases, leading to a pseudonormal filling pattern on mitral inflow velocity measurement. A restrictive filling pattern may be seen in cats with severe diastolic dysfunction and severely elevated left atrial pressure and is evidenced by E/A ratio >2, decreased deceleration time, and shortened isovolumic relaxation time. Assessment of mitral inflow patterns of diastolic function is fraught with challenges such as summation of E and A waves in cats with HRs >150 beats/min and is affected by changes in preload and afterload.

Additional Testing

- Assessment of secondary (reversible) causes of concentric ventricular hypertrophy such as systemic hypertension or hyperthyroidism is necessary in cats with echocardiographic evidence of left ventricular concentric hypertrophy. Systemic blood pressure should be measured in all cats with left ventricular concentric hypertrophy, and the serum thyroxine level measured in cats >6 years of age with left ventricular concentric hypertrophy.
- Genetic testing for a *MYBPC3* mutation may be considered in Maine Coon and Ragdoll cats, or the ALMS1 mutation in Sphynx cats, to assess the risk of developing HCM or if they are intended for breeding. There is a high prevalence, 34%, of genetically affected symptom-free Maine Coon cats in the general breeding population. A test with negative results does not rule out HCM because cats may have HCM from other causes or unidentified genetic mutations. Cats with positive test results should be evaluated by echocardiography to assess whether there is phenotypic evidence of disease, and echocardiography should be repeated annually in genotypically affected cats. Homozygous affected cats should not be used for breeding.

Therapy

- There is a lack of studies in which the impact of treatment on the clinical course of disease in symptom-free cats with HCM is evaluated. The clinician

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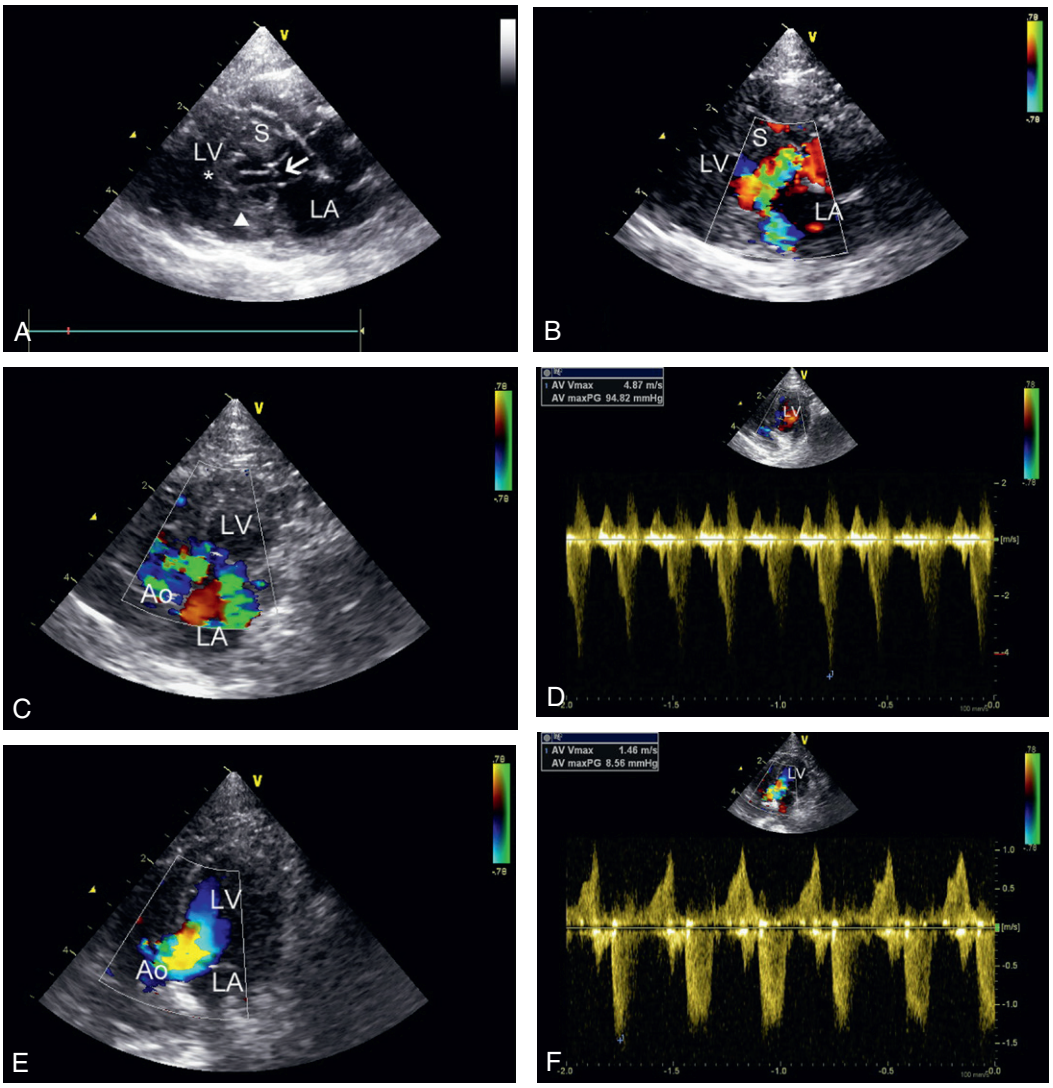


Fig. 8.4 Echocardiographic assessment of systolic anterior motion (SAM) of the mitral valve in a cat with severe hypertrophic obstructive cardiomyopathy (HOCM). The right parasternal long-axis left ventricular outflow tract view allows visualization of the anterior mitral valve and shows SAM of the mitral valve **(A)**. Papillary muscles (*asterisk*) are severely hypertrophied and anteriorly displaced, which pull the chordae tendineae and the anterior leaflet (*arrow*) toward the severely hypertrophied basilar interventricular septum (*S*). The left ventricle (*LV*) free wall (*triangle*) also has concentric hypertrophy. Color flow Doppler imaging of the left ventricular outflow tract shows a double turbulent jet where the anterior mitral leaflet obstructs the left ventricular outflow tract and also creates mitral regurgitation seen on right parasternal long-axis left ventricular outflow tract view **(B)** and left apical five-chamber view **(C)**. From the left apical five-chamber view, continuous wave Doppler imaging is used to measure the aortic blood flow velocity, which is 4.87 m/s, corresponding to a severely increased LV/aortic pressure gradient of 95 mm Hg with the modified Bernoulli equation **(D)**. After 2 weeks of atenolol administration, a recheck echocardiogram shows minimal SAM on color flow Doppler imaging **(E)**, and continuous wave Doppler imaging shows normal left ventricular outflow tract velocity of 1.5 m/s corresponding to a normal LV/aortic pressure gradient of 8.5 mm Hg **(F)**. *Ao*, Aorta; *LA*, left atrium.

is forced to form an opinion, often based on personal experience, on the anecdotal experience of an expert, or on small studies in which specific echocardiographic variables that may not actually pertain to the overall course of the disease are examined. Therefore there is great controversy about whether symptom-free cats should be treated and with what specific medical therapy. Ideally the fundamental goals in treatment of symptom-free cats would include reduction in left ventricular hypertrophy, improvement in diastolic function, reduction in risk of ATE, and prolongation to time of heart failure. However, there are little data that confirm or deny that any particular treatment has a considerable effect on these goals. It is likely that many cats with mild-to-moderate HCM may live long lives without any clinical consequences of the disease, and committing to lifelong daily to twice daily medications may be unnecessary. However, many veterinarians feel compelled to treat a patient with a disease, and some owners demand treatment for their pets, even if there is only a theoretic case for using a drug. Consequently, whenever HCM is diagnosed in a cat, the veterinarian must explain the pros and cons of

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treatment options to each owner and try to let the owners make an informed decision based on their wishes and lifestyle.

- The decision of whether to treat a symptom-free cat with HCM may include severity of SAM of the mitral valve, severity of left ventricular hypertrophy, size of the LA, presence of tachyarrhythmias, client factors associated with medicating daily to twice daily indefinitely, and patient temperament. Whatever the initial choice, response to therapy should dictate whether dose adjustment, changing drug class, or discontinuation of therapy is warranted.
- In a prospective, blinded, placebo-controlled study, the effects of atenolol compared with diltiazem in symptom-free cats with HCM were evaluated. Results were relatively underwhelming. Atenolol had modest effects to improve one variable of diastolic function and slightly reduced septal thickness, and there were no significant effects of diltiazem on diastolic function, left atrial size, or left ventricular hypertrophy. Atenolol also reduced the severity of SAM of the mitral valve, and diltiazem did not. An earlier uncontrolled pilot study reported a reduction in left ventricular hypertrophy in cats treated with diltiazem, which has not been demonstrated in subsequent controlled studies. The AQUA study reported that atenolol improved the owner's subjective overall assessment of their cat's quality of life compared with placebo but had no effect in mobility quantified by an accelerometer, or in a detailed questionnaire about quality of life. There was no difference in 5-year cardiac mortality in a nonrandomized, retrospective study of asymptomatic cats placed on atenolol versus no treatment, although the cats in the atenolol group had more severe disease than the cats in the no treatment group.
- Prophylactic antithrombotic agents such as clopidogrel are indicated in cats with any of the following: moderate to severe left atrial dilation, spontaneous echocardiographic contrast or a thrombus on an echocardiogram, or a previous episode of ATE.
- ACE inhibitors are unlikely to impact the severity of left ventricular hypertrophy or diastolic function on the basis of a placebo-controlled, prospective, blinded clinical study. However, they may be considered in cats with severe HCM and moderate-to-severe left atrial dilation with high risk of heart failure.
- Once heart failure develops, use of furosemide and an ACE inhibitor are standard therapies. Unfortunately, heart failure typically represents the end stage of the disease, and survival time often averages months to a year despite medical therapy. Beta blockers are not started in acute heart failure because these patients may have a poorer outcome when receiving beta blockers. Cats already receiving a beta blocker may continue to receive it, but the dose or frequency may need to be reduced. Beta blockers may be considered in cats with severe tachyarrhythmias that have concurrent heart failure. Diltiazem, a calcium channel blocker, can be used to treat supraventricular tachyarrhythmias.
- Controversy exists regarding the use of pimobendan for cats with HCM and heart failure. Potential pharmacodynamic benefits of pimobendan could include increased left atrial function, preload reduction, positive lusitropy (increases diastolic relaxation), and positive inotropy (useful in cats with myocardial failure from end-stage HCM or other cardiomyopathies). A couple of studies in healthy cats have not demonstrated an increase in left ventricular systolic function, although others demonstrated pimobendan increased right ventricular, right atrial, and left atrial systolic function. In one study, acute pharmacodynamic effects of pimobendan in preclinical HCM cats included increased left atrial function, and mildly increased left ventricular and right ventricular systolic function. There may be a dichotomy of clinical impacts of pimobendan in cats with heart failure, depending on presence of left ventricular outflow tract obstruction. In a prospective study of cats with heart failure caused by HCM, there was no difference in 6-month successful outcome of lack of heart failure progression or survival/ secondary morbidity end points in cats given pimobendan in the combined group of cats with SAM and without SAM ($P = .77$).[24] Cats with SAM and CHF had worsened outcome on pimobendan, and cats without SAM had better outcome. Conversely, a retrospective study documented survival benefit in cats with heart failure secondary to HCM with no myocardial failure that were treated with pimobendan and other standard cardiac medications (MST = 626 days) compared to cats not given pimobendan (MST = 103 days).[25] However, the group of cats not receiving pimobendan had a shorter survival time than is often clinically expected, with highly variable literature reported survival times in retrospective studies (92–654 days) in cats with heart failure due to HCM. Dosing for pimobendan in cats is typically 0.25 mg/kg orally every 12 hours, although the plasma drug concentration at this dose is 10 times higher with a longer elimination half-life compared to canine pharmacokinetic data.

Beta-blockers

Atenolol is currently the preferred beta blocker and is a selective B1 blocker administered BID usually but sometimes q24h depending on HR.

- **Atenolol:** 6.25 to 12.5 mg per cat q12h. Atenolol is supplied as 25-mg tablets.

Calcium channel blockers

- **Diltiazem** is currently the preferred calcium channel blocker and is available in three forms:
  - **Cardizem:** 7.5 mg PO q8h. This product is supplied as 30-mg tablets. One-quarter tablet is given q8h.

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- **Dilacor XR:** 30 mg PO q12h. This product is supplied as 120-, 180-, and 240-mg capsules. Each large capsule can be opened to yield two, three, or four 60-mg tablets, which are then halved to achieve a 30-mg dose.
- **Cardizem CD:** 45 mg PO q24h. Cardizem CD is supplied as 180-mg capsules that contain many smaller capsules. The larger capsule can be opened and the smaller capsules divided into a number that produces an appropriate dose; this should be done by a compounding pharmacy.

Key Point

I prefer to use atenolol for treatment of symptom-free cats with moderate to severe left ventricular hypertrophy, moderate to severe SAM, or concurrent tachyarrhythmias and HCM.

Prognosis

- Prognosis is dependent on stage of disease, with negative prognostic indicators including left atrial dilation, CHF, intracardiac thrombus, or ATE. Prognosis of preclinical HCM is good short term, but there is a 12% to 30% risk of developing congestive heart failure or arterial thromboembolism over a 10-year period of time based on two large-sale studies. Risk of heart failure or ATE after 1 year is 1 in 15 cats, after 5 years it increases to 1 in 4.4 cats, and after 10 years it is 1 in 3.5 cats. Cats with preclinical HCM die sooner (MST 10.9 years) than cats that are apparently healthy. Heart failure is the most common clinical sequelae (64%) followed by arterial thromboembolism (16%), then sudden death (2%–8%). Maine Coon cats had a younger age of first cardiovascular event of 2.5 years old compared to 7 years old for other breeds in one study, and had high death rate of 50% over the 10-year study. Risk factors for development of CHF were septal hypertrophy (HR 1.36) and left atrial dilation (HR 1.05), and risk factors for ATE were septal and free wall hypertrophy (HR 1.36 and 1.28, respectively) and left atrial dilation (HR 1.07).
- Symptom-free cats with mild-to-moderate hypertrophy and no LA enlargement are thought to have a good long-term prognosis. Symptom-free cats with obvious wall thickening and LA enlargement are likely at higher risk for developing heart failure. These cats are also thought to be at risk for developing thromboembolic disease.
- In general, cats that have heart failure have a poor prognosis. Survival times range from several months to 1.3 years. The survival time of cats with heart failure was not shown to vary based on age (i.e., once a cat develops heart failure regardless of its age, the survival time remains the same).
- In general, cats with aortic thromboembolism have a poor to grave prognosis. Only 15% to 35% of cats with ATE survive to hospital discharge, although survival analysis is complicated by euthanasia. Single limb ATE conferred a better prognosis with higher likelihood of recovery from the acute event in 68% to 93% of cats. Typical long-term survival times in cats that are not euthanized at the time of the diagnosis may be several months (4–8 months). Cats with concurrent heart failure and ATE had shortest survival time of 77 days in one study. The cause of death in cats that recover from acute ATE is usually heart failure, followed by recurrent ATE. Prolonged survival times have been reported by the FATCAT clinical study of cats with ATE (from various cardiomyopathies) treated with clopidogrel (442 days) compared with those treated with aspirin (192 days). A small, retrospective study of combination therapy of rivaroxaban and clopidogrel in 18 ATE cats discharged from the hospital reported prolonged survival time of 502 days with low ATE recurrence rate of 16.7%.
- Owners should always be warned of the potential for sudden death in cats with HCM, even symptom-free cats with mild HCM. Incidence of sudden death in preclinical HCM ranges from 2% to 8%, and incidence in clinical cats is not defined. Postulated causes of sudden death are ventricular arrhythmia or large intracardiac thrombus.
- Transient myocardial thickening (TMT) is often a retrospective diagnosis characterized by resolution of left ventricular concentric hypertrophy within several months of time. Cats with TMT are often younger (mean age 2 years) than primary HCM cats, with a majority (70%) suffering from antecedent stressful events such as anesthesia just prior to the diagnosis. Cardiac troponin I levels are typically markedly increased. Left atrial dilation and heart failure is common in cats with TMT. Reverse ventricular remodeling typically occurs over 2 to 5 months of time, with normalization of left ventricular concentric hypertrophy and left atrial size, allowing most cat to be weaned off cardiac medications. Causes of TMT in humans include storage diseases leading to intracellular accumulation of metabolic products, or interstitial infiltration of proteins, cells, or fluid seen in amyloidosis or myocarditis. Infectious etiologies causing myocarditis may include *Toxoplasma*, FIV, *Bartonella*, or eosinophilic myocarditis. Feline wildfire burn victims have also been demonstrated to have increased ventricular wall thickness, which may represent myocardial edema.

FELINE (IDIOPATHIC) DILATED CARDIOMYOPATHY

DCM is a disease of the ventricular myocardium (predominantly left) characterized by *primary* systolic myocardial failure.

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GENERAL COMMENTS

- Before 1987, DCM was one of the most commonly diagnosed heart diseases in cats. Most cases at that time were likely a secondary cardiomyopathy associated with nutritional taurine deficiency. In current times, DCM is uncommon in cats and is most often caused by primary idiopathic DCM.
- Idiopathic DCM is a diagnosis of exclusion, ruling out secondary causes of myocardial failure such as taurine deficiency, tachycardiomyopathy, severe volume overload leading to secondary myocardial failure (i.e., severe mitral insufficiency or large left to right shunting congenital heart diseases), or toxicity such as doxorubicin cardiotoxicity.
- The underlying etiology of idiopathic DCM remains unknown and may represent a common endpoint to many processes. A heritable cause has not been identified in cats but is an important cause of DCM in humans where 30% to 50% of cases are caused by mutations in more than 30 genes.
- Another secondary and reversible cause of myocardial failure that mimics idiopathic DCM is severe hypocalcemia, a syndrome reported in a cat with primary hypoparathyroidism.

Taurine Deficiency-Induced Myocardial Failure

- Taurine deficiency-induced myocardial failure is associated with low plasma, whole blood, and tissue taurine concentrations that may be reversible after taurine supplementation.
- Taurine is an essential sulfur-containing amino acid in cats and has an obligatory role in bile salt conjugation. Taurine is synthesized by the liver and brain from dietary sulfur amino acids (cysteine), and the rate of synthesis in the cat is much lower than its loss though conjugation of cholic acid. Taurine homeostasis in cats depends on dietary intake, endogenous synthesis, enterohepatic turnover, microbial degradation in the large intestine, and urinary and fecal losses.
- Typical diet history may include feeding home-cooked diets low in taurine (such as chicken breast) or feeding solely one canned food for many years.
- Not all taurine-deficient cats have myocardial failure (20%–65% of cats). The other factors required for taurine deficiency causing myocardial failure are unknown.
- The role that taurine, an essential amino acid in the cat, plays in the maintenance of myocardial function remains unknown. Taurine has diverse functions, with the most important including modulation of contractility (possibly through myocardial calcium homeostasis), metabolic and osmotic regulation of the myocardium, membrane stabilization of photoreceptor cells of the retina, and neuroinhibitor actions in the central nervous system.

Key Point

Although myocardial failure secondary to taurine deficiency is now quite rare in cats, all cats with myocardial failure should be assumed to be taurine deficient until shown not to be taurine responsive.

PATHOPHYSIOLOGY

- The underlying abnormality leading to clinical manifestations in cats with idiopathic DCM is primary systolic myocardial failure. End-systolic left ventricular volume increases, owing to a reduction in myocardial pump function. As a result, stroke volume and cardiac output decrease. Neurohumoral compensatory mechanisms promote an increase in intravascular volume and end-diastolic pressures, stimulating eccentric hypertrophy (dilation). At these larger left ventricular end-diastolic volumes, the geometry of the ventricle is such that small changes in chamber dimension during systole provide adequate stroke volume and cardiac output; however, working at these larger volumes is energetically inefficient for the ventricle. At any point in this degenerative process that end-diastolic pressures rise too high or cardiac output drops too low, the patient may have signs of CHF or low-output heart failure. respectively. Biventricular failure, including pulmonary edema and pleural effusion, is common in cats because both the left and right ventricles are affected.
- Often cats are not diagnosed until they have symptoms for heart failure at an end stage of the disease.

SIGNALMENT AND PRESENTING COMPLAINTS

- Strong breed predispositions are not observed in DCM but it has been reported in the following breeds: Persian, Domestic Shorthair, Domestic Longhair, Birman, Siamese, and Burmese.
- It is an adult-onset disease, with mean age reported to range from 7 to 9 years but with a wide range of 5 months to 14 years.
- No sex predilection is evident.
- Cats with DCM may have a variable period of lethargy, anorexia, and malaise before overt signs of CHF, including dyspnea and tachypnea.
- Cats may have no prior signs but an acute onset of CHF or systemic thromboembolism.

PHYSICAL EXAMINATION

- A gallop heart sound is the most common auscultation finding (79% of cats), and a soft left parasternal systolic murmur is less commonly present (17% of cats). Most cats with DCM have heart failure, with pleural effusion more common than pulmonary edema (91% compared with 36%). Cats usually exhibit tachypnea, dyspnea, and orthopnea, and lung sounds are often muffled. Femoral

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arterial pulses are usually weak. Right-sided heart failure may also be present, including signs of jugular distension and a pendulous abdomen. A retinal examination is necessary to evaluate for taurine deficiency-associated central retinal lesions because taurine deficiency is the main differential for idiopathic DCM. Please refer to the previous general section on cardiomyopathies for a complete discussion of physical examination abnormalities.

ANCILLARY TESTS

- Measurement of whole blood and plasma taurine blood concentration is an essential test for cats with echocardiographic evidence of myocardial failure.
- Ideally, both whole blood and plasma levels are measured, but if only one can be measured because of financial constraint, whole blood is recommended. Plasma taurine concentration is subject to significant fluctuations depending on the fasted state of the cat. In cats fed diets adequate in taurine, plasma taurine concentration was critically low (<30 nmol/mL) in 9 (26%) of 35 normal cats that were fasted for 71 hours and in 2 normal cats that were fasted for only 23 hours.
- Taurine deficiency is defined as plasma taurine concentration <50 nmol/mL or whole blood taurine concentration of ≤250 nmol/mL. There are strict guidelines for obtaining and handling blood samples for taurine analysis; please refer to laboratory specifications.

ECHOCARDIOGRAM

DCM is defined as primary myocardial failure, which is diagnosed by a decreased FS of <26% and an increased end-systolic diameter >11 mm (Fig. 8.5; see Table 8.1). There is a secondary compensatory eccentric hypertrophy that is identified as an increased end-diastolic left ventricular diameter (>18 mm). E-point to septal separation is another index of systolic function and is increased (>4 mm). Left atrial dilation occurs secondary to elevated left ventricular filling pressure and is identified as a left atrial/aortic ratio > 1.5. Other subjective findings often include right atrial dilation and right ventricular eccentric hypertrophy, mild pericardial effusion, and pleural effusion. Mild centrally arising AV valvular insufficiency secondary to decreased AV valve coaptation is also commonly present. Spontaneous echocardiographic contrast or an atrial thrombus may be present.

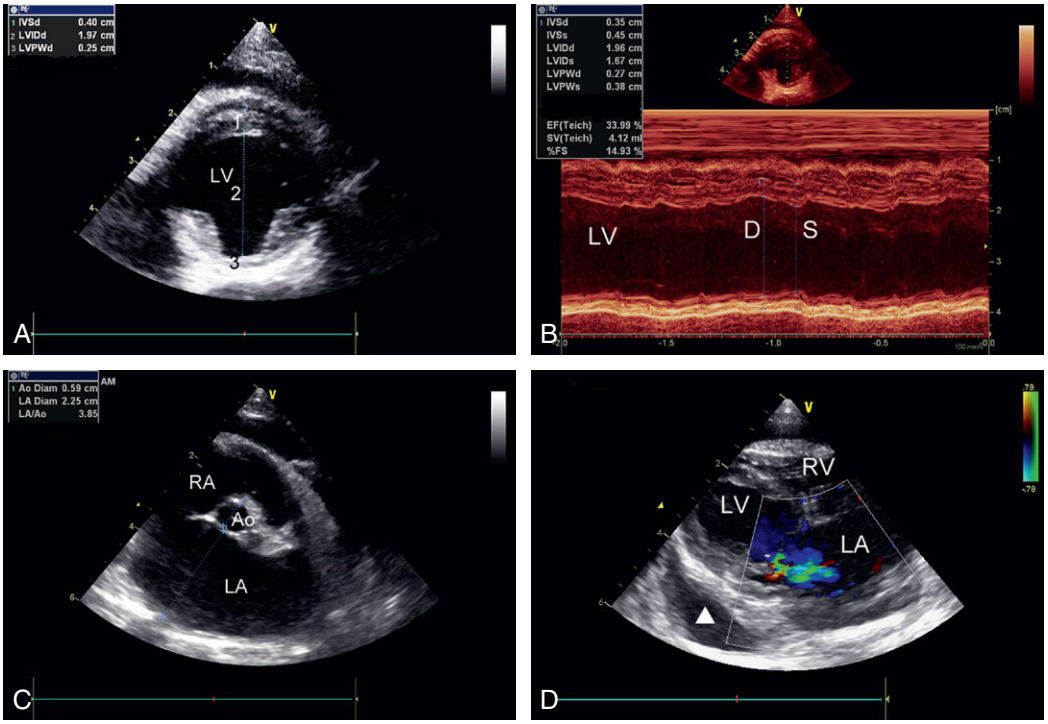


Fig. 8.5 Echocardiogram of a cat with severe dilated cardiomyopathy. This two-dimensional echocardiogram from the right parasternal cross-sectional view at the level of the papillary muscles shows severe eccentric hypertrophy of the left ventricle (*LV*), which is a compensatory mechanism to normalize stroke volume in the face of severe biventricular systolic myocardial failure **(A)** and a thin left ventricular free wall (*LVPWd*). **(B)** Placing the cursor through the septum and free wall in this view, an M-mode tracing is obtained, which allows measurement of the left ventricular chamber diameter in end diastole (*D*) and end systole (*S*) and calculation of fractional shortening (*FS*). This M-mode shows severe myocardial failure evidenced by increased end-systolic diameter, marked reduction in FS (15%), and compensatory increase in left ventricular end-diastolic diameter (eccentric hypertrophy). **(C)** There is severe left atrial dilation, which is assessed by measurement of the aortic and left atrial diameters on the first frame of aortic valve closure with two-dimensional echocardiography, and with calculation of the left atrial to aortic ratio, which in this cat is severely increased at 3.85 (<1.5 normal). Color flow Doppler interrogation of the mitral valve often shows mild centrally arising functional mitral regurgitation secondary to severe ventricular volume overload. There is also evidence of pleural effusion (*triangle*) **(D)**. *Ao*, Aorta; *RA*, right atrium; *RV*, right ventricle; *LV*, left ventricle.

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THERAPY

- For acute CHF, see the earlier section on treatment of CHF in the discussion of therapy in the general information section. Pimobendan (1.25 mg PO BID or 0.25 mg/kg PO BID) is indicated for treatment of heart failure, and cats with low output heart failure (hypotension, cardiogenic shock) may also require a dobutamine CRI.
- Chronic therapy for cats with DCM and heart failure is similar to other cardiomyopathies and also includes pimobendan and possibly digoxin (0.007 mg/kg PO q48h or ¼ of a 0.125-mg tablet orally once a day for cats weighing >4 kg, or q48h for cats weighing <4 kg). Avoidance of negative inotropes such as beta blockers is necessary to avoid life threatening decompensation. Digoxin or diltiazem would be appropriate antiarrhythmic choices for rapid supraventricular tachyarrhythmias. Clopidogrel is also likely necessary for prevention of possible ATE, which is common in cats with DCM (18% of cats had echocardiographic evidence of intracardiac thrombi in one study).
- Treatment of taurine deficiency-induced myocardial failure
  - Taurine 250 mg PO BID is empirically administered until results of the blood taurine levels are obtained, and if deficiency is documented, taurine supplementation may be continued long term. Clinical signs typically improve within 2 weeks, and by 3 to 6 weeks there is echocardiographic evidence of decreased end-systolic left ventricular diameter, increased FS, and decreased end-diastolic left ventricular diameter. Myocardial failure should improve but may persist for up to a year after supplementation.
  - Most taurine-responsive cats have complete reversal of echocardiographic and clinical evidence of myocardial failure with supplementation of taurine after several months. Occasionally cats may have residual mild myocardial failure (left ventricular shortening fraction, 25%–30%); however, these cats are generally symptom free and rarely require any form of therapy other than maintaining normal plasma taurine concentrations.
  - Diuretics and ACE inhibitors can be discontinued when clinical signs resolve and cardiac size is smaller on radiographs or echocardiogram, usually after several months of therapy. The ACE inhibitor should be removed first and then the diuretic tapered over a period of 2 weeks. The owner should be taught to monitor the respiratory rate while withdrawing heart failure medications, and clinical and radiographic evaluation should be repeated 1 week after withdrawing medications to detect any decline in the cat's condition. Likewise, pimobendan may be weaned off after several months, once the heart size reduces and the myocardial failure improves.
  - The diet should be altered to maintain normal plasma taurine concentrations (>60 nmol/mL). Taurine supplementation is often continued lifelong.
  - Taurine concentration in plasma and whole blood should be monitored periodically to be certain that the diet fed and taurine supplementation are maintaining concentrations within acceptable limits.

PROGNOSIS

- Prognosis for idiopathic DCM is grave, but there are minimal data documenting clinical characteristics and outcome. Typically diagnosis of DCM is at end stage of disease when cats have fulminant heart failure. Cats treated with pimobendan had fourfold extension in survival time, and lived 49 days compared to cats not given pimobendan who lived only 12 days. Preclinical cats with myocardial failure are rarely identified, but these cats may live months to years before succumbing to heart failure or ATE, if treated with pimobendan. Cats with signs of low-output heart failure or fulminant heart failure have a grave prognosis. These cats usually either die soon after admission from cardiogenic shock or succumb quickly to refractory CHF or ATE.
- Taurine deficiency-induced myocardial failure confers a good prognosis if the cat can survive the first several weeks of therapy. In one large study, 30% of cats with myocardial failure died within the first week after diagnosis. Hypothermia and thromboembolic disease were associated with a poor prognosis. Early cardiovascular death within the first month occurs in approximately 38% of treated cats. However, if a cat survives after the first 2 weeks of therapy, there is a 96% chance of long-term survival with complete recovery if there is documented taurine deficiency.

FELINE RESTRICTIVE CARDIOMYOPATHY

RCM is a diverse group of conditions characterized by restriction of diastolic filling caused by an increased ventricular stiffness. The clinical picture is of a normal ventricle with normal wall thickness, normal ventricular size and systolic function, but presence of left atrial or biatrial dilation with normal AV valves. Specific clinical and morphologic criteria for this diagnosis in the cat have not been as clearly defined as they have in humans.

GENERAL COMMENTS

- In human medicine, RCM is divided into two main categories: endomyocardial fibrosis and interstitial myocardial fibrosis. Without the use of invasive diagnostic procedures such as endomyocardial biopsy or necropsy examination, it is impossible to differentiate the myocardial fibrosis form of RCM

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from UCM; therefore the two diseases in cats have often been combined in the literature as RCM/UCM.
- Etiology in cats remains idiopathic, and no familial basis has been reported in cats.

PATHOPHYSIOLOGY

- The characteristic functional abnormality of RCM is increased left ventricular stiffness causing impaired diastolic filling and elevated diastolic-filling pressure. Fibrosis occurs within the myocardium, subendocardium, or endocardium, with an end result of marked diastolic dysfunction. Once left ventricular filling pressure increases to >25 mm Hg, pulmonary edema or pleural effusion develops (i.e., left CHF). Tachyarrhythmias may accentuate the diastolic dysfunction and contribute to further increase in diastolic-filling pressure.
- LV disease predominates. In most cases, systolic function is preserved. In the endomyocardial fibrosis form of RCM, a fibrotic band may bridge the LV and distort the mitral valve to create both mitral regurgitation and systolic obstruction of blood flow in the LV.

SIGNALMENT AND PRESENTING COMPLAINTS

- Signalment is difficult to report accurately because there is little agreement among cardiologists as to which cases fall within this classification compared with patients with UCM. The myocardial fibrosis form has been described as an animal model of human RCM in 35 cats at the Animal Medical Center. RCM represented 4.5% of all feline cardiac cases seen at the Animal Medical Center in that report. Seventy-one percent of cats were male, with a mean age of 10 ± 4 years (range 1.5–17.1 years). In contrast, a previous case series reported a female predisposition (71% of cats) and a mean age of 7.1 years ± 3 years. A majority of cats (32/35 in one report) have clinical signs of heart failure.

PHYSICAL EXAMINATION

- Auscultation abnormalities are invariably present and less common than those in cats with HCM; 36% of cats with RCM had a murmur, 23% of cats had a gallop, and 14% of cats had an arrhythmia in one report. Please refer to the previous general section of cardiomyopathies for a complete discussion of physical examination abnormalities.

ECHOCARDIOGRAM

- Left atrial or biatrial dilation in the face of normal left ventricular size, normal systolic function, and minimal left ventricular hypertrophy are hallmark echocardiographic findings. Intracardiac thrombi or spontaneous echocardiographic contrast are common (reported in 23% of cases). In cats with endomyocardial fibrosis, the endocardium is hyperechoic, thickened, and there is often a fibrotic band bridging from the papillary muscles/free wall to the interventricular septum. This fibrotic band often distorts the papillary muscle shape and involves the mitral valve apparatus to create both mitral regurgitation and intraventricular systolic obstruction of blood flow out the LV (Fig. 8.6).
- A restrictive ventricular filling pattern is the hallmark characteristic functional abnormality seen in patients with RCM and occurs when there is severe diastolic impairment leading to markedly elevated left atrial pressure and a high left atrial to left ventricular diastolic pressure gradient. It is characterized by an increased early diastolic left ventricular filling velocity (>1 m/s), a decreased atrial filling velocity (<0.4 m/s), an increased E/A ratio (>2), a shortened deceleration time (normal is 59 ± 14 m/s), and decreased isovolumic relaxation time (normal is 55 ± 13 m/s). Tissue Doppler imaging of the mitral annulus also would demonstrate diastolic dysfunction with reduced E′ velocity.

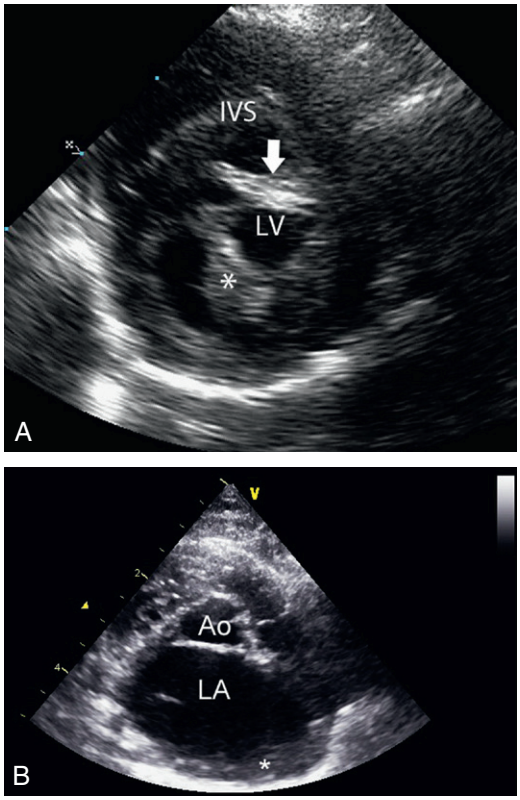


Fig. 8.6 Echocardiogram of a cat with severe endomyocardial fibrosis and restrictive cardiomyopathy. **(A)** This two-dimensional echocardiogram of the left ventricle (*LV*) at the level of the papillary muscles shows a large fibrotic bridging band (*arrow*) tethering the anterior left ventricular free wall and papillary muscle (*asterisk*) to the interventricular septum (*IVS*). **(B)** There is severe left atrial dilation, which is assessed by measurement of the aortic and left atrial diameters on the first frame of aortic valve closure in the right parasternal cross-sectional view with two-dimensional echocardiography and with calculation of the left atrial to aortic ratio. In this cat, it is severely increased at 2.74 (≤1.5 normal). Spontaneous echocardiographic contrast (*asterisk*) is also seen within the left atrium. *Ao*, Aorta; *LA*, left atrium.

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ANCILLARY TESTS

- As with other forms of myocardial disease, ancillary tests rarely help discriminate the diagnosis from other cardiomyopathies.
- Arrhythmias appear to be common in cats with RCM and may include supraventricular tachycardia (7%–34% of cats), VPCs or ventricular tachycardia (up to 28% of cats), or less likely atrial fibrillation (3% of cats).
- There is often dramatic atrial dilation seen on thoracic radiographs. Radiographic evidence of heart failure is common (32/35 cats) and may include cardiogenic pulmonary edema and pulmonary venous distension (13/35 cats), signs of right heart failure (pleural effusion, dilated caudal vena cava, hepatomegaly, +/− ascites in 6/35 cats), or biventricular failure (13/35 cats).

THERAPY

- For acute CHF, see the earlier section on treatment of CHF in the discussion of therapy in the general information section.
- No specific therapy for controlling the fibrous tissue reaction is available.
- Beta blockers or calcium channel blockers are not effective for improving diastolic function caused by fibrosis but may be of benefit to control persistent significant tachycardia if present. Likewise, antiarrhythmic medications are indicated for treatment of ventricular or supraventricular tachyarrhythmias.
- Antithrombotic therapy is recommended in cats with moderate to severe atrial dilation, intracardiac thrombus, or spontaneous echocardiographic contrast. Cats with RCM have a high risk of ATE (45% incidence in one study), likely because they are often diagnosed at an end stage where there is severe atrial dilation. Clopidogrel is typically the drug of choice for cats with risk of ATE, although cats that have suffered from ATE or have heavy spontaneous echo contrast or intracardiac thrombus may be treated with combination therapy, as previously described.
- Pimobendan may be considered in cats with RCM and CHF because it may improve atrial function, decrease preload, and has positive lusitropic effects (improves relaxation). However, overall diastolic function is unlikely to be improved because the main problem is increased ventricular stiffness from fibrosis.

PROGNOSIS

- Prognosis is typically poor because most cats are diagnosed at an end stage when there are signs of heart failure or ATE. In a report of 35 cats, MST was 3.4 months (0.1–52 months). Heart failure (18/35 cats), ATE (5/18 cats), and sudden cardiac death (3/35 cats) are the most common causes of death. Since the use of pimobendan, the outcome may be slightly improved with MST of 273 days in 14 cats with RCM over a 10-year period of time, which was shorter than cats with HCM (MST of 865 days). Heart failure was present in 12 or 14 (86%) of RCM cats compared to 19 of 50 (38%) of HCM cats, and ATE occurred in 2 of 14 (14%) RCM cats compared to 5 of 50 (10%) HCM cats.

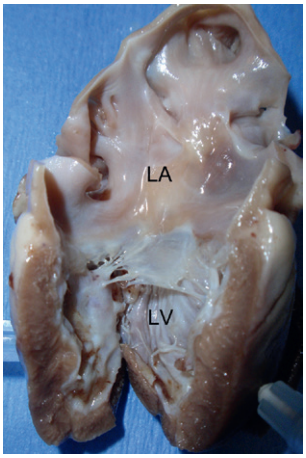


Fig. 8.7 Gross disease of a cat with severe endomyocardial fibrosis and restrictive cardiomyopathy. Gross pathologic examination of a heart with severe endomyocardial fibrosis of the left ventricle (*LV*). There is diffuse, thick endocardial and subendocardial fibrosis of the LV, causing severely increased left ventricular diastolic-filling pressures and leading to severe left atrial dilation and heart failure. *LA*, Left atrium.

PATHOLOGY

- Endocardial, subendocardial, and/or myocardial fibrosis are hallmark lesions of RCM (Fig. 8.7). Diffuse interstitial fibrosis is commonly present within the myocardium, and some cats have larger patchy regions of replacement fibrosis. In addition, many cats have evidence of myocyte disarray, which is also seen in cats with HCM. Endocardial fibrosis may cause fibrous adhesions between papillary muscles and the myocardium, with distortion and fusion of the chordae tendineae and mitral valve leaflets. Extreme LA and left auricular enlargements are common, and right atrial dilation may also be present.

NONSPECIFIC FELINE CARDIOMYOPATHIES

NCM is a nebulous category including cats with significant left atrial or biatrial dilation despite normal to near-normal systolic function and normal to near-normal LV wall thickness in the absence of severe AV valvular dilation. Previous nomenclature for this group of cats was unclassified cardiomyopathy. There can be difficulty in discriminating NCM from RCM in cats where the restrictive ventricular filling pattern cannot be diagnosed due to high heart rates and fusion of

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transmitral Doppler ventricular E and A filling waves. There is a lack of specific criteria for diagnosis of NCM. It is possible this category could represent transition from one form of cardiomyopathy to another, atrial myopathy, or ventricular myocardial disease not specific to hypertrophy or fibrosis.

GENERAL COMMENTS

- In recent years, increasing numbers of cats have been recognized with obviously abnormal hearts, many having heart failure, but not fitting into any recognized disease classification such as HCM, RCM, or DCM. There is a lack of specific criteria for diagnosis of NCM. It is likely the abnormalities are acquired, but the etiology remains unknown.
- Although no controlled studies have been performed, taurine deficiency or metabolic abnormalities (e.g., hyperthyroidism) have not been consistent findings in affected cats.

PATHOPHYSIOLOGY

- The pathophysiologic condition is unknown; however, clinical observations suggest diastolic dysfunction similar to that described for RCM is the predominant functional abnormality in these cats. Atrial myopathy is another hypothesis for the atrial dilation, but this has not been confirmed.

SIGNALMENT AND PRESENTING COMPLAINTS

- There is no sex or breed predisposition for NCM, and mean age in a case series of 11 cats was 8.8 ± 4.8 years.
- Similar to RCM, many cats have clinical signs of dyspnea (64%), tachypnea, and nonspecific signs of lethargy (27%), anorexia, or weight loss.

PHYSICAL EXAMINATION

- A murmur is variably present in cats (50% in one case series). An arrhythmia is less common (18% in one case series), and a gallop heart sound may also be present. Physical examination is similar to that of other forms of myocardial disease.

ECHOCARDIOGRAM

By nature of the definition, the echocardiographic findings are extremely variable. Left atrial or biatrial dilation is the hallmark abnormality seen on an echocardiogram in the face of a relatively normal LV (i.e., normal chamber size, no significant left ventricular concentric hypertrophy, and normal to nearly normal systolic function) (Figs. 8.8 and 8.9). There may be mild systolic dysfunction with mildly increased end-systolic left ventricular internal diameter and mildly decreased FS. Various patterns of mild regional myocardial hypertrophy may be observed in the septum or LV free wall of some cats. Enlargement of the right heart is variable but may be marked in some cases. Mild AV valvular insufficiency is often present on color flow Doppler imaging. Diastolic dysfunction may be evident on tissue Doppler imaging or PW Doppler interrogation of mitral inflow. Atrial thrombus or spontaneous contrast may be seen in cats with atrial dilation.

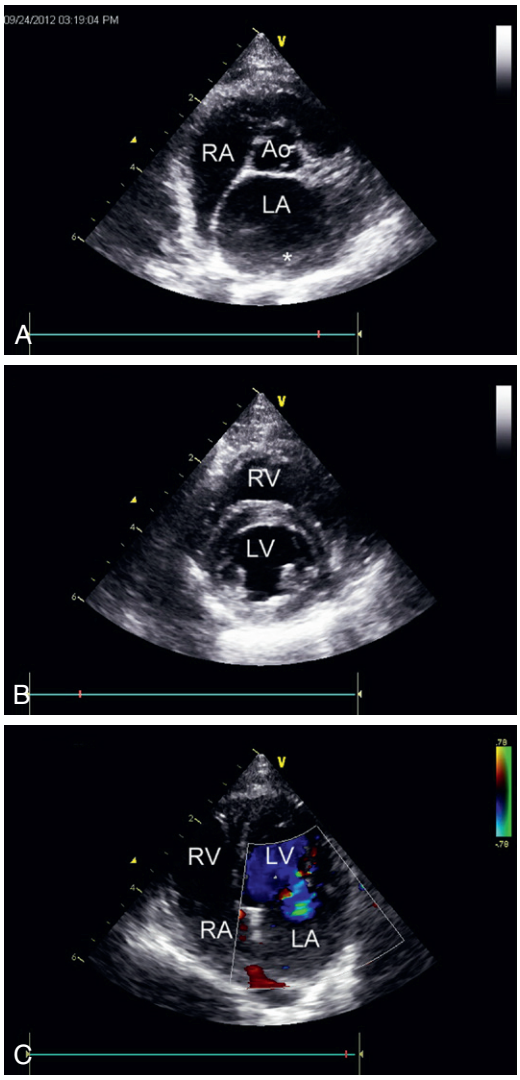


Fig. 8.8 Echocardiogram of a cat with severe unclassified cardiomyopathy and severe left atrial dilation. **(A)** This cat with severe UCM has severe left atrial dilation, evidenced by a marked increase in left atrial diameter to aortic diameter ratio of 2.47 (≤1.5 normal) measured in the right parasternal cross-sectional view with two-dimensional echocardiography. There is also spontaneous echocardiographic contrast (*asterisk*) in the left atrium. **(B)** The left and right ventricles appear normal, with normal left ventricular wall thickness and normal systolic function. **(C)** Color flow Doppler imaging of the mitral valve from the left apical four-chamber view depicts mild centrally arising mitral regurgitation and severe left atrial dilation. *Ao*, Aorta; *LA*, left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

ANCILLARY TESTS

- Thoracic radiographs often reveal severe cardiomegaly and atrial dilation. Pleural effusion is also common (50% of cats in a case series of 22 cases), and pulmonary edema was reported less frequently (9% of cases).

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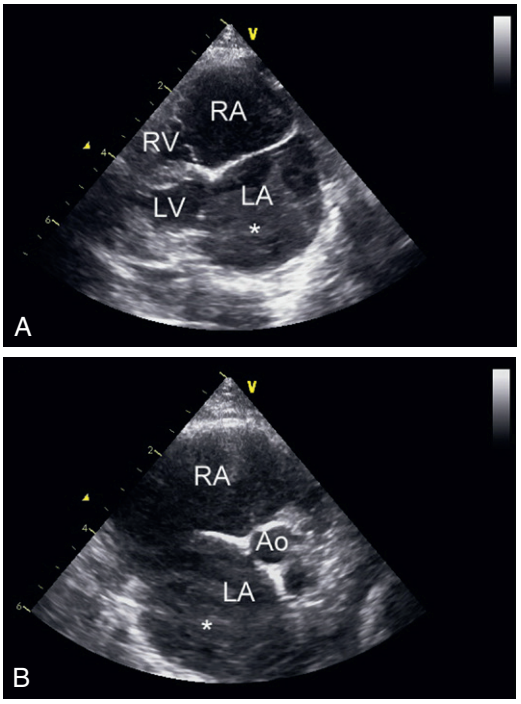


Fig. 8.9 Echocardiogram of a cat with severe unclassified cardiomyopathy (UCM) and biatrial dilation. UCM may cause biatrial dilation, which is seen in this right parasternal four-chamber view **(A)** and in the right parasternal cross-sectional view **(B)**. There is spontaneous echocardiographic contrast (*asterisk*) within the left atrium. With the right parasternal cross-sectional view, the left atrial diameter to aortic diameter ratio is severely increased at 3.42 (≤1.5 normal), indicating severe left atrial dilation, and the right atrium (*RA*) is subjectively also severely dilated. *Ao*, Aorta; *LA*, left atrium; *LV*, left ventricle; *RV*, right ventricle.

- An electrocardiogram may show arrhythmias such as VPCs (50% of cases), supraventricular tachycardia, atrial premature complexes, or atrial standstill (18% of cases). Atrial standstill is rare in cats and may be the cause of the atrial dilation and atrial myopathy in some cats with NCM.

THERAPY

- For acute CHF, see the earlier section on treatment of CHF in the discussion of therapy in the general information section. Pimobendan is indicated in these patients. For cats with chronic CHF, furosemide and ACE inhibitors are recommended (see Therapy, general comments).
- Treatment is essentially the same as outlined in the previous RCM section.
- Anticoagulant therapy is often recommended because atrial dilation is present in cats with NCM.

PROGNOSIS

- The prognosis is generally based on clinical presentation, echocardiographic and radiographic evidence of elevated diastolic pressures, and response to therapy.
- Symptom-free cats with mild LA enlargement are thought to have a good long-term prognosis. Symptom-free cats with marked LA enlargement are likely to be at higher risk for developing heart failure.
- In general, cats that have heart failure have a poor prognosis. However, cats that have heart failure and respond favorably to therapy may do well for prolonged periods of time.
- Cats with ATE have a poor prognosis. Cats that survive the thromboembolic episode may do well for extended periods; however, these cats are generally at risk for recurrence of ATE.

ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY

ARVC is a rare myocardial disease in cats (diagnosed in 2 of 287 cats with cardiomyopathy) and is more commonly seen in humans and Boxer dogs. ARVC is characterized by fatty or fibrofatty replacement of cardiomyocytes in the right ventricle and right atrium, sometimes extending in a milder degree to the left ventricular myocardium. No familial pattern has been reported in cats, and etiology remains unknown in cats.

GENERAL COMMENTS

Little is known about ARVC in cats, and there is only one case series of 12 cats diagnosed with ARVC in the medical literature. Unlike other cardiomyopathies, the predominant feature of ARVC is right heart enlargement, with left atrial dilation also possible in some cats. ARVC causes signs of right heart failure, including pleural effusion, jugular venous distension, ascites, and hepatomegaly.

PATHOPHYSIOLOGY

Fatty or fibrofatty replacement of cardiomyocytes in the right atrial and ventricular myocardium causes systolic myocardial failure, chamber dilation, and elevated right heart filling pressures. Once right ventricular diastolic pressure exceeds approximately 15 mm Hg, right-sided heart failure develops, including pleural effusion and ascites. The fatty replacement of the cardiomyocytes also leads to malignant ventricular and sometimes supraventricular tachyarrhythmias, which may cause sudden cardiac death. The same disease may also occur in the left atrial myocardium and cause left atrial dilation.

SIGNALMENT AND PRESENTING COMPLAINTS

- There is no breed or sex predilection for ARVC in cats, and a mean age of diagnosis was 7.3 ± 5.2 years in one case series of 11 cats with ARVC.
- Presenting complaints are often referable to heart failure and include dyspnea, tachypnea, abdominal distension, lethargy, anorexia, or weight loss.

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PHYSICAL EXAMINATION

- A majority of cats have clinical evidence of right-sided heart failure (67% of cats in a case series). A right parasternal holosystolic murmur is commonly present (67% of cats) and is caused by functional tricuspid regurgitation.

ECHOCARDIOGRAM

- Severe right atrial dilation and right ventricular eccentric hypertrophy (increased end-diastolic diameter of the right ventricle) are the classic echocardiographic abnormalities (Fig. 8.10). Sometimes ventricular aneurysms may be identified. Mild-to-moderate centrally arising tricuspid regurgitation is often present, secondary to right ventricular and atrial dilation and functional tricuspid regurgitation. Varying degrees of left atrial dilation may be present. Pleural effusion, hepatic venous distension, and possibly ascites are common in cats with severe ARVC and right-sided heart failure. Mild pericardial effusion also may be present secondary to right-sided heart failure. Intraatrial thrombus or spontaneous echocardiographic contrast is also commonly seen.
- The main differential diagnosis for ARVC is tricuspid valve (TV) dysplasia. Therefore careful evaluation of the TV for malformations suggestive of TV dysplasia is necessary because TV dysplasia confers a better prognosis than ARVC.

ANCILLARY TESTS

- Thoracic radiographs typically show severe right-sided heart enlargement and evidence of right-sided heart failure, including pleural effusion (67% of cats), ascites (33% of cats), and +/− dilated caudal vena cava (17% of cats).
- Arrhythmias are very common in cats with ARVC, which may include VPCs (75% of cats in a case series), ventricular tachycardia (38% of cats), or atrial fibrillation (50% of cats).

THERAPY

Therapy is identical to cats with DCM; please refer to that section.

PROGNOSIS

Prognosis for cats with ARVC is grave. In a case series of 12 cats with ARVC, 75% of cats died or were euthanized because of profound right-sided heart failure or ATE, with an MST of 1 month.

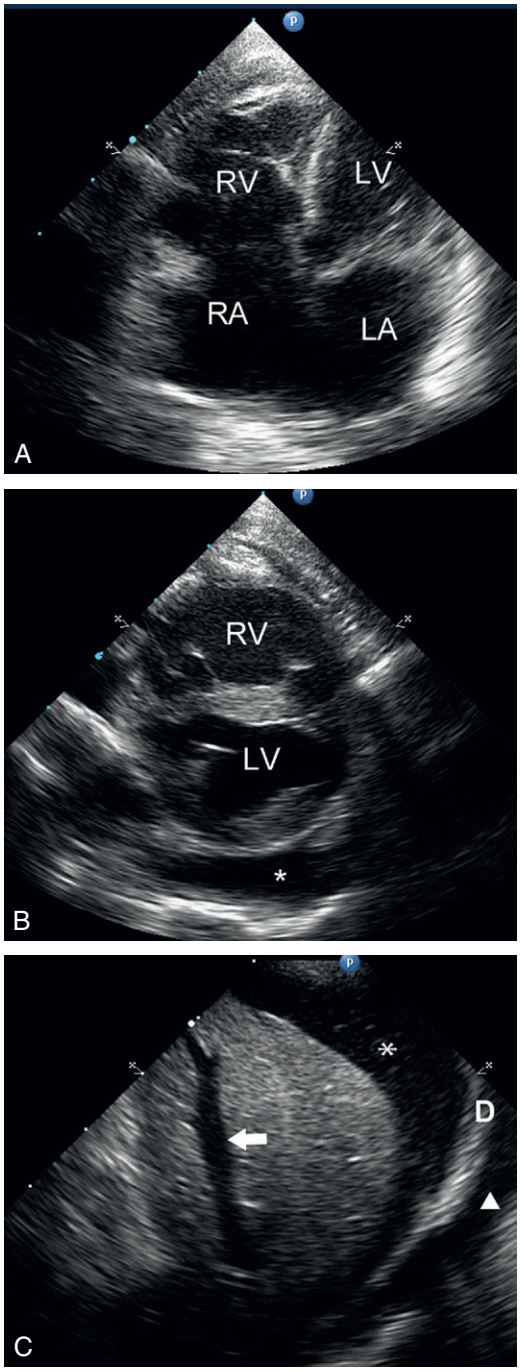


Fig. 8.10 Echocardiogram of a cat with severe arrhythmogenic right ventricular cardiomyopathy (ARVC) and right-sided heart failure. Two-dimensional echocardiograms from the left apical four-chamber view **(A)** and right parasternal cross-sectional view at the level of the papillary muscles **(B)** show severe right ventricular eccentric hypertrophy secondary to the severe right ventricular myocardial failure, severe right atrial dilation, and pleural effusion (*asterisk*). **(C)** Right-sided heart failure is present, including hepatomegaly, hepatic venous distension (*arrow*), ascites (*asterisk*), and pleural effusion (*triangle*). *D*, Diaphragm; *LA*, left atrium; *LV*, left ventricle; *RA*, right atrium; *RV*, right ventricle.

SECONDARY CARDIOMYOPATHIES

THYROTOXIC HEART DISEASE

Thyrotoxic heart disease is a secondary cardiomyopathy caused by direct and indirect effects of high-circulating thyroid hormone on the cardiovascular system. In the current era of earlier detection of hyperthyroidism, most cardiac changes seen on echocardiography (i.e., thick left ventricular and septal walls) are mild and are reported in 37% of hyperthyroid cats.

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General Comments

- Thyrotoxic heart disease is a frequently recognized secondary cardiomyopathy that may be confused with HCM, a primary myocardial disease.
- Thyrotoxic heart disease or hyperthyroidism does not cause HCM.
- The prevalence and severity of thyrotoxic heart disease has been decreasing in recent years, likely as a result of increased awareness and, as a consequence, early diagnosis and treatment of hyperthyroidism.
- Isolated hyperthyroidism rarely causes CHF in current times, but cats may also be affected with a concurrent (additional) hypertrophic cardiomyopathy that has been exacerbated by hyperthyroidism.

Pathophysiology

- The effects of thyroid hormone on the heart are thought to be both direct and indirect.
- The sum effect of excess thyroid hormone (hyperthyroidism) is a heart that operates at a faster rate (tachycardia), is hypertrophied, can contract faster and more powerfully (enhanced contractility), and has a propensity to abnormal electrical depolarizations (arrhythmias).
- Although these might at first glance sound like beneficial changes (bigger, faster, stronger, more excitable), the thyrotoxic state greatly strains the energy economy of the heart and increases the overall work of the heart. In addition, the thyrotoxic heart, although hyperkinetic when the patient is at rest, has less "reserve capacity" available for when increased cardiac work is necessary (e.g., exercise). This situation placed on top of preexisting cardiac disease (e.g., HCM, RCM, DCM, valvular disease) can lead to decompensation of a previously well-compensated cardiac disease.
- Reduced systemic vascular resistance in the presence of an increased intravascular volume (not documented in cats) associated with significant increases in cardiac output are what define the high-output state of the cardiovascular system in hyperthyroid cats. This high-output state can (especially in the presence of underlying primary cardiac disease) progress, resulting in clinically apparent signs of CHF in hyperthyroid cats.

Signalment and Present Complaints

- There is no sex or breed predisposition. Affected cats are typically adult to geriatric, with a mean age reported as 13 years (range 4–22 years).
- Most cats are seen for routine examination or because of signs referable to increased metabolic activity, such as polyphagia, polyuria/polydipsia, or weight loss.
- On rare occasions, cats may have CHF or low-output heart failure.
- Cats with acute exacerbation of thyrotoxicosis (thyroid storm), may have collapse, weakness, dyspnea, or sudden death.

Physical Examination

- Classic signs of hyperthyroidism include evidence of weight loss and unkempt hair coat.
- Systolic heart murmur (53%–54%) or gallop heart sound may be present.
- Sinus tachycardia is often present.
- A thyroid nodule is usually palpable (83%–90% of cases), typically bilateral (two-thirds of cases).
- Normal to increased femoral pulse strength and increased strength of apical cardiac beat (precordial impulse) are also commonly identified.

Ancillary Tests

- Electrocardiography
  - Sinus tachycardia is commonly present.
  - There are tall R waves, which are suggestive of left ventricular hypertrophy or dilation.
  - Variable arrhythmias, including atrial premature complexes and VPCs, are present.
  - Uncommonly, intraventricular conduction disturbances are seen.
- Thoracic radiography: common findings include the following:
  - Generalized cardiomegaly may be present, with or without LA enlargement.
  - When CHF is present, pulmonary edema and pleural effusion are equally likely to be present.
- Echocardiography
  - Reported echocardiographic changes in cats with hyperthyroidism include increased aortic root dimension, LA enlargement, increased end-diastolic or end-systolic left ventricular dimensions, mild-to-moderate concentric hypertrophy of the LV free wall or septum, and an increased (or, rarely, decreased) left ventricular shortening fraction. Typical echocardiographic changes in current times include mild left ventricular concentric hypertrophy. SAM of the mitral valve may be present, but may increase suspicion of primary HCM.
  - There are reports of cats with myocardial failure demonstrating marked increases in left ventricular end-diastolic and end-systolic dimensions, moderate to severe LA enlargement, and a reduction in shortening fraction. The relationship of this presentation to a deficiency of the amino acid taurine is unknown, but it may also represent late irreversible changes associated with hyperthyroidism.

Therapy

- For acute CHF, see the section on treatment of CHF in the discussion of therapy in the general information section.
- Signs of CHF may be difficult to control before beginning to control the hyperthyroid state. Begin with pharmacologic manipulations, including methimazole, furosemide, and an ACE inhibitor in cats with CHF. Untreated cats should not be anesthetized or

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isolated for iodine-131 until their acute hyperthyroid state has been attenuated with medical therapy.
- In cats with asymptomatic thyrotoxicosis, therapy is aimed at controlling the hyperthyroid state (i.e., methimazole or radioactive iodine therapy). Typically ^{131}I therapy is delayed until the renal function is assessed during the euthyroid state achieved by methimazole.
- Beta-adrenergic blockade is a common recommendation in the literature. There is no contraindication to its use, but benefits have not been documented in most cases. In fact, in hypertensive hyperthyroid cats, beta blockers effectively controlled tachycardia but were ineffective at controlling hypertension in 70% of cats.
- Beta-adrenergic blockade may be considered in the following situations:
  - For the management of significant supraventricular or ventricular tachyarrhythmias.
  - In hyperthyroid cats undergoing anesthetic procedures.
  - In the treatment of suspected concurrent severe HCM. However, the addition of atenolol may be postponed until a recheck echocardiogram is done several months after the euthyroid state to assess for potential improvement in the echocardiographic abnormalities, before adding atenolol.
- Antihypertensive treatment with amlodipine (0.625–1.25 mg per cat q24h to BID, uptitrating based on follow-up BP measurements) is needed in cats showing target organ damage resulting from hypertension, in cats with persistent systolic arterial blood pressure > 180 mm Hg post-hyperthyroid treatment, or in cats with new onset of hypertension after a euthyroid state is achieved.

Prognosis

- Symptom-free cats can be managed very well without the use of specific cardiovascular therapy with appropriate therapy for the hyperthyroid state, and most evidence indicates that the cardiovascular changes are reversible. The outcome of cats with secondary cardiomyopathy (including hyperthyroidism and systemic hypertension) was good, with 80% of cats surviving over 10 years in one study.
- Many cats with CHF can be managed successfully if the hyperthyroid state is controlled, unless there is concurrent severe and unrelated primary cardiac disease.
- Most cats with severe systolic myocardial failure have a poor prognosis because the changes appear to be irreversible, unless influenced by taurine deficiency.

ACROMEGALIC HEART DISEASE

Acromegalic heart disease is a secondary cardiomyopathy caused by direct and indirect effects of increased circulating growth hormone (hypersomatotropism).

GENERAL COMMENTS

- Acromegaly is a rare disorder in cats, with between 100 and 200 cases reported worldwide to date.
- It is caused by a growth hormone-secreting tumor of the pituitary gland.
- Clinical suspicion of acromegaly is increased in cats with severe insulin-resistant diabetes mellitus.
- In a case series of 14 cats, all had insulin-resistant diabetes mellitus and enlargement of the liver, heart, kidneys, or tongue. Various cardiovascular abnormalities were seen in most of the affected cats.

PATHOPHYSIOLOGY

- The pathogenesis of heart disease in cats with acromegaly is unclear. Growth hormone causes anabolic effects of increased myocardial hypertrophy; abdominal organomegaly (liver, kidneys); excess growth of pharyngeal tissues that results in stertor; enlarged tongue; and broadening and flattening of the forehead. The growth hormone also antagonizes the effects of insulin and causes insulin-resistant diabetes mellitus.

SIGNALMENT AND PRESENTING COMPLAINTS

- Presenting complaints commonly include polyuria/polydipsia, polyphagia, and weight loss referable to uncontrolled diabetes.
- Although no breed predilections have been identified, almost all of the reported cases have occurred in older, neutered, male cats.

PHYSICAL EXAMINATION

- Systolic murmurs were noted in 9 (64%) of the 14 cats described.
- Physical features of acromegaly include prognathia inferior, organomegaly (especially kidneys and liver), increased body size, and weight gain.
- Signs of CHF may develop late in the course of the disease and have been reported in 6 (43%) of 14 cats with acromegaly, including pulmonary edema (5/6), ascites (2/6), and pleural effusion (1/6).
- Central nervous system signs (i.e., circling and seizures) caused by enlargement of the pituitary tumor may be seen (2/14 cats in one report).

ANCILLARY TESTS

- Electrocardiography: abnormalities were not detected in any of the 14 cats reported.
- Thoracic radiography: mild-to-moderate cardiomegaly was identified in 12 of 14 (86%) cats.
- Echocardiography: septal and left ventricular wall concentric hypertrophy, resembling HCM, was identified in seven of eight cats examined.
- Diagnosis of acromegaly
  - A tentative diagnosis is made based on the presence of severe insulin-resistant diabetes mellitus or renal failure in a cat with clinical features of acromegaly.

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- Documentation of a pituitary mass on computed tomographic scan or magnetic resonance imaging provides further support.
- The diagnosis of acromegaly is confirmed by demonstration of extremely high basal serum growth hormone concentrations (22–131 µg/L reported in all 14 cats in one study).

THERAPY

- In general, therapy is aimed at controlling the diabetic state and renal failure. If CHF is present, supportive care (diuretics and ACE inhibitors) may also be beneficial.
- Successful therapy for feline acromegaly has not been reported. Potential therapeutic modalities include radiation therapy, medical therapy with somatostatin analog octreotide, or hypophysectomy.
- Supportive therapy for CHF should be used in those cats with consistent clinical findings. (See the section on treatment of CHF in the discussion about therapy in the general information section.) Of the six reported cases of CHF, four of these cats died, three of which had concurrent renal failure.

PROGNOSIS

- The short-term prognosis is good. Pituitary tumors grow slowly, and neurologic signs are uncommon. Diabetes can be relatively well controlled with high doses of insulin.
- Mild-to-moderate CHF responds fairly well to symptomatic therapy.
- Most cats eventually died or were euthanized owing to refractory CHF or renal failure. Reported survival ranged from 4 to 24 months after diagnosis.

NEOPLASTIC INFILTRATION OF THE HEART

GENERAL COMMENTS AND HISTORICAL PERSPECTIVE

- Neoplastic infiltration of the heart is rare in cats.
- Echocardiography is generally required for nonsurgical detection.
- Cardiac tumors reported in cats include the following:
  - Lymphoma
  - Chemodectoma
  - Hemangiosarcoma
  - Metastatic pulmonary carcinoma
  - Metastatic mammary gland carcinoma
- Lymphoma is the most common tumor of the feline myocardium. Reported cardiac abnormalities in cats with lymphoma include complete heart block, pericardial effusion, and CHF.
- Echocardiographic findings in cats with diffuse neoplastic infiltration of the myocardium can mimic those of HCM, although often the myocardium is irregular and has abnormal echogenicity compared with normal adjacent regions of the myocardium.
- Regression of neoplastic infiltration was reported in one cat with lymphoma after treatment with combination chemotherapy.

DRUGS, TOXINS, AND PHYSICAL INJURY

- A large number of drugs and toxins are reported to cause myocardial injury in domestic animals, but very few are likely to be encountered in a clinical small animal practice. Of these, doxorubicin has received the most attention in cats.
- Decreased FS and increased left ventricular end-systolic dimensions were reported in four of six experimental cats given cumulative doses of doxorubicin of 170–240 mg/m^2. However, clinical signs of heart failure were not observed, even after a cumulative dose of 300 mg/m^2, and no cat showed ECG abnormalities during the study. As in other species, pathologic studies revealed extensive areas of myocyte vacuolization and myocytolysis. Similar clinical observations have been reported in cats with malignancies treated with doxorubicin. None had overt heart failure, and arrhythmias were only rarely observed.

INFECTIOUS MYOCARDITIS

- Infectious myocarditis is a rare myocardial disease in cats and is characterized by the presence of myocardial necrosis with or without degeneration and inflammation. Liu and associates described a syndrome of acute nonsuppurative myocarditis in 25 young cats (mean age, 2.6 years). Most cats died unexpectedly, and necropsy revealed focal or diffuse infiltration of the endocardium and myocardium with mononuclear cells and a few neutrophils. A viral cause was suspected but never identified.
- One report describes a transmissible myocarditis/diaphragmitis in cats. No organism has been isolated, but transmission between cats by injecting blood from infected cats into other cats does reliably reproduce the disease. All cats had high fever (103.8°F–105.7°F), were lethargic, and were partially anorexic. Complete blood counts and chemistries were normal in all cats for 6 weeks, except for an elevation of creatine phosphokinase in three of seven cats. The disease resolved on its own in these cats. Necropsy revealed pale 1- to 3-mm discrete foci surrounded by hemorrhage on ventricular myocardium and on the diaphragm. No clinical signs referable to the cardiovascular system were noticed.
- The relationship of endomyocarditis to the other cardiomyopathies of cats is unknown. Other reported causes of myocarditis in cats include toxoplasmosis, *Streptococcus canis* myocarditis, lymphoplasmacytic myocarditis secondary to *Bartonella* infection, and metastatic infection from sepsis or bacterial endocarditis.

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- Echocardiographic abnormalities seen in infectious myocarditis may include hyperechoic, hypertrophied regions of the ventricular myocardium, a nodular or granular appearance of the myocardium, ventricular and atrial dilation, myocardial failure, or pericardial effusion.

SUMMARY

- HCM is very common in cats and represents the largest percentage of cardiac diseases currently diagnosed in the cat.
- Nonspecific and RCMs are the next most common feline myocardial diseases and are often combined into one group because of difficulty in differentiation of the two cardiomyopathies in many cats. Treatment is likely the same for NCM and RCM.
- DCM is infrequently diagnosed, and most cases are idiopathic. Taurine deficiency-induced myocardial failure mimics DCM and must be tested for by measurement of blood taurine concentration because it is a curative disorder.
- Treatment of CHF is essentially the same for all forms of cardiomyopathy and includes furosemide, an ACE inhibitor, and often an antithrombotic agent for prophylaxis of ATE. Additional disease-specific therapies are prescribed on the basis of the underlying cardiac disease or contributing factors. Pimobendan is indicated for treatment of heart failure secondary to most forms of cardiomyopathies except hypertrophic obstructive cardiomyopathy.
- Of the secondary cardiomyopathies discussed, only nutritional (taurine-responsive) myocardial failure and thyrotoxic heart disease are encountered with any frequency. Both of these disorders have been well classified, and both respond dramatically to appropriate specific therapy. The other secondary cardiomyopathies occur infrequently and are generally poorly understood. The general approach, diagnosis, and therapy for these disorders are similar to those for other feline cardiomyopathies.
- One must recognize that the associated clinical and diagnostic findings frequently overlap, often making a definitive diagnosis difficult. Echocardiography is the one diagnostic aid that reliably allows differentiation among the different cardiomyopathies encountered in cats; however, even with a thorough ultrasound examination, distinctions may often still be unclear.

? Frequently Asked Questions

A MURMUR HAS BEEN RECENTLY INCIDENTALLY AUSCULTATED IN AN OVERTLY HEALTHY CAT. WHAT ARE APPROPRIATE RECOMMENDATIONS TO THE OWNER?

Murmurs are commonly auscultated in cats and may be caused by either pathologic abnormalities, such as underlying cardiomyopathy, or may be benign. It is impossible to distinguish the cause of the murmur by auscultation alone. The presence of a gallop heart sound or an arrhythmia further increases suspicion of underlying cardiac disease and strengthens the need for an echocardiogram. HCM is a common cause of murmurs in symptom-free cats, with prevalence rates of 18% to 62% (depending on criteria used for diagnosis of HCM) in overtly healthy cats with murmurs. An echocardiogram is recommended to evaluate the cause of a murmur in cats because some symptom-free cats have severe underlying cardiac disease, warranting treatment. If an echocardiogram is not possible, the NT-proBNP biomarker blood test is a test to assess the likelihood of moderate or severe underlying cardiomyopathy. False negatives occur with some frequency in mild hypertrophic cardiomyopathy.

DO CATS WITH SUSPECTED HEART FAILURE REQUIRE AN ECHOCARDIOGRAM BEFORE MEDICAL THERAPY IS STARTED?

Furosemide, clopidogrel, and usually an ACE inhibitor are standard medical therapy for cats with CHF. If CHF is likely according to clinical signs, auscultation abnormalities, thoracic radiographs, and positive proBNP, furosemide and an ACE inhibitor may be started without having a confirmatory echocardiogram. Ideally, an echocardiogram would be performed in the near future to refine the diagnosis and help aid in treatment decisions for additional diagnostic therapy. Pimobendan should be given to cats with heart failure who will not be further assessed with an echocardiogram, as long as a loud heart murmur is not present.

SHOULD I EMPIRICALLY TREAT A CAT WITH A NEWLY DIAGNOSED MURMUR WITH CARDIAC MEDICATIONS IF THE OWNER DECLINES FURTHER DIAGNOSTIC TESTING?

No, because the cause of the murmur is not known, empiric treatment with cardiac medications is not justified in cats with murmurs incidentally diagnosed. Murmurs may be benign or caused by underlying heart disease of many causes. Not all cats with heart disease require medications; therefore empiric treatment of a cat with a murmur is not recommended. However if the cat shows signs of CHF such as dyspnea, tachypnea, lethargy, anorexia, and weight loss, thoracic radiographs would be necessary to evaluate for presence of pulmonary edema or pleural effusion and, if present, should be treated with furosemide and likely an ACE inhibitor.

WHAT TYPE OF ANESTHETIC PROTOCOL MODIFICATIONS ARE NEEDED IN CATS WITH HEART DISEASE?

Cats with cardiomyopathy may be at risk for fluid overload, so the fluid rate is often decreased to a lower rate of 5 mL/kg/h. Anticholinergic medications should not be empirically given but may be administered if there is bradycardia. ACE inhibitors should not be given for 12 to 24 hours before anesthesia is administered because they may contribute to refractory hypotension while the patient is under anesthesia. Atenolol may be continued the morning of the procedure in patients chronically medicated with it. Typical acceptable protocols include premedication with an opioid and induction with propofol or alfaxan and benzodiazepine or midazolam. There are various risk categories for anesthesia depending on the underlying cardiac disease, severity of disease, and the presence of heart failure or significant arrhythmias. Protocol adjustments are often based on the underlying cardiomyopathy and severity. Cats with heart failure should not undergo elective anesthetic procedures, and have a high risk of adverse anesthetic events.

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SUGGESTED READINGS

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Chapter

9

Cor Pulmonale and Pulmonary Thromboembolism

Jennifer C. Chan and Lynelle R. Johnson

INTRODUCTION

Cor pulmonale is defined as right-sided heart failure caused by pulmonary disease. It is characterized by clinical signs related to fluid accumulation, or by radiographic or echocardiographic evidence of right ventricular overload. By definition, with cor pulmonale, pulmonary hypertension (PH) must be present in order for the right heart to fail. Pulmonary hypertension is defined by increased pressure within the pulmonary vasculature. Mechanisms for PH include precapillary pulmonary hypertension due to increased pulmonary vascular resistance (associated with a variety of pulmonary diseases) or increased pulmonary blood flow, and postcapillary pulmonary hypertension due to increased pulmonary venous pressure (i.e., increased left-sided heart pressure).

Pulmonary embolism is a disorder that can lead to acute or chronic PH. Pulmonary arterial obstruction can result from lodging of clot material (pulmonary thromboembolism [PTE]) or from embolization of fat, septic material, neoplastic cells, or heartworms in the pulmonary arteries or capillary bed. PTE is a complication of many commonly encountered diseases and is associated with a grave prognosis.

PHYSIOLOGY

- In health, the pulmonary vasculature is a low-pressure system with a systolic pressure of about 25 mm Hg and diastolic pressures of about 8 mm Hg. Pulmonary circulatory pressures are maintained at a level much lower than systemic pressures to reduce the workload on the thin-walled right ventricle. The pulmonary circulation maintains this low right ventricular pressure in the face of increases in pulmonary pressure and cardiac output through recruitment of closed capillaries and distension of existing capillaries. Chronic elevations of pulmonary pressures due to lung disease can lead to right-sided heart remodeling and eventually right-sided heart failure or cor pulmonale.
- Pulmonary vascular resistance is controlled by hypoxic pulmonary vasoconstriction and the balance between endothelial pulmonary vasodilators and vasoconstrictors.
  - Hypoxic pulmonary vasoconstriction works on a local level to match pulmonary blood flow to oxygenation within the lung, resulting in vasoconstriction of poorly oxygenated lung tissue. When disrupted by systemic conditions (i.e., sepsis) or by global hypoxia, diffuse pulmonary vasoconstriction can occur, leading to pulmonary hypertension and hypoxemia.
  - Examples of local pulmonary vasodilators include nitric oxide and prostacyclin, and examples of pulmonary vasoconstrictors include endothelin-1, thromboxane, and superoxide. The nitric oxide pathway is one of the most clinically important pathways in treatment of pulmonary hypertension. Nitric oxide is derived from L-arginine, and it is able to diffuse into smooth muscle where it triggers an increase in intracellular cGMP, which in turn decreases intracellular calcium. The end result is vasodilation. An imbalance in vasodilators and vasoconstrictors can lead to an increase in pulmonary arterial pressure and ventilation-to-perfusion mismatch. Modulation of vasodilation and vasoconstriction is the basis of current therapies for pulmonary hypertension.
- PTE results in abnormal gas exchange, altered vascular reactivity, changes in pulmonary mechanics, and loss of ventilatory control. Physical obstruction of large pulmonary arteries leads to increased vascular pressure and reactive pulmonary vasoconstriction caused by release of clot-associated factors such as thromboxane that increase vascular resistance. Secondary alterations in pulmonary physiology worsen and perpetuate derangements in gas exchange. Release of humoral mediators such as serotonin from platelets results in bronchoconstriction and increased airway resistance. Surfactant function is altered by embolization, resulting in loss of elastic recoil and atelectasis, decreased pulmonary compliance, and a progressive increase in right-to-left shunting. These factors, along with an expansion of alveolar dead space from an increase in nonperfused lung regions, lead to an increase in the work of breathing. Altered central control of ventilation contributes to relentless tachypnea.

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ETIOLOGY

- Cor pulmonale can result from disorders that affect the pulmonary vasculature, such as obstructive or obliterative diseases of the pulmonary circulation, or from sustained hypoxic vasoconstriction associated with chronic parenchymal or tracheobronchial disease. Rarely, an increase in pulmonary blood flow will result in PH. Not all animals with pulmonary vascular, airway, or parenchymal disorders will develop PH and cor pulmonale, and it is likely that genetic or other influences contribute to the pathophysiology. PH and cor pulmonale appear to be encountered more commonly in dogs than in cats. Primary PH is relatively uncommon; however, various pulmonary conditions can lead to secondary PH in the dog or cat, including chronic tracheobronchial disorders, pneumonia, or interstitial lung disease (Box 9.1). Often, definitive characterization of the respiratory disease resulting in PH cannot be achieved. A minority of affected animals will have overt clinical signs of right-sided heart failure (cor pulmonale).
- PTE is a secondary condition seen in association with diseases that cause stasis of blood flow, alter endothelial integrity, or increase coagulability. PTE has been linked most commonly with immune-mediated hemolytic anemia, neoplasia, sepsis, protein-losing nephropathy, cardiac disease, and hyperadrenocorticism (Box 9.2). Clinically silent pulmonary embolism occurs in a majority (82%) of dogs undergoing total hip replacement surgery. It might also occur in other diseases but could go unrecognized until signs associated with chronic thromboembolic disease or massive embolization occur. Small pulmonary thromboemboli are rapidly lysed and removed by the local fibrinolytic system; however, occlusion of larger pulmonary arteries or massive showering of emboli to a large circulatory volume can lead to acute right ventricular overload.

Box 9.1 Modified from the American College of Veterinary Internal Medicine Consensus Statement on Pulmonary Hypertension Classification.

CLASS	MECHANISM	SPECIFIC DISEASES
1	Pulmonary arterial hypertension	Idiopathic Left to right congenital cardiac shunts Pulmonary veno-occlusive disease Pulmonary capillary hemangiomatosis
2	Increased left-sided heart pressure (postcapillary)	Myxomatous mitral valve disease (endocardiosis) Mitral stenosis Aortic valve stenosis Dilated cardiomyopathy Myocarditis
3	Chronic airway, pulmonary parenchymal disease, and/or hypoxia	**Airway** Airway collapse Brachycephalic obstructive airway disorder Inflammatory airway disease Bronchiectasis **Parenchymal** Interstitial lung disease Infectious pneumonia Neoplasia **Primary hypoxia** High altitude
4	Pulmonary thromboemboli	
5	Parasitic	Heartworm Angiostrongylus
6	Multiple etiologies	

Box 9.2 Predisposing Conditions for Pulmonary Thromboembolism

Immune-mediated hemolytic anemia
Neoplasia
Sepsis
Protein-losing nephropathy/enteropathy
Cardiac disease
Hyperadrenocorticism
Central catheter use
Hemodialysis
Total parenteral nutrition
Hip replacement surgery

CLINICAL PRESENTATION

HISTORY AND CLINICAL SIGNS

- Dogs or cats with PH and cor pulmonale can be of any age, depending on the underlying cause of elevated pulmonary artery pressures. In general, there is a history of signs referable to the pulmonary system or to congestive heart failure. Animals can display any combination of signs, including lethargy, weakness, cough, respiratory distress, tachypnea, abdominal distention, and syncope, with syncope being more commonly found in PH than in other cardiopulmonary disorders. Historical features and clinical signs are not specific for PH or cor pulmonale but might suggest the underlying cardiopulmonary disease.
- PTE is generally a disorder of older animals, and history and clinical signs reflect the underlying disease process. Clinical recognition of this disorder is difficult because of nonspecific systemic and

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cardiorespiratory signs. Animals with PTE can demonstrate weight loss, lethargy, and anorexia caused by the primary disease process with acute or gradual development of tachypnea and breathlessness. Certainly, secondary PTE should be suspected in an animal with a predisposing condition (see Box 9.2) that has acute-onset tachypnea, cyanosis, or hypoxemia that is refractory to oxygen therapy.

Key Point

PTE occurs secondary to a variety of underlying conditions. Affected animals have signs reflecting the primary systemic disease along with refractory respiratory distress.

PHYSICAL EXAMINATION

- Animals with cor pulmonale will generally display tachypnea or respiratory distress caused by fluid accumulation (ascites or pleural effusion) or caused by an underlying cardiopulmonary disease. A systolic heart murmur due to mitral or tricuspid regurgitation is found in most dogs with PH and cor pulmonale. However, not all dogs have a detectable murmur. Animals that have clinical signs of overt right-sided heart failure can display jugular venous distention, hepatomegaly, ascites, or, less commonly, subcutaneous edema. Careful auscultation of the lungs is warranted to assess for underlying airway or pulmonary parenchyma disease; coarse crackles can be ausculted in conjunction with bronchomalacia or infectious or inflammatory lung disease.
- Dogs and cats with PTE have tachypnea and hyperpnea that is not alleviated by oxygen administration. Cough is relatively uncommon. Harsh lung sounds or loud bronchovesicular sounds can be detected; crackles or wheezes are less frequent. Physical examination abnormalities, such as pale mucous membranes in the case of immune-mediated hemolytic anemia or a pot-bellied appearance caused by hyperadrenocorticism, reflect the underlying disease.

DIAGNOSTIC TESTING

LABORATORY TESTING

- Basic laboratory tests generally reflect the underlying disease and do not add to the diagnosis of PH, cor pulmonale, or PTE. The diagnosis of PTE is particularly problematic. Testing for plasma D-dimer, a breakdown product resulting from the action of plasmin on cross-linked fibrin, has been shown in human medicine to have high sensitivity but low specificity for the diagnosis of PTE. In veterinary medicine, several diseases can result in a positive D-dimer test, although the magnitude of the elevation might enhance the suspicion of pulmonary embolization. However, a negative test result does not entirely exclude the possibility of PTE.
- Hypercoagulability can be assessed by performing thromboelastography, a test that assesses the kinetics of clot formation, clot stability, and clot strength. Although this test appears useful in establishing a hypercoagulable state, there is no information available on its correlation with embolization.
- Pulse oximetry and arterial blood gas analysis are useful for detecting abnormal gas exchange. Hemoglobin saturation with oxygen (S_pO_2) is related to arterial oxygen partial pressure by a sigmoidal relationship, with values above 95% indicating normoxemia. Below 95%, values for S_pO_2 lie on the exponential part of the curve, and small changes in S_pO_2 reflect very large changes in arterial oxygen. Thus pulse oximetry provides only a crude estimate of lung function while arterial blood gas analysis provides a more precise assessment of oxygenation and can be used to follow response to therapy. Arterial blood gas analysis in the animal with PTE often reveals hypoxemia, hypocapnia, and an increased alveolar-to-arterial gradient; however, normal arterial oxygenation does not exclude the diagnosis of PTE. Some animals with PTE will respond to supplemental oxygen administration with normalization of arterial oxygen levels, although many have continued tachypnea because of altered control of ventilation. Animals with additional cardiac or pulmonary pathology that increases shunt fraction will not necessarily have a complete response to exogenous oxygen supplementation.
- Heartworm testing should be performed in animals with cor pulmonale or PH.
  - For dogs, a heartworm antigen test is recommended for screening. If a high suspicion exists but heartworm antigen testing is negative, microfilariae and heat-treated heartworm antigen testing could be considered. Heat treatment is thought to cause antibody dissociation from the heartworm antigen and thus creates a test with higher sensitivity, although lower specificity. The presence of microfilariae should be assessed in any heartworm positive dog to help guide treatment recommendations. Microfilariae could be detected via examination of the buffy coat and by the more sensitive modified Knott's test. If microfilariae are present, a more severe reaction following macrocyclic lactone could occur and pretreatment with antihistamine and glucocorticoids should be considered.
  - Cats, unlike dogs, can have very transient clinical and radiographic signs when infected with heartworm. In addition, cats often have a low number of worms of only one sex; therefore feline heartworm antibody testing is recommended initially to determine exposure. Concurrent submission of heartworm antigen is also recommended and

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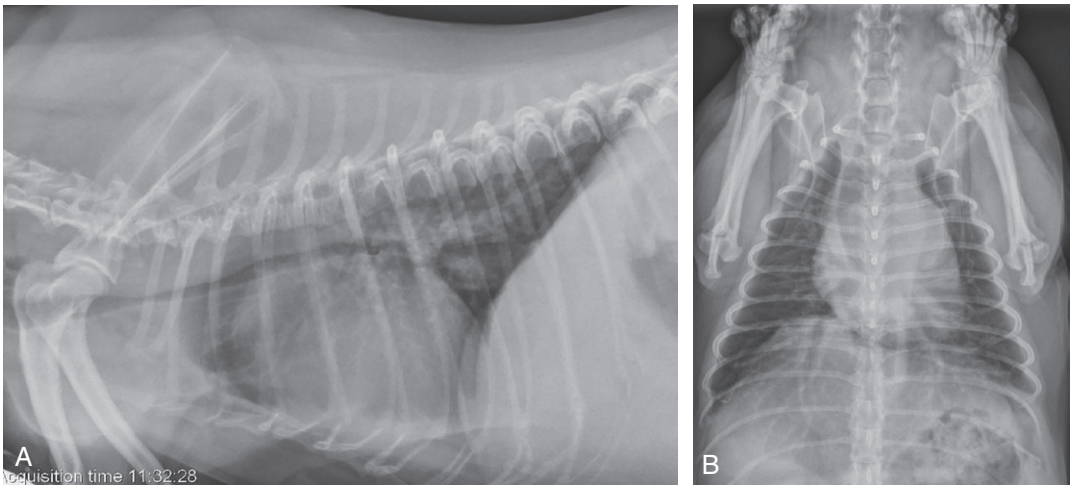


Fig. 9.1 There is generalized enlargement of cardiac silhouette on the right lateral **(A)** and dorsoventral **(B)** views in this 9-year-old Chihuahua with intrathoracic tracheal collapse and bronchomalacia. Pulmonary arteries are enlarged and do not taper appropriately. Numerous pleural fissure lines are identified, and there is a diffuse heavy interstitial and bronchial pattern identified within the lung.

carries 100% specificity. Most cats are also amicrofilaremic although a heat-treated antigen test can be considered when suspicion is high, and echocardiography can be used to confirm the diagnosis.

RADIOGRAPHS

- Radiographic evidence of right ventricular enlargement in an animal with lung or pulmonary vascular disease is supportive of cor pulmonale (Fig. 9.1). The pulmonary pattern will depend on the underlying disease process associated with PH.
- Radiographs in animals with PTE can reveal regional oligemia, lack of normal vascular tapering, or enlarged central pulmonary arteries; however, in the clinical setting, radiographic changes are rarely specific. Pulmonary infiltrates can be interstitial, alveolar, or lobar in dogs and cats. Alveolar infiltrates can represent hemorrhage, edema, or infarction. Cardiomegaly and mild to moderate pleural effusion are common in dogs and cats. Importantly, normal chest radiographs are reported in 7% to 27% of dogs and cats with necropsy-confirmed PTE, and PTE should be a top differential diagnosis in an animal with marked respiratory distress and normal thoracic radiographs.

Key Point

Normal thoracic radiographic findings in a tachypneic animal that fails to respond to oxygen administration should raise suspicion for pulmonary embolism.

ELECTROCARDIOGRAPHY

- Reported abnormalities with right ventricular enlargement caused by PH or PTE include deep S waves in leads II and aVF and a right axis deviation. Right atrial enlargement is supported by tall or peaked P waves; however, electrocardiographic evaluation of right ventricular enlargement is insensitive, and abnormalities have been reported in <15% of dogs with PH. The electrocardiogram should be closely examined for rhythm disturbances that can be found in dogs with PH.

ECHOCARDIOGRAPHY

- Two-dimensional echocardiography can provide subjective evidence of PH leading to cor pulmonale (when pulmonic stenosis has been ruled out). Supportive evidence of right ventricular overload includes right ventricular dilation and concentric hypertrophy, dilation of the main pulmonary artery, reduced distensibility of the right pulmonary artery, systolic flattening of the interventricular septum, and paradoxical septal motion. Doppler echocardiography can be used to estimate pulmonary artery pressure when tricuspid regurgitation or pulmonic insufficiency is present (Fig. 9.2). With use of the modified Bernoulli equation (pressure gradient = $4 \times$ velocity^2), spectral Doppler allows an estimation of right ventricular systolic pressure through measurement of tricuspid regurgitation velocity and estimation of pulmonary artery diastolic pressure through measurement of pulmonic insufficiency velocity, although there is substantial variation among observers and echocardiography can under or over-estimate the pressure gradient compared to catheterization. PH is suspected when the tricuspid regurgitant jet exceeds 2.8 m/s or a pulmonic insufficiency jet exceeds 2.2 m/s, although the accuracy of Doppler assessment of PH can vary substantially from measurements derived from right-heart catheterization data, at least in dogs with mitral valve disease.

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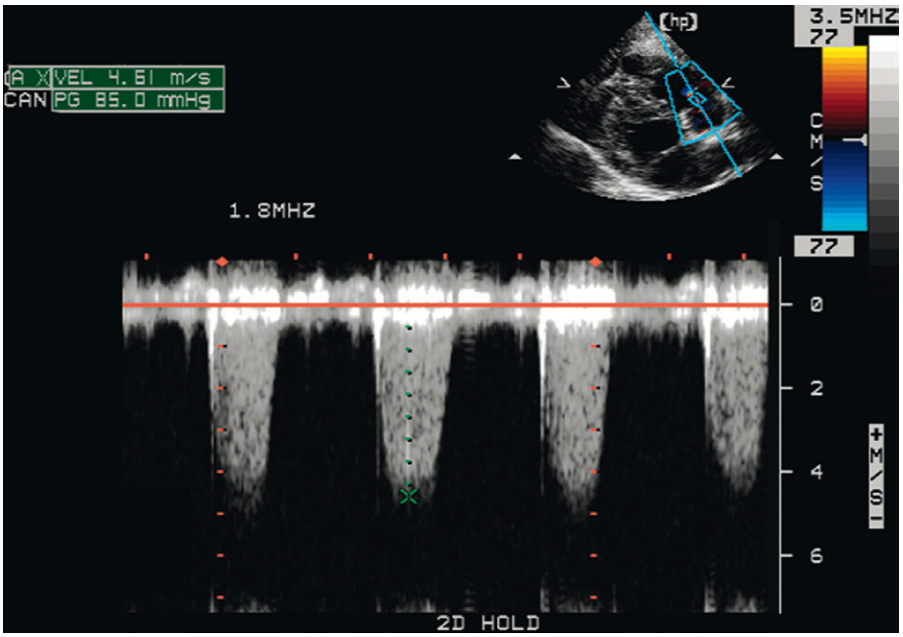


Fig. 9.2 Tricuspid regurgitant jet in a dog with pulmonary hypertension. On the basis of the modified Bernoulli equation, the right-ventricular-to-right-atrial-pressure gradient is 85 mm Hg. (Courtesy Dr. Fiona Meyers, University of Queensland.)

- Echocardiographic features consistent with pulmonary embolization overlap with those found in PH. In some cases, a thrombus can be visualized within the heart or great vessels. Therefore, echocardiography should be considered in animals with predisposing conditions for PTE that have an acute onset of respiratory distress because right ventricular dilation, pulmonary artery enlargement, or septal flattening could suggest pulmonary embolization.

AIRWAY AND PULMONARY PARENCHYMA EVALUATION

- Airway lavage allows for identification of airway-related inflammation, infection, and sometimes neoplasia. Aerobic and mycoplasma cultures are routine but anaerobic culture should be considered if increased mucus is noted, as mucus creates an anaerobic environment for bacterial growth.
  - Tracheal wash can be considered when diffuse disease is suspected because this test can be performed under heavy sedation rather than general anesthesia. A transtracheal wash is often reserved for larger dogs (>25 kg) but a sterile transoral endotracheal wash can be performed on animals of any size.
  - Bronchoalveolar lavage requires more expertise and requires general anesthesia. Bronchoalveolar lavage is recommended when disease is segmental, and collecting two to three washes from different lung lobes is recommended.
  - Bronchoscopy is recommended to assess for airway collapse and bronchiectasis as causes for PH when anesthesia is considered a viable option for the animal.
- Lung biopsy is indicated to investigate causes of PH that cannot be readily detected using the diagnostics described, such as interstitial fibrosis and vaso-occlusive diseases (i.e., pulmonary veno-occlusive disease or pulmonary capillary hemangiomatosis).

Key Point

Echocardiography should be considered in dogs suspected of having PTE.

CATHETERIZATION AND ADVANCED IMAGING

- Direct measurement of pulmonary artery pressure through right-heart catheterization is the gold standard for diagnosing PH. Unfortunately, it requires sedation or general anesthesia, and animals with PH are at increased risk for anesthetic complications. Therefore, catheterization is rarely performed in the clinical situation.
- Pulmonary angiography has been considered the gold standard for diagnosis of PTE in humans, and use of contrast helical CT to confirm PTE is increasing. Definitive angiographic diagnosis of PTE depends on visualization of an intraluminal filling defect in a pulmonary artery or loss of visualization of an artery. However, neither modality is commonly used in veterinary medicine because each requires anesthesia.
- Ventilation/perfusion scanning uses technetium-99m-labeled macroaggregated albumin as a vascular marker and technetium-99m-labeled diethylenetriamine penta-acetic acid as a ventilatory marker to define segmental or lobar perfusion defects in areas

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of normal ventilation. Ventilation scans are rarely performed in nonanesthetized animals; however, perfusion scanning alone can be completed without anesthesia and can assist in documentation of perfusion deficits. This is a safe, noninvasive technique for evaluation of PTE; however, it is somewhat nonspecific because perfusion deficits could reflect thrombosis or simply a lung region experiencing hypoxic pulmonary vasoconstriction.

THERAPY

GENERAL TREATMENT OF PULMONARY HYPERTENSION

- Treatment of PH includes (1) directly addressing PH to alleviate clinical signs, (2) treating the underlying cause, and (3) minimizing cardiac remodeling.
- Not all dogs and cats with PH are treated. Treatment is sometimes recommended only in animals that have clinical signs of disease (i.e., those with syncope and exercise intolerance) or those with more moderate to severe right-sided heart remodeling.
- Direct treatments for PH:
  - Phosphodiesterase-5 inhibition
    - Sildenafil (Viagra): Sildenafil is a phosphodiesterase-5 inhibitor and is often the first line of treatment for PH. Sildenafil works by inhibiting breakdown of intracellular cyclic guanosine monophosphate (cGMP), the second messenger for nitric oxide; an increase in cGMP leads to an increase in arterial relaxation. The starting dose is 1 mg/kg q8–12h and can be escalated up to 3 mg/kg q8h. Sildenafil has been shown to be effective in dogs with various respiratory etiologies of PH. Response to therapy is often based on improvement in clinical signs rather than changes in echocardiographic Doppler gradients. Close in-hospital monitoring after starting sildenafil is recommended in dogs with PH caused by left-sided heart disease because increased pulmonary edema can develop following use of this therapy. Sildenafil is generally well tolerated, but gastrointestinal effects and cutaneous flushing can be noted.
    - Tadalafil (Cialis): Tadalafil is a long-acting, more selective phosphodiesterase-5 inhibitor compared to sildenafil. Due to its long-acting nature, it can theoretically be administered once daily in dogs. Tadalafil reportedly has less P-glycoprotein inhibition in comparison to sildenafil, which in theory minimizes adverse effects. Tadalafil decreased pulmonary arterial pressure in beagles with induced pulmonary arterial hypertension, and in one study of 23 dogs, a dose of 2 mg/kg PO q24h improved quality of life in dogs with severe PH, although it did not demonstrate superiority compared to sildenafil based on owner perception and peak tricuspid regurgitant flow velocity. Adverse effects were minimal but included hyporexia.
  - L-Arginine: L-arginine is a substrate for nitric oxide synthesis and decreases pulmonary arterial pressures and pulmonary vascular resistance in humans. A dose of 100 mg/kg PO q8h of L-arginine has been shown to increase plasma arginine concentrations but improvements in PH in dogs have not been demonstrated.
  - Pimobendan: Pimobendan is a phosphodiesterase-3 inhibitor and calcium sensitizer that acts an inodilator.
    - Pimobendan increases both left and right ventricular stroke volume and lessens precapillary pulmonary hypertension in a canine model. Pimobendan should be considered in dogs with left-sided heart disease prior to sildenafil because it increases cardiac contractility, improving both left-sided cardiac output and PH.
    - Combination therapy of sildenafil with pimobendan did not increase survival time in a clinical cohort of dogs with severe PH due to chronic pulmonary disease in one retrospective study but additional studies are warranted.

TREATMENT OF PULMONARY THROMBOEMBOLISM

- Definitive treatment of PTE is challenging, and the decision to pursue aggressive therapy is complicated by the fact that the diagnosis of embolization is difficult to confirm antemortem. Agents that cause clot lysis locally (tissue plasminogen activator) or systemically (streptokinase, urokinase) are expensive, require intensive monitoring, and can be associated with complications. In addition, efficacy is questionable. Ancillary therapies, including control of the primary disease process and oxygen supplementation, are essential in management. In practice, effort is directed toward preventing the occurrence of PTE through prophylactic therapy with drugs such as clopidogrel and low-molecular-weight heparin or rivaroxaban (see Frequently Asked Questions).

CONCLUSION

- PH should be suspected in any dog with cardiopulmonary disease that exhibits excessive lethargy, exercise intolerance, and particularly syncope.
- Once PH has been identified by echocardiographic assessment in a dog lacking left-sided heart disease, a respiratory workup is warranted to identify the underlying etiology.
- Current treatments for PH focus on modulating local endothelial vasoconstriction-vasodilation balance.

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- Pulmonary thromboembolism remains a challenging antemortem diagnosis, and prophylactic therapy in diseases associated with a high risk for PTE is warranted.

Frequently Asked Questions

WHAT CLINICAL FINDINGS WOULD SUPPORT THE DIAGNOSIS OF COR PULMONALE, AND HOW COULD THIS BE CONFIRMED?

Animals with clinical signs relative to cor pulmonale generally display respiratory abnormalities (tachypnea, hyperpnea, and/or cough) and also exhibit signs of right-sided heart failure (ascites, jugular venous distention, and/or subcutaneous edema). A history of syncope should raise suspicion for PH. Radiographically, cor pulmonale is evident as right-sided heart enlargement. Right atrial enlargement and dilation of the caudal vena cava support the diagnosis. Two-dimensional echocardiography reveals eccentric dilation of the right ventricle most commonly. With chronic PH or PH in a young animal, right ventricular hypertrophy can be noted. Doppler echocardiography can enhance suspicion of PH by detection of a velocity jet >2.8 or 2.2 m/s in the presence of tricuspid regurgitation or pulmonic insufficiency, respectively.

WHAT TESTS CONFIRM THE DIAGNOSIS OF PTE AND PROVIDE SUPPORT FOR INSTITUTION OF ANTICOAGULANT THERAPY?

Unfortunately antemortem diagnosis of PTE remains challenging, and definitive diagnosis is often not achieved in the clinical setting. Suspicion for PTE should be present when an animal with a recognized predisposing condition (see Box 9.2) develops acute onset of respiratory distress. Normal thoracic radiographic findings do not preclude the diagnosis. Supportive evidence of PTE would include a positive D-dimer test, echocardiographic evidence of acute right ventricular overload, and perfusion deficits on pulmonary scintigraphy in an animal with refractory respiratory distress. Anticoagulant therapy with low-molecular-weight heparin or rivaroxaban is often instituted in a patient with these findings to limit further deposition of clot material and to enhance clot breakdown. Thrombolytic therapy is rarely used because of the risk of generating a systemic fibrinolytic state or creating ischemia-reperfusion injury. Because of the difficulty in establishing a diagnosis of PTE and the morbidity and mortality associated with this secondary complication, prophylactic antithrombotic therapy should be considered in animals with recognized predisposing conditions.

WHAT DRUGS CAN BE USED AS PROPHYLAXIS FOR PTE?

The drug historically used to inhibit platelet aggregation is aspirin, although the dose that achieves this effect has not been clearly identified and not all animals will exhibit reduced platelet function due to genetic differences in drug metabolism. Newer drugs, such as clopidogrel, have some effect on platelet aggregation, although the precise dosing in dogs is unclear and efficacy in PTE has not been established. Although not evaluated in the setting of PTE, clopidogrel at a dose of 18.75 mg per cat appears to decrease the risk of aortic thromboembolism in animals with cardiomyopathy. Low-molecular-weight heparin (enoxaparin, dalteparin) acts through inhibition of coagulation, with enhanced factor X versus factor II inhibition compared with unfractionated heparin. This class of drug can be used concurrently with antiplatelet drugs; however, the pharmacokinetics are not fully understood and pharmacodynamic studies in PTE are lacking.

WHAT TYPE OF TREATMENT CAN BE CONSIDERED FOR PULMONARY HYPERTENSION?

Aggressive management of underlying cardiopulmonary conditions should be instituted. Animals with chronic bronchitis or small airway collapse often require corticosteroids (oral or inhaled) and extended-release theophylline (10 mg/kg PO q12h [dog] or 15–19 mg/kg PO in the evening [cat]). Interstitial lung diseases are less likely to respond to medical therapy. In either group of animals with respiratory dysfunction, supplemental oxygen therapy either at home or in the hospital can improve clinical presentation. Use of sildenafil (1–3 mg/kg PO q8–12h) or tadalafil (2 mg/kg/d) can be considered for animals with signs related to PH. Clinical signs and quality of life are improved with therapy, although echocardiographic estimates of pulmonary artery pressure can remain unchanged.

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Chapter 10

Heartworm Disease

Justin D. Thomason

Heartworm infection, primarily in the dog and cat, has been reported in many countries and occurs throughout the United States. Infections are most common in tropical and semitropical regions, and the risk is greater in dogs that live outdoors. Most infected dogs are medium to large breeds and 3 to 8 years of age at the time of diagnosis.

Numerous mosquito species serve as vectors after acquiring microfilariae (larval stage 1, L_1) while feeding on a canine host. Cats living either indoors or outdoors are infected by mosquitoes that have acquired microfilariae from infected dogs. The infection rates in cats are lower than dogs, although in some regions the infection rates in cats are higher than expected.

Development of microfilariae (L_1) to infective larvae (L_3) within the mosquito occurs in 1 to 4 weeks, requiring less time as the ambient temperature increases. Infective larvae are deposited on the host's skin as the mosquito feeds and the larvae enter the bite wound in small numbers, usually less than 10 larvae.

In dogs, infective larvae (L_3) migrate through host tissue while molting (to L_4 and L_5), and L_5 (1–2 cm in length) arrive in the pulmonary arteries in approximately 100 days. Adult worms develop largely in the caudal lobar arteries by 150 to 200 days after L_3 inoculation. Adult *Dirofilaria immitis* are long (approximately 15 cm, males; 25 cm, females) nematodes that reside primarily in the pulmonary arteries but can move into the right ventricle when the worm burden is high. Microfilariae (L_1) are produced by gravid females 6 to 7 months after infection.

Microfilariae (L_1) are usually detectable in infected dogs not receiving monthly or injectable prophylaxis. However, in highly endemic regions, from 25% to 50% of infected dogs do not have circulating microfilariae because they are entrapped in the pulmonary capillaries by host immune mechanisms. The number of circulating microfilariae does not correlate to adult heartworm burden. Adult heartworms can live 3 to 5 years, while microfilariae (L_1) live for 1 to 3 years.

Most dogs are highly susceptible to heartworm infection, and most infective larvae (L_3) develop into adults. A lower percentage of cats inoculated with L_3 have adult infections, and the burden is often only one to three worms, but it can be higher in highly endemic regions. Further evidence of resistance in cats is the short survival time of many L_5 in the pulmonary arteries; adult worms probably survive no longer than 2 years. Another difference between heartworm disease in dogs and cats is that in the cat, larvae are more likely to migrate to ectopic sites such as the brain, systemic arteries, and subcutaneous (SC) sites.

Key Point

Adult heartworms are present in the pulmonary arteries approximately 6 months after transmission from the mosquito.

PATHOPHYSIOLOGY

- Heartworms damage the pulmonary arterial endothelial lining within 3 days of experimental heartworm transplantation. Pulmonary arteries containing heartworms have endothelial swelling, widened intercellular junctions, sloughing of longitudinal strips of endothelium, and adhesion of many activated leukocytes and platelets to the damaged areas. Endothelial damage decreases nitric oxide production and increases endothelin production (endothelial dysfunction). Endothelial dysfunction also increases permeability to serum proteins and water, permitting leakage of small arterioles into the perivascular interstitium. Trophic factors are released by damaged endothelium and activated platelets and leukocytes. These factors (such as platelet-derived growth factor) stimulate migration and multiplication of smooth muscle cells within the tunica media. Endothelin causes vasoconstriction and vascular fibrosis. Pathognomonic villi of heartworm disease consist of the rapidly dividing smooth muscle cells and the collagen produced by them.
- Arteriographic abnormalities are seen within weeks of heartworm transplantation into dogs. Lobar arteries, especially those to the caudal and accessory lung lobes, dilate, become tortuous, and develop aneurysms. Smaller distal arteries lose their normal tapering and may be occluded. Arteries with a diameter smaller than the adult heartworms frequently appear to be abruptly pruned. Blood flow is frequently obstructed and diverted to nonaffected lung lobes.

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- Increased permeability of the vascular surfaces of smaller vessels produces perivascular fluid infiltration, which can be seen as an interstitial pattern coalescing to an alveolar pattern on radiographs. These changes are probably a combination of vascular disease, perivascular fluid leakage, and inflammation.
- The severity of cardiopulmonary disease is determined by worm numbers, host immune response, duration of infection, and host activity level. Active dogs have more disease than inactive dogs for any given worm burden. Sedentary dogs can harbor more than the typical 10 to 20 worms associated with infections in endemic regions without developing clinical signs. However, frequent exertion/activity increases pulmonary arterial disease and can precipitate clinical signs and even right-sided congestive heart failure (CHF). Pulmonary disease is also exacerbated by natural or drug-induced worm death. High worm burdens are most often the result of inoculation from many mosquitoes over one season. Very large exposure over a short time span can result in caval syndrome the following year, typically in young dogs. Decreased flow and inadequate recruitment of collateral arterioles result in inadequate pulmonary flow in response to exertion. Increased blood flow/volume into nonaffected lung lobes contributes to the development of pulmonary hypertension (PH). PH increases right ventricular afterload leading to right heart hypertrophy/dilation and right-sided CHF (especially in active dogs). Right-sided CHF is characterized by high central venous pressure, hepatic congestion, and ascites. In general, small dogs tolerate infections and treatment less well than large dogs.
- Heartworm-associated inflammatory mediators induce immune responses in the lungs and kidneys (immune-complex glomerulonephritis). Leakage of plasma and inflammatory mediators from small vessels and capillaries causes parenchymal lung inflammation and edema. Pulmonary arterial constriction causes increased flow velocity, especially with exertion, and resultant shear stresses further damage the endothelium. The process of endothelial damage, vasoconstriction, increased flow velocity, and local ischemia is a vicious cycle. Inflammation with ischemia can result in irreversible interstitial fibrosis.

PATHOPHYSIOLOGY IN CATS

- Pulmonary arterial disease in the cat is similar to that in the dog, although small arteries develop more severe muscular hypertrophy. Arterial thrombosis is caused by both blood clots and worms lodged in narrow lumen arterioles.
- Immature heartworms can reach the lungs of cats within 100 days, at which time pneumonitis develops. Clinical signs are coughing and wheezing, which resemble asthma and usually improve spontaneously. The feline heartworm antibody test usually becomes positive at this time. Subsequently, when L_5 mature to adult heartworms 6 to 7 months after infection, cats usually begin to experience episodic coughing and dyspnea. As worms die, an intense pneumonitis develops, and even one dead worm can produce severe pneumonitis, thromboembolism, and death.
- The worst pulmonary disease is seen after adult heartworms have died and their fragments are swept distally into the small pulmonary arteries. The response at the pulmonary arterial surface is an exacerbation of that seen with live heartworms. Villous proliferation is exuberant, and there are thrombi and a granulomatous inflammatory reaction around the dead heartworms. Blood flow is severely reduced, sometimes with no pulmonary arterial flow going to the caudal lung lobes. The caudal lung lobes frequently consolidate and fail to function as blood gas exchangers.

CLINICAL SYMPTOMS

- Clinical signs are a reflection of the number of infecting heartworms, the duration of infection, and the response of the canine host. Most dogs are symptom free. Coughing, tachypnea, and dyspnea are the most common signs and are usually associated with parenchymal disease in the caudal lung lobes. This disease is focused around the pulmonary arteries, with inflammatory fluids accumulating, owing to increased vascular permeability to serum proteins and fluids.
- Decreased ability to exercise and the sequence of right ventricular dilation, hypertrophy, and failure are the responses to fixed vascular resistance and PH. This fixed resistance impedes arterial flow and increases the work of the right ventricle. Severe disease restricts the ability to recruit arteries to transport the high blood flow needed for exercise and decreases the ability to exercise. This resistance to flow is complicated by the exaggerated hypertensive response to alveolar hypoxia (arteriolar constriction) that accompanies heartworm infection. Patients with heart failure frequently have had increased exercise before presentation and demonstrate ascites and hepatomegaly. Cardiac cachexia develops within a few weeks after the development of right-sided CHF.
- Hemoptysis may occur, especially after the heartworms have been killed by adulticide, and is the result of vascular and airway wall rupture. This blood loss normally starts in an area of severe vascular and parenchymal disease in the caudal lung lobes, probably when coughing ruptures vascular and airway walls.

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DIAGNOSIS

- Monthly oral prophylaxis induces embryo stasis, and serologic antigen testing is required for diagnosis. Antigen detection tests are all very sensitive and specific. Infections acquired during the previous mosquito season are first detected by antigen testing 7 to 8 months later. The level of antigenemia is directly related to the number of mature female worms that are present. At least 90% of dogs harboring three or more adult females will test positive. For low-worm-burden suspects, a commercial laboratory-based microwell titer test (DiroCHEK by Synbiotics, Kansas City, Missouri) is most sensitive. In general, strong, quick, positive reactions correlate with relatively high worm burdens. However, strong reactions also occur following worm death.

ANCILLARY TESTS

Echocardiography

- In dogs, echocardiography is relatively unimportant as a diagnostic tool unless caval syndrome and/or PH are suspected. Worms seen in the right heart and vena cava are associated with high-burden infection and with caval syndrome. With severe disease, changes associated with PH are often present. The following are a number of abnormalities on the echocardiogram that support a diagnosis of PH:
  - Underloaded left ventricle
  - Underloaded pulmonary veins
  - Underloaded left atrium
  - Right ventricular dilation with hypertrophy
  - Right atrial dilation
  - Dilated pulmonary trunk and arteries
  - Flattening of the interventricular septum
  - Paradoxic septal motion
  - High-velocity tricuspid regurgitation
  - High-velocity pulmonic regurgitation
  - Upward diastolic billowing of the pulmonic valve leaflets
  - Prolonged duration from initial to maximum right ventricular outflow tract systolic flow velocity
  - Decreased tricuspid annular plane systolic excursion

Electrocardiography

- Usually the electrocardiogram of a heartworm-infected dog is normal. Right ventricular hypertrophic patterns are seen when there is severe, chronic PH and are associated with overt or impending right-sided CHF. Heart rhythm disturbances are usually absent or mild, but atrial fibrillation is an occasional complication of severe right atrial dilation (especially in large breeds).

DIAGNOSIS OF FELINE HEARTWORM INFECTION

- The diagnosis is based on historical and physical findings, index of suspicion, echocardiography, and serologic findings. Cats may have a positive antigen test result 8 months after L_3 inoculation. However, less than 50% of infected cats will test positive if they have less than three adult female worms or immature infections. Anti-heartworm antibodies are produced by 90% of infected cats and may first appear 2 to 3 months post-L_3 infection and are usually present by 5 months. Antibodies can persist for at least 6 months after worm death. Also, antibodies induced by larvae can persist after macrolide prophylaxis has been instituted and has killed the pre-adults. Thus a positive antibody test result is consistent with L_4 and L_5 larvae, adult infection, or persisting antibody following death of larvae or adults. A negative heartworm antibody test result indicates a 90% probability of absence of infection. Microfilaremia is unusual and typically transient in cats.

Key Point

The combination of vomiting, eosinophilia, and hyperglobulinemia warrant a high index of suspicion of heartworm infection in cats.

Echocardiography

- In cats, echocardiography is useful because worms can usually be imaged. Parallel white echogenic lines may be seen in the right heart and pulmonary arteries. The best view is obtained in the right parasternal short-axis angulated to image the right and left main pulmonary arteries. High worm burdens may be associated with worms in the right heart.

MICROFILARIAL TESTS IN DOGS

- The problems with microfilarial detection tests are related to their sensitivity and the incidence of occult or non-microfilaremic infections.

DIRECT SMEAR

- This is a simple test wherein a drop of anticoagulated blood is placed on a slide and examined under the microscope. The test is quick and simple and, if microfilariae of *D. immitis* are detected, the diagnosis of heartworm disease is made. A positive test result eliminates the need for a microfilarial concentration test. However, the following are shortcomings of the direct smear:
  - It is 20% to 25% less sensitive in detecting microfilariae than the concentration tests (i.e., false-negative result).
  - It will be negative in the 25% to 50% of heartworm infections wherein circulating microfilariae are absent (so-called occult or amicrofilaremic infections).

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Concentration Tests

- Although a drop of blood is usually held back for the direct smear (which, if positive, means that the more time-consuming and more expensive concentration test does not have to be performed), the direct smear test result is usually negative because most dogs are not infected. A negative direct smear test result does not rule out heartworm infection.
- The two types of concentration tests are the modified Knott test and the filter test. Both are of similar sensitivity, specificity, cost, and labor. The choice between the two is largely one of tradition, habit, or practice. Both tests require that the examiner be able to distinguish the microfilariae of *D. immitis* from *Acanthocheilonema reconditum*. This challenge may be more difficult with the filter tests. Between 25% and 50% of infected dogs lack circulating microfilariae and thus concentration tests often fail to identify infection.

False-Negative Antigen Tests in Dogs

- Although false-negative antigen test results occur, the incidence is less than that of microfilaria tests and occult infections. False-negative results may be caused by the following:
  - Infections <7 months after L_3 inoculation
  - Few worms (<3)
  - No female worms
  - Examiner interpretation
- In most cases, false-negative test results are associated with early or mild infections; microfilarial test results will also be negative, and radiographic changes will be absent or minimal.
- Heartworm-infected dogs living in highly endemic regions have a higher worm burden than infected dogs in regions where infections are less common. Heartworm-infected dogs that have never received prophylaxis have higher worm burdens than those that have been receiving prophylaxis. The heartworm burden can be estimated by the degree of antigenemia, and this burden is often directly proportional to the severity of disease. The potential utility of worm-burden estimate is that the risk of postadulticide thromboembolic complications is greater in high heartworm burden infections. Nonetheless, thoracic radiographs are probably more predictive of this complication risk, and antigen level alone is not to be used to assess the risk of pulmonary thromboembolism. As previously mentioned, small-breed dogs and cats have a greater risk of thromboembolic complications likely related to the naturally smaller pulmonary arteries, regardless of worm burden.

MINIMUM DATABASE

- Thoracic radiography is the single test that provides the most information as to the severity of disease. All dogs with symptomatic infections have visible abnormalities. Dogs with severe disease have a worse prognosis and require an alternative treatment protocol.
- Complete blood count.
- Serum chemistry profile.
- Urinalysis.

THORACIC RADIOGRAPHS

In dogs, the thoracic radiography is the single test that provides the most information as to disease severity. Attention should be given mostly to the main pulmonary artery segment (the 1 o'clock position on the ventrodorsal [VD] or dorsoventral [DV] projection) and the caudal lobar pulmonary arteries. The latter are best evaluated on the DV projection. Severe disease is characterized by a large main pulmonary artery segment and dilated, tortuous caudal lobar pulmonary arteries. If the latter are twice the diameter of the ninth rib at their point of superimposition, then the infection has produced severe disease. Normally these arteries are not larger than the ninth rib.

Fluffy, ill-defined parenchymal infiltrates of variable extent often surround the caudal lobar arteries and are usually worst in the right caudal lobe. The infiltrate may improve with cage confinement with or without antiinflammatory dosages of a corticosteroid.

In cats, the caudal lobar arteries normally appear relatively large but are larger still with heartworm infection. Patchy parenchymal infiltrates may also be present in cats with respiratory signs. The main pulmonary artery segment usually is not visible because of its relatively midline location.

COMPLETE BLOOD COUNT

- Eosinophilia and basophilia are common during heartworm infection and other more common parasitic infections. *A. reconditum* infection produces an eosinophilia of even greater magnitude than *D. immitis* infection. Anemia is a marker of severe heartworm disease, especially when secondary to caval syndrome.

SERUM CHEMISTRIES

- Serum chemistries are often unremarkable with uncomplicated heartworm infection. However, hyperglobulinemia can be present with chronic heartworm infections, and hemoglobinemia is often evident with caval syndrome and less often with thromboembolism. Increased blood urea nitrogen and serum creatinine may be the result of dehydration. However, both renal amyloidosis and immune-complex glomerulopathy eventually cause azotemia.

URINALYSIS

- Proteinuria is common in animals with severe and chronic infections and is caused by immune-complex glomerulonephritis or amyloidosis.

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Hemoglobinuria is evident with caval syndrome or severe lysis associated with pulmonary thromboembolism or microangiopathy (severe, class 3, disease).

Key Point

Thoracic radiography is the most important diagnostic test for determining the severity of heartworm disease.

CLASSIFICATION OF HEARTWORM DISEASE

- Class 1
  - No or minimal signs
  - Normal or equivocal radiographic abnormalities
  - Absence of anemia
  - Absence of thrombocytopenia
  - Absence of hemoglobinuria
- Class 2
  - Mild to moderate symptoms
  - Symptoms usually are intermittent
- Class 3
  - Cough often severe and at rest
  - Weight loss-unthrifty
  - Syncope
  - Exercise intolerance
  - Ascites (right-sided CHF)
  - Anemia
  - Thrombocytopenia
  - Hemoglobinuria

THERAPY

- Before adulticide administration, it is recommended to initiate doxycycline (5–10 mg/kg q12h) for approximately 4 weeks in combination with a monthly macrocyclic lactone. The aim of doxycycline treatment is to eliminate *Wolbachia*, a gram-negative, intracellular, intrafilarial bacterium that is believed to incite pulmonary vasculature and possibly renal inflammation. This inflammation is believed to contribute to heartworm death-associated clinical signs. *Wolbachia* organisms are largely eliminated by this treatment and are unlikely to reappear in less than 3 months. The concentration of microfilaria, if present, will also be reduced. If doxycycline is not available then minocycline (5–10 mg/kg q12h) may be used.
- It is also recommended to initiate a monthly macrocyclic lactone, such as ivermectin, at the time that doxycycline treatment is started. Not only will the macrocyclic lactone gradually eliminate microfilariae (L_1), but also, the combination of doxycycline and a macrocyclic lactone is believed to kill some of the adult heartworms. Many microfilariae are removed from the circulation, and those that are ingested by mosquitos and subsequently molt produce L_3 that are noninfectious. The monthly macrocyclic lactone will close a window of opportunity for reinfection. In addition, during the 1 month of doxycycline and macrocyclic lactone treatment, more melarsomine-resistant immature worms will mature to melarsomine-sensitive age (at least 4 months after L_3 infection).
- Melarsomine should be administered to most infected dogs. The first injection is recommended after 4 weeks of doxycycline and macrocyclic lactone treatment. For all class 1 and 2 patients, one injection of melarsomine is administered intramuscularly at 2.5 mg/kg, deep in the epaxial musculature with a 22-gauge needle (1 in. for large dogs). After drawing up the dose of melarsomine, place a new needle on the syringe. Pressure is applied during and for 1 minute after the needle is withdrawn to prevent the drug from seeping into SC tissues. Approximately 1 month later, two injections of melarsomine (by the previous method) separated by 24 hours are administered. These two injections are administered on alternate sides. This protocol is sometimes referred to as graded kill.
- After each melarsomine injection, patients must be kept strictly confined and inactive for 4 to 6 weeks to minimize the risk of pulmonary thromboembolism. The adverse effects of melarsomine are otherwise limited to local inflammation, brief low-grade fever, and ptyalism. Hepatotoxicity is a rare complication.
- We have encountered a high proportion of toy and miniature breed dogs that experience severe pulmonary thromboembolic complications after the double melarsomine injections (third and fourth injections). It is our practice to administer melarsomine as single injections 4 to 6 weeks apart to a total of three injections to toy and miniature dogs. Postadulticide thromboembolic complications have been less with this protocol.
- Class 3 patients should be stabilized before melarsomine administration. However, doxycycline and a macrocyclic lactone treatment should be initiated if possible. Stabilizing treatment variably includes cage confinement, oxygen, corticosteroid (prednisone, 1 mg/kg once daily) and heparin (100–300 U/kg SC q6-8h for 1–3 weeks before the graded-kill melarsomine protocol). Consider sildenafil (2.0 mg/kg q8h) to reduce pulmonary hypertension and improve pulmonary perfusion. Sildenafil (20-mg tablets) is commercially available at reasonable cost.
- Some class 3 patients have overt right-sided CHF (hepatic congestion and ascites). These patients require additional drugs to reduce pulmonary hypertension, increase right ventricular contractility, suppress renin-angiotensin-aldosterone activity, and reduce sodium and fluid overload. The labor intensiveness and numbers of drugs administered to these patients is daunting. Clinical judgment is required.

Key Point

Use the graded-kill melarsomine protocol.

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PATIENTS WITH RIGHT-SIDED CONGESTIVE HEART FAILURE

- Dogs with ascites may need to be treated with furosemide (1–2 mg/kg q12h), low-dose angiotensin-converting enzyme (ACE) inhibitor such as enalapril (0.25 mg/kg and possibly increased to 0.5 mg/kg q12h after 1 week), sildenafil (2 mg/kg q8h), and pimobendan (0.2–0.3 mg/kg q12h). Spironolactone (1–2 mg/kg q12h) may be used in combination with furosemide. Digoxin, digitoxin, and arteriolar dilators, such as hydralazine and amlodipine, should not be administered. Digoxin is not effective for cor pulmonale, and arteriolar dilators (occasionally even ACE inhibitors) are likely to cause systemic hypotension. In our experience, treatment of the PH and cage confinement will often result in resolution of the right-sided CHF.

POSTADULTICIDE THROMBOEMBOLIC COMPLICATIONS

- Pulmonary thromboembolism can occur from 2 to 30 days after treatment, and signs are most likely from 10 to 21 days after treatment. Possible clinical signs include coughing, hemoptysis, tachypnea, dyspnea, lethargy, and anorexia. Physical findings variably include pale or gray mucous membranes, pulmonary crackles, and pyrexia. Abnormal laboratory data may include an inflammatory leukogram, thrombocytopenia, and prolonged activated clotting time or prothrombin time. Local or disseminated intravascular coagulation may be comorbidities. Treatments to consider include oxygen, cage confinement, prednisone at an antiinflammatory dosage (1.0 mg/kg q24h), heparin (100–300 U/kg SC q6-8h), and/or antiplatelet therapy (aspirin or clopidogrel) for several days to 1 week. Aspirin (5–7 mg/kg q24h) or clopidogrel (2–4 mg/kg q24h) is less labor intensive than heparin treatment but may be less effective.

CONFIRMING ADULTICIDE EFFICACY

- The graded-kill melarsomine protocol kills all or most worms in approximately 75% of dogs. Antigen testing is performed 4 to 6 months after the third dose of the graded-kill protocol. Many test results will be negative at 4 months, but some will become negative only after 6 months. A strongly positive test result after 6 months should be followed by retreatment (two injections 24 hours apart).

EFFECT OF IVERMECTIN ON ADULT HEARTWORMS IN DOGS

- Ivermectin administered monthly for 2 to 3 years to dogs beginning at 5 to 7 months after L_3 inoculation eradicates most adult worms. The addition of doxycycline (5–10 mg/kg q12h) for 4 weeks provides more rapid adulticide activity than ivermectin alone. However, the use of ivermectin is not a substitute for melarsomine treatment because the "slow-kill" may allow pulmonary disease to progress in the interim. We have encountered some disastrous outcomes in dogs treated in this fashion. In addition, this treatment may contribute to macrocyclic lactone resistance (see the following on lack of efficacy).

TREATMENT OF FELINE HEARTWORM INFECTION

- There is no current satisfactory approach to heartworm infections in cats. Although infection often is lethal, a safe and effective melarsomine protocol has not been developed. Thus all cats in heartworm-endemic regions should receive drug prophylaxis. The adult heartworm lifespan in cats is probably no longer than 2 years. Cats may remain symptom free, experience episodic vomiting or episodic dyspnea (resembling asthma), may die suddenly from pulmonary thromboembolism, or, rarely, have CHF. With each heartworm death, pulmonary complications occur.
- Cats are managed conservatively with restricted activity and corticosteroid therapy, such as prednisone or prednisolone (1.0–2.0 mg/kg q24h). Steroids reduce the severity of vomiting and respiratory signs. The hope is that episodes of pulmonary complications will not prove fatal as the worms die. Barring superinfection, it is thought that 25% to 50% of cats can survive with this approach. Serial antibody testing can be used to monitor heartworm status.
- Surgical retrieval of worms from the right atrium, right ventricle, and vena cava can be attempted in patients with high worm burden detected by echocardiography. An endoscopic basket or horsehair catheter can be advanced through the right jugular vein under fluoroscopy.

MICROFILARICIDE TREATMENT IN DOGS

- Treatment aimed at killing microfilariae acutely is not recommended; rather it is sufficient to eliminate microfilariae, when present, during the process of treatment with doxycycline and a macrocyclic lactone. Arguments in favor of not attempting to kill microfilariae acutely include the following:
  - Lack of proven disease associated with microfilaremia
  - Potential for adverse reactions
  - Monthly prophylactic drugs, in combination with doxycycline, result in gradual disappearance of microfilariae from the blood, even if gravid female worms persist
- High-dose ivermectin (50 µg/kg) and standard dose milbemycin oxime (0.5 mg/kg, one dose) are acutely microfilaricidal. In the face of high microfilarial

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counts (> 40,000/mL), adverse reactions are common. Usually, the microfilarial concentration is <10,000/mL, and mild adverse reactions occur in only approximately 10% of dogs. Most adverse reactions are limited to brief ptyalism and defecation occurring within hours and lasting up to several hours.

- However, some dogs, especially small dogs, with high microfilariae counts may develop tachycardia, tachypnea, pale mucous membranes, diarrhea, and even shock. Treatment includes intravenous fluids and a soluble corticosteroid. Recovery is rapid when treatment is administered quickly. Microfilariae counts are not routinely performed, and thus severe reactions are seldom expected. If microfilaricide treatment is chosen, it should be administered in the morning, and the patient observed for the day and discharged in the late afternoon or evening.
- Most veterinarians have chosen not to administer a microfilaricidal drug but rather to use doxycycline and a macrocyclic lactone, realizing that the microfilariae will gradually disappear. Milbemycin oxime should not be used under this logic because the prophylactic dose is microfilaricidal. The primary argument for adhering to the traditional approach (i.e., treating microfilariae) is that microfilaremic dogs serve as a reservoir for mosquitoes to acquire the microfilariae and possibly transmit them to another dog or cat. However, we are of the opinion that this risk is small.

Key Point

Rapid kill of microfilaria is unnecessary.

CAVAL SYNDROME IN DOGS

- Caval syndrome is uncommon except in very highly endemic regions such as Florida and the Gulf Coast. This syndrome typically occurs in young adult dogs that have received a massive exposure to L_3 over a short time. Thus large numbers (>100) of worms mature in the heart and pulmonary arteries at the same time. The right-sided heart chambers become engorged with worms, and many extend into the venae cava. Tricuspid regurgitation occurs, and pulmonary outflow tract and right-sided heart inflow become abnormal. Cardiovascular collapse results. Secondary complications include hemolysis, hemoglobinuria, acute hepatic failure, and renal failure.
- Unless appropriate treatment is provided, death usually occurs within 24 hours. Treatment is removal of the worms (with minimum sedation) through the right jugular vein with a long (preferably flexible) alligator forceps or horsehair catheter. Subsequent to worm removal, supportive fluid therapy is provided. Adulticide treatment of some remaining worms may be required after recovery (i.e., in 1–3 weeks). The success rate of this treatment is excellent, but experience is essential.

ANCILLARY TREATMENTS FOR CANINE HEARTWORM DISEASE

Several ancillary drugs or treatments are sometimes used in the treatment of heartworm disease. They are usually limited to the treatment of severe (class 3) heartworm disease. The indications and actions of these treatments are summarized in Table 10.1.

HEPARIN

- Heparin should be considered for dogs with class 3 disease with the following conditions:
  - Symptomatic pulmonary thromboembolism
  - Hemoglobinuria
  - Severe thrombocytopenia
- Heparin (100–300 U/kg SC q8h for 3–7 days) helps reduce the severity of pulmonary thrombosis; thus it helps improve blood flow, and by inhibiting fibrin production, it reduces platelet consumption, red blood cell damage, hemolysis, anemia, and hemoglobinuria. Heparin is combined with cage confinement.

CAGE CONFINEMENT

- For class 3 disease, it is important to minimize the need for cardiac output and pulmonary blood flow. Exertion increases the strain on the right ventricle and increases the risk of pulmonary thrombosis. Cage confinement or another form of severe confinement should be continued for 4 to 6 weeks.

OXYGEN

- Oxygen promotes pulmonary arterial dilation and is a useful component for dogs that are severely debilitated by class 3 disease and whenever symptomatic pulmonary thromboembolism occurs, either before or following adulticide treatment. Some class 3 disease patients have been kept in oxygen-cage confinement for as long as 3 weeks. Such patients usually also receive heparin.

Table 10.1 Indications and Actions of Ancillary Treatments for Canine Heartworm Disease

TREATMENT	EFFECT	INDICATION
Doxycycline or minocycline	*Wolbachia* therapy; impairs microfilaria development	Often recommended in all cases of heartworm infection
Aspirin or Clopidogrel	Platelet inhibition	Severe pulmonary arterial disease
Heparin	Anticoagulant	Thrombosis Hemoglobinuria
Steroids	Antiinflammatory	Parenchymal lung disease secondary to pulmonary thromboembolism Allergic pneumonitis

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CORTICOSTEROIDS

- These drugs reduce the inflammation associated with vasculitis and pulmonary thromboembolism but do not improve pulmonary arterial disease itself. With extended use, they actually reduce pulmonary blood flow by promoting coagulation. When vasculitis and/or pulmonary thrombosis occur, plasma and inflammatory cells leak out of the smaller arterioles, leading to granulomatous inflammation. Corticosteroids help reduce the severity of inflammation. The question arises as to whether corticosteroids should be given prophylactically because all dogs with heartworm disease will experience at least mild granulomatous inflammation and pulmonary thrombosis, which occurs from 3 to 21 days after administration of adulticide (most severe from 10 to 17 days). Some clinicians prescribe prednisone (1.0 mg/kg q24h) beginning a few days after adulticide treatment and continuing for 1 to 2 weeks. Reduced fever and improved appetite are potential benefits.
- Corticosteroids are indicated whenever there is clinical, laboratory, and radiographic evidence of moderate to severe pneumonitis and/or pulmonary thromboembolism (whether before or after adulticide treatment). Clinical signs include tachypnea, dyspnea, pale or gray mucous membranes, coughing, and hemoptysis. An inflammatory leukogram and thrombocytopenia are usually present. The duration of use is generally 5 to 10 days, until there is clinical and radiographic evidence of significant improvement. Potential adverse actions of corticosteroids are GI ulceration and decreased pulmonary blood flow resulting from enhanced thrombosis. These effects are both time and dose related.

COMPLICATIONS OF HEARTWORM DISEASE

- Most complications of heartworm disease are the result of severe pulmonary myointimal proliferation and are thus associated with or indicative of class 3 infections.

PULMONARY THROMBOEMBOLISM

- Preadulticide and postadulticide thrombosis is common. Clinical signs are the following:
  - Coughing
  - Tachypnea
  - Dyspnea
  - Pyrexia
  - Hemoptysis
  - Leukocytosis
  - Thrombocytopenia
- Pulmonary thromboembolism, although possible at any time, is most likely to occur and is most severe during the first month after adulticide treatment. Diagnosis can be assisted with thoracic radiographs and D-dimer assay. Treatment is cage confinement, corticosteroids, and heparin.

THROMBOCYTOPENIA

- Variable reductions of platelet counts are commonly seen, especially with class 3 disease. Thrombocytopenia may occur before but is most common and severe after adulticide treatment. Thrombocytopenia is consumptive as the result of vasculitis, thromboembolism, and/or disseminated intravascular coagulopathy. Treatment is heparin.

HEMOGLOBINURIA

- Red blood cell lysis caused by severe pulmonary thromboembolism and fibrin deposition is an occasional complication of class 3 disease and/or the caval syndrome. Treatment is heparin for class 3 disease and worm retrieval for the caval syndrome.

CARDIAC CIRRHOSIS

- Severe PH leading to chronic right-sided CHF can cause gradually progressive hepatic congestion, leading to fibrosis and failure. There is no effective treatment.

EOSINOPHILIC PNEUMONITIS

- Approximately 10% to 20% of dogs with occult heartworm infection experience allergic or eosinophilic pneumonitis. The syndrome produces severe respiratory signs (coughing-dyspnea). However, this is an early syndrome and is usually not associated with severe PH. The pathophysiologic process involves immune entrapment of microfilariae in the pulmonary capillaries (occult infection results) with an unusually severe eosinophilic infiltrate. Pulmonary crackles may be ausculted and radiograph interpretation may be confused for cardiogenic pulmonary edema (left-sided CHF), *Pneumocystis carinii* infection, or pulmonary blastomycosis. The treatment is corticosteroids, and the response is rapid and complete. Adulticide treatment should be administered as soon as the symptoms have resolved.

EOSINOPHILIC GRANULOMATOSIS

- A small percentage of dogs with immune-mediated occult disease have eosinophilic inflammation and infiltration of numerous organs, with the most severe manifestation being multiple, variable-sized pulmonary eosinophilic granulomas. Granulomas may develop in the bronchi and the trachea. Eosinophilic pleural effusion may develop. This syndrome responds initially to corticosteroids, but relapse with progressive pulmonary disease is usual. The addition of azathioprine, cyclophosphamide, or mycophenolate has been beneficial. However, death is the end result in most cases, even after the heartworms have been killed.

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Key Point

Exercise restriction and controlled leash walks are recommended for 4 to 6 weeks after melarsomine therapy.

HEARTWORM PROPHYLAXIS

- Heartworm prophylaxis is recommended for all dogs and cats at risk of infection. Heartworm testing is an integral component of prophylactic therapy. All dogs given heartworm prophylaxis for the first time should be both microfilaria free and antigen free. Annual antigen testing is recommended in all dogs >6 to 7 months of age, regardless of the type of heartworm prophylaxis used. Although some have questioned the need for annual testing, poor compliance is an ongoing problem. In addition, some strains of larvae are resistant to treatment, and this resistance will result in adult infection. Failure to properly increase the drug dosage in large, rapidly growing puppies may also cause prophylactic failure.

MONTHLY PROPHYLAXIS

- A number of drugs are available for heartworm prophylaxis in dogs and cats, with some providing extended parasite control. They are very effective when administered monthly because larvae up to 4 months of age post-L_3 inoculation are readily killed by these agents. Adult infection is unlikely if a monthly dose is occasionally missed. The monthly use of these drugs causes gradual disappearance, over 5 to 9 months, of circulating microfilariae in dogs with adult heartworms. Thus antigen-detecting serologic tests should be used for annual screening. It is best to test 7 to 8 months either after the mosquito season begins or after the previous mosquito season ends.
- Invermectin, milbemycin oxime, moxidectin, and selamectin are recommended. All are safe as prescribed for all breeds. Milbemycin oxime, however, kills microfilariae (L_1) quickly, and shock can occur in the face of high L_1 concentrations. Thus milbemycin oxime is not administered as a preventative to dogs with microfilariae.

REPORTS OF LACK OF EFFICACY

- Lack of efficacy (LOE) of a heartworm preventive product is defined as an animal testing heartworm positive regardless of appropriateness of dosage or administration consistency. There are many possible reasons for LOE, including resistance to macrocyclic lactones, inadequate compliance, variation in pharmocodynamics, variation in pharmacokinetics, or combinations of these.
- In vitro studies have identified microfilariae that are less susceptible to high doses of all the macrocyclic lactones. These microfilariae exhibit an allele on the P-glycoprotein gene that differs from the general population.
- In addition, studies have demonstrated decreased susceptibility of the MP3 strain (originally collected in northeast Georgia) to single monthly doses of commercial heartworm products. It is possible that the adulticide treatment of dogs with macrocyclic lactones, rather than melarsomine, may be selecting for populations of resistant microfilariae through prolonged drug exposure. Therefore it is recommended to treat dogs with adult heartworms with melarsomine and avoid the "slow-kill" protocol when possible.

Key Point

Because of reported lack of efficacy of heartworm preventative product, avoid the "slow-kill" protocol when possible.

SUGGESTED READINGS

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Chapter

11

Pericardial Disorders and Cardiac Tumors

Amanda E. Coleman*

- Most clinically significant pericardial disorders in small animals are associated with pericardial effusion, defined as fluid accumulation within the pericardial space in excess of normal. Excessive intrapericardial fluid can result in cardiac tamponade, a life-threatening phenomenon characterized by compromised cardiac filling and output.
- The most common causes of pericardial effusion in dogs are neoplastic, accounting for 38% to 71% of cases.
- Hemodynamically significant pericardial effusion seldom occurs in cats. In this species, pericardial effusions most commonly occur in association with congestive heart failure (CHF), are of small volume, and do not produce cardiac tamponade. Consequently, these effusions are most often an incidental manifestation of underlying disease rather than the primary cause for presenting signs.

PERICARDIAL EFFUSION AND CARDIAC TAMPONADE

PATHOPHYSIOLOGY

- In health, the pericardial space contains a small amount of fluid, an ultrafiltrate of plasma that serves to lubricate the heart during the cardiac cycle. Normal intrapericardial pressure is subatmospheric and mirrors that of the pleural space.
- Small increases in the volume of intrapericardial fluid can be tolerated without hemodynamic consequence. However, once the reserve volume of the pericardium is exceeded, further increases are associated with a potentially dangerous rise in intrapericardial pressure.
- *Cardiac tamponade*, a phenomenon that occurs during the decompensated phase of pericardial effusion development, results from intrapericardial pressure elevations that are sufficient to compress the right-sided cardiac chambers during diastole, interfering with normal systemic venous return. The result is a reduction in cardiac stroke volume and impaired cardiac output.
- The volume of pericardial effusion that can be tolerated without development of cardiac tamponade is closely related to rate of fluid accumulation. In experimental canine models involving the rapid infusion of fluid or air into the pericardial space, volumes of 60 to 80 mL of fluid and 8.7 ± 1.9 cm^3 of air per kilogram of body weight are sufficient to produce cardiac tamponade. In contrast, dogs with slowly developing pericardial effusions can tolerate fluid volumes exceeding 1 to 1.5 L, which reflect the ability of the pericardium to adapt over a longer time course, stretching to allow greater increases in fluid volume with less incremental pressure rise.

INCIDENCE AND PATIENT SIGNALMENT

- At a university referral veterinary hospital (University of Minnesota Veterinary Medical Center), pericardial effusions were confirmed as the primary cause for clinical signs in 1 of every 233 new canine admissions from January 1999 to December 2001, accounting for approximately 7% of dogs with clinical signs of heart disease and representing an incidence of 0.43%.
- Symptomatic pericardial effusions most commonly occur in older and larger dogs, with median age and body weight at the time of presentation ranging from 9.1 to 9.7 years and 31.2 to 34.8 kg, respectively.
- Dogs of the Golden Retriever, Labrador Retriever, German Shepherd, Saint Bernard, Newfoundland, and Great Dane breeds appear to be predisposed to the development of pericardial effusion. In one report, Saint Bernard dogs were diagnosed at a younger age than dogs of other commonly represented breeds.

CLINICAL PRESENTATION

Historical and physical examination findings in patients with hemodynamically significant pericardial effusion are attributable to the consequences of increased systemic venous pressure and

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decreased forward cardiac output (systemic arterial hypotension).

History and Presenting Complaints

- In most cases, clinical signs in dogs with pericardial effusions are nonspecific and have an apparently acute onset.
- The clinical presentation of patients with symptomatic pericardial effusion is influenced by the rate of pericardial fluid accumulation. For those in which fluid development is gradual, time allows for pericardial stretch and compensatory neurohormonal mechanisms, which fight to maintain cardiac output at the expense of elevated systemic venous pressures. In these patients, signs of right-sided CHF predominate. In these cases, owners might report weakness or lethargy, decreased exercise tolerance, exertion-related syncope, reduced appetite, or abdominal distension caused by ascites.
- In contrast, dogs affected by acute cardiac tamponade have a clinical picture that is characterized by a sudden decline in cardiac output, with signs of weakness, collapse, increased respiratory effort, or shock.
- Additional presenting complaints can include cough, vomiting, diarrhea, polyuria, and polydipsia. In a 2017 study of dogs presented to the emergency department of a university teaching hospital, 25 (51%) of 49 had a history of vomiting within 48 hours of presentation.

Physical Examination Findings

- Dogs with pericardial effusion can have clinical signs that range from subtle to those associated with hemodynamic collapse due to severe cardiac tamponade. Because the clinical picture of these patients can be nonspecific in nature, a thorough physical examination is critical to raise the clinician's index of suspicion for pericardial effusion.
- Common physical examination findings in animals with pericardial effusion include muffled heart sounds, generalized weakness, poor pulse quality, tachycardia, and tachypnea. Abdominal distention with a palpable fluid wave caused by ascites is also noted in 49% to 63% of cases.
- In dogs with cardiac tamponade, careful physical examination usually discloses jugular venous distension, a consequence of elevated right-sided cardiac filling pressures. In dogs with long or thick hair coats, clipping of the jugular furrow can make this more obvious.
- Pulsus paradoxus can be identified. Pulsus paradoxus is defined as a phasic variation in pulse quality that is associated with respiration and is characterized by a decrease in pulse quality during inhalation due to a fall in systolic pressure >10 mm Hg.

Key Point

The finding of a combination of muffled heart sounds, jugular venous distension, and weak pulse quality or pulsus paradoxus should heighten concern for the presence of pericardial effusion.

DIAGNOSTIC FINDINGS

Thoracic Radiography

- Thoracic radiography can be useful in the diagnostic evaluation of suspected pericardial effusion, although its limitations should be recognized. In addition to supporting or facilitating a diagnosis of pericardial effusion in some cases, radiographs can demonstrate pulmonary metastases, radiopaque intrapericardial foreign bodies, or lymphadenomegaly. In dogs with heart base tumors, radiographic findings can suggest this as the underlying etiology.
- In patients with small-to-moderate volumes of pericardial effusion, the cardiac silhouette can be normal in size (as reported in up to 48% of cases) or variably enlarged. In cases without enlargement of the cardiac silhouette, pericardial effusion might remain undetected without additional diagnostic testing, such as echocardiography.
- Radiographic signs whose presence support a diagnosis of pericardial effusion include (Fig. 11.1):
  - An enlarged and rounded ("globoid") cardiac silhouette without specific chamber enlargement pattern(s)
  - Sharply delineated margins of the cardiac silhouette, attributable to decreased or absent motion during the cardiac cycle
  - Convexity of the dorsocaudal aspect of the cardiac silhouette
  - Signs consistent with right-sided CHF, including widening of the caudal vena cava, hepatomegaly, ascites, pleural effusion, or a combination of these
  - Abnormally small pulmonary vasculature caused by pulmonary undercirculation
- Radiography has poor diagnostic accuracy for the identification of pericardial effusion in dogs with cardiac tamponade. One study of thoracic radiographs from 50 dogs with symptomatic pericardial effusion found that 22% did not have cardiac silhouette enlargement, 42% did not have a "globoid" cardiac silhouette, and 43% did not have convexity of the dorsocaudal aspect of the cardiac silhouette. Further, these radiographic variables had poor specificity when compared between dogs with cardiac tamponade due to pericardial effusion and normal dogs or dogs with heart disease not associated with pericardial effusion.

Electrocardiography

- Electrocardiography has poor sensitivity for the detection of pericardial effusion. Most dogs with

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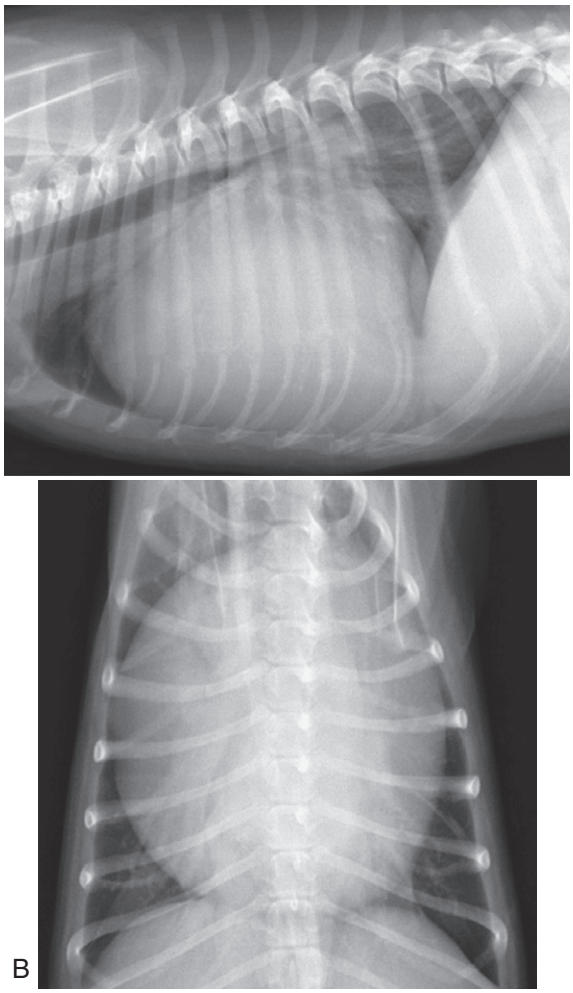


Fig. 11.1 Orthogonal thoracic radiographs from a dog with a large volume of pericardial effusion. **(A)** The lateral projection shows a markedly enlarged cardiac silhouette, dorsal tracheal deviation, and overlap of the cardiac silhouette and ventral diaphragm. **(B)** The ventrodorsal projection shows bilateral contact between the cardiac silhouette and the costal margins. The edge of the cardiac silhouette is sharply delineated, and the lung fields are clear of any infiltrate that would indicate the presence of left-sided congestive heart failure.

pericardial effusion have a normal sinus rhythm, often with sinus tachycardia.

- QRS complexes can be of low voltage (i.e., R-wave amplitude <1 mV in all limb leads), a finding that is present in 50% to 57% of cases (Fig. 11.2A and B).
- Ventricular ectopy and, less commonly, supraventricular tachycardia and atrial fibrillation, can also be noted.
- *Electrical alternans* during sinus rhythm, defined by a beat-to-beat variation in the amplitude of the QRS complex (Fig. 11.2C), is suggestive of pericardial effusion. This phenomenon results from the to-and-fro swinging of the heart within the fluid-filled pericardial sac relative to stationary limb leads, which results in changes in the orientation of the mean electrical axis relative to the lead axis with each beat. Electrical alternans is present in only 20% to 64% of dogs with pericardial effusion.
- ST segment elevation is a well-described finding in the earliest stages of acute human pericarditis. Although mentioned in one study of canine pericardial effusion, ST segment change has not been well characterized in this species.

Echocardiography

- Echocardiography is the most sensitive and specific noninvasive diagnostic tool for confirmation and semiquantitative estimation of pericardial effusion. In addition, this modality allows for characterization of the potential hemodynamic significance of the effusion and evaluation for and localization of potential underlying etiologies.
- With two-dimensional echocardiography, pericardial effusion is typically seen as a circumferential anechoic or hypoechoic space surrounding the heart, the size of which is proportional to the volume of effusion. In large-volume pericardial effusions, the heart might "swing" freely within the pericardial space: this beat-to-beat change in cardiac position is responsible for the surface electrocardiographic finding of electrical alternans.
- Occasionally it can be difficult to differentiate pericardial and pleural effusions. The former are characterized by a smooth, round outer contour, whereas the latter are more irregularly marginated. In cases with both pericardial and pleural effusion, the pericardium is easily perceived as an echogenic structure with anechoic or hypoechoic space on either side.
- Echocardiographic evidence of cardiac tamponade is seen as diastolic collapse of the free wall of the right atrium, right ventricle, or both. The right atrial free wall should be outwardly curved throughout the cardiac cycle, reflecting the normal positive transmural distending pressure. Although the right atrium normally contracts in volume with atrial systole, pronounced inversion or collapse of the right atrial free wall that persists throughout atrial diastole provides evidence of elevated intrapericardial pressure and transient reversal of transmural pressure (Fig. 11.3). In severe cases, the right ventricle can be affected as well, with echocardiographic signs ranging from transient and localized concavity of the right ventricular free wall to near-complete right ventricular chamber obliteration throughout diastole (Fig. 11.3).

In cases with cardiac tamponade, the left ventricular cavity is usually small with increased end-diastolic wall thickness (i.e., left ventricular "pseudohypertrophy") as a consequence of decreased venous return. Experienced observers might also identify a distended caudal vena cava with decreased collapsibility, gallbladder wall thickening and edema, or both.

- In addition to confirming the presence and estimating the quantity of pericardial effusion, echocardiography is valuable for the detection of associated

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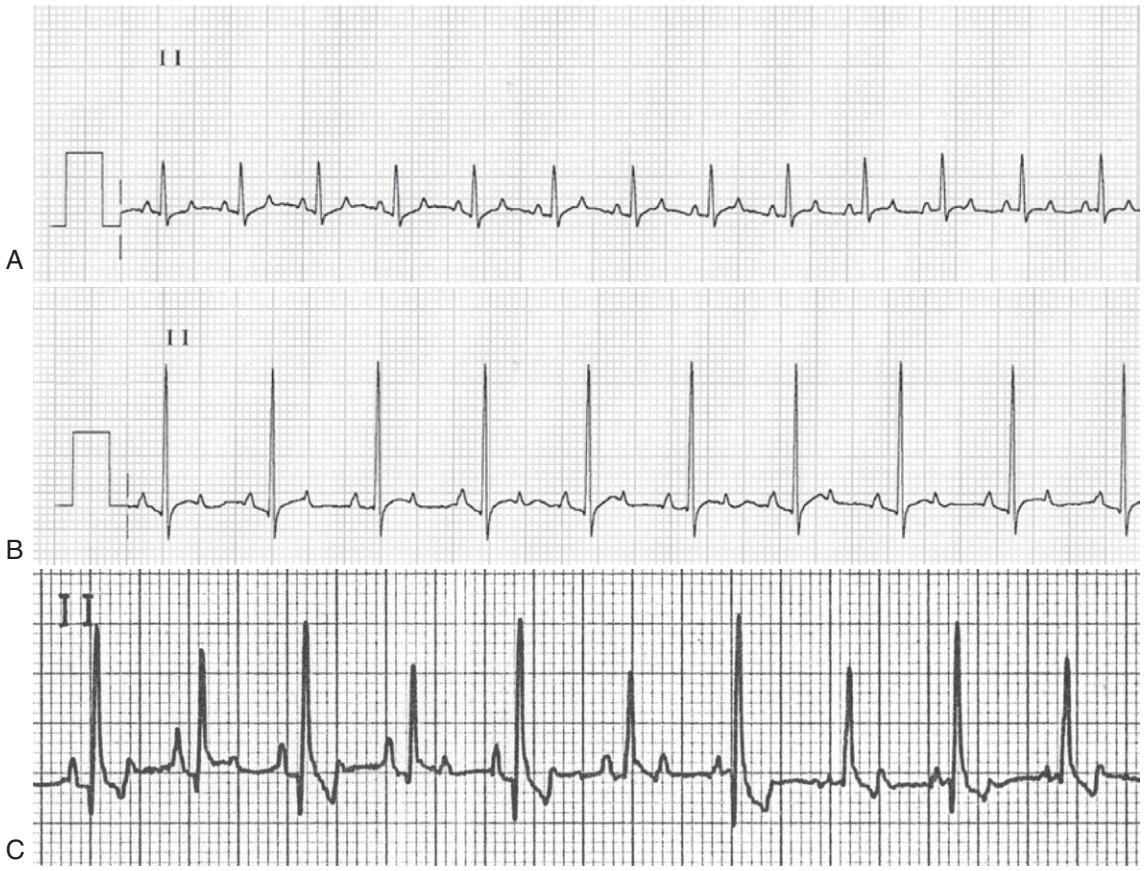


Fig. 11.2 Electrocardiograms (lead II) from two dogs with pericardial effusion. Paper speed, 25 mm/s; calibration box, 1 mV. Recordings **(A)** and **(B)** were taken from the same dog before and after pericardiocentesis, respectively. **(A)** Prior to pericardiocentesis, the QRS complexes are of low voltage (R wave <1 mV) and heart rate is 140 beats/min. **(B)** After pericardiocentesis, R-wave amplitude is almost 2 mV and heart rate is 100 beats/min. Recording **(C)** was taken from a different dog with a large volume of pericardial effusion, and shows beat-to-beat variation in the amplitude and contour of the QRS complexes characteristic of electrical alternans. (A and B, Modified from Tobias AH: Pericardial disorders. In Ettinger SJ, Feldman EC, eds: *Textbook of veterinary internal medicine*, ed 6, St. Louis, 2005, Saunders; C, Courtesy Dr. Teresa DeFrancesco, Raleigh, North Carolina.)

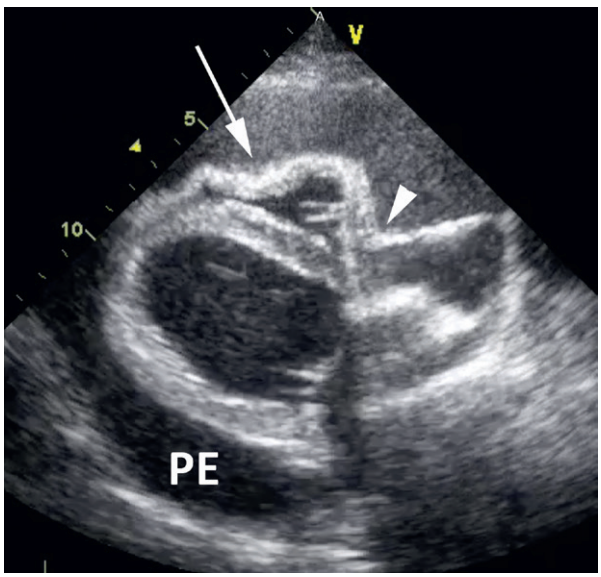


Fig. 11.3 Echocardiographic evidence of cardiac tamponade in a dog with pericardial effusion (*PE*). This two-dimensional echocardiographic image was recorded from a right parasternal window and shows diastolic inversion of the right atrial (*arrowhead*) and right ventricular (*arrow*) walls.

intrapericardial or pericardial masses. Whereas histopathologic examination is necessary to confirm and definitively establish tumor type, the echocardiographic location and characteristics of an intrapericardial mass provide information about its most probable classification. This is particularly important given that prognoses associated with the various causes of pericardial effusion differ tremendously with tumor of origin. Echocardiographic findings specific to the two most common tumor types identified in dogs are discussed in "Echocardiographic Features of Specific Tumor Types."

- In one study, the reported sensitivity and specificity of echocardiography for detection of a cardiac mass of any type, when performed by a board-certified veterinary cardiologist or cardiologist-in-training, was 82% and 100%, respectively. With repeat examinations, sensitivity of this test increased to 88%. Older studies report sensitivity values ranging from 17% to 63%, and in a more recent report on surgical outcomes in dogs with right atrial hemangiosarcoma (HSA) undergoing surgical resection, only 48% had a mass evident on echocardiography.

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Key Point

The presence of pericardial fluid facilitates the detection of intrapericardial masses, which is particularly relevant to the delineation of HSA and, to some extent, heart base tumors. Pericardial fluid forms an echolucent zone around the right atrium and auricle and the ascending aorta, the locations from which these tumors most commonly arise. In the absence of pericardial fluid, these locations can be obscured by lung interference. *Consequently, whenever immediate access to ultrasonography and the clinical condition of the patient permits, pericardiocentesis should be deferred until a thorough echocardiographic examination has been completed.*

- Echocardiography can also demonstrate findings consistent with other less common underlying causes of pericardial effusion, including intrapericardial cysts, thrombi associated with left atrial perforation, and the presence of abdominal viscera within the pericardial sac in cases of peritoneopericardial diaphragmatic hernia.

Echocardiographic features of specific tumor types

Cardiac hemangiosarcoma

- The positive and negative predictive values of echocardiography for detection of right atrial HSA vary from 92% to 100% and 64% to 87%, respectively. False negative studies are common.
- The following features are characteristic of cardiac HSA (Fig. 11.4):
  - These tumors most commonly arise from the wall of the right atrium or right atrial appendage, protrude into the pericardial space, and move with the heart. They can also protrude into the right atrial lumen, spread to involve other areas of the heart base and pericardium, and involve the right atrioventricular groove.
  - Right atrial masses are usually visible from right parasternal echocardiographic windows. However, these tumors can be small and elusive. Imaging from left parasternal locations to provide alternate imaging planes, especially of the right auricle, is necessary to demonstrate the presence of HSA in some cases.
  - Hemangiosarcoma typically contains small hypoechoic spaces, giving the tumor an inhomogeneous, mottled, or cavitary appearance. These tumors can also be cystic.

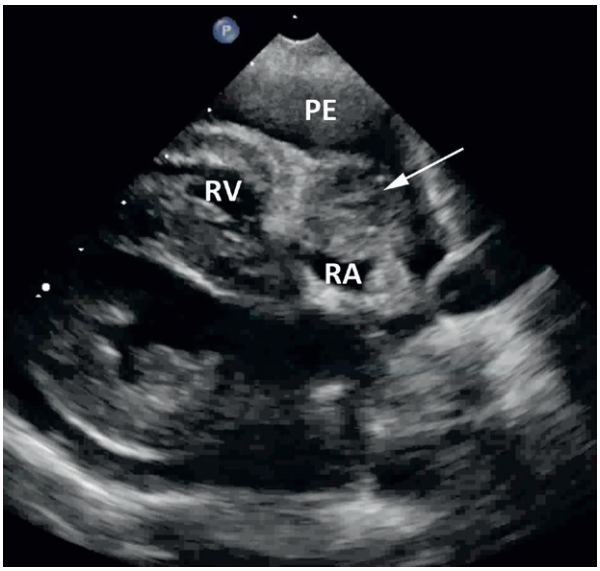


Fig. 11.4 Two-dimensional echocardiographic image recorded using a right parasternal window from a dog with cardiac hemangiosarcoma. A cavitary and cystic mass *(arrow)* is associated with the right atrial (*RA*) wall. *PE*, Pericardial effusion; *RV*, right ventricle.

Heart base tumors

- The reported positive and negative predictive values of echocardiography for detection of heart base tumors are 89% and 93%, respectively.
- Heart base tumors are usually associated with the aortic root and can vary from small round structures attached to the ascending aorta to extensive masses that surround the aorta and main pulmonary artery (Fig. 11.5). Compression or invasion of the atria and major blood vessels can be seen.

Advanced Imaging Techniques

- In people with pericardial effusion, advanced cross-sectional imaging modalities including computed tomography (CT) and cardiac magnetic resonance are considered superior means for characterization of cardiac tumors. At the time of writing, these tools have not been extensively evaluated in veterinary patients with pericardial effusion.
  - The diagnostic utility of multidetector CT was evaluated in 11 dogs with naturally occurring pericardial effusion. Although CT did not improve the investigators' ability to detect cardiac tumors when compared to echocardiography, it did expose noncardiac metastatic and concurrent neoplastic disease, offering a potential advantage over traditional techniques by allowing more complete disease staging.
  - The utility of cardiac magnetic resonance for enhancing the detection of cardiac masses has also been evaluated in a small number of dogs with pericardial effusion. In these cases, cardiac magnetic resonance was not able to improve the diagnosis of cardiac tumors compared to echocardiography. However, the authors described a significant learning curve and suggested that with substantial training, the accuracy of this modality might improve.
  - Future studies could elucidate a benefit of these modalities in cases of pericardial effusion in which a cardiac mass is not detected or findings are equivocal based on transthoracic echocardiography alone.

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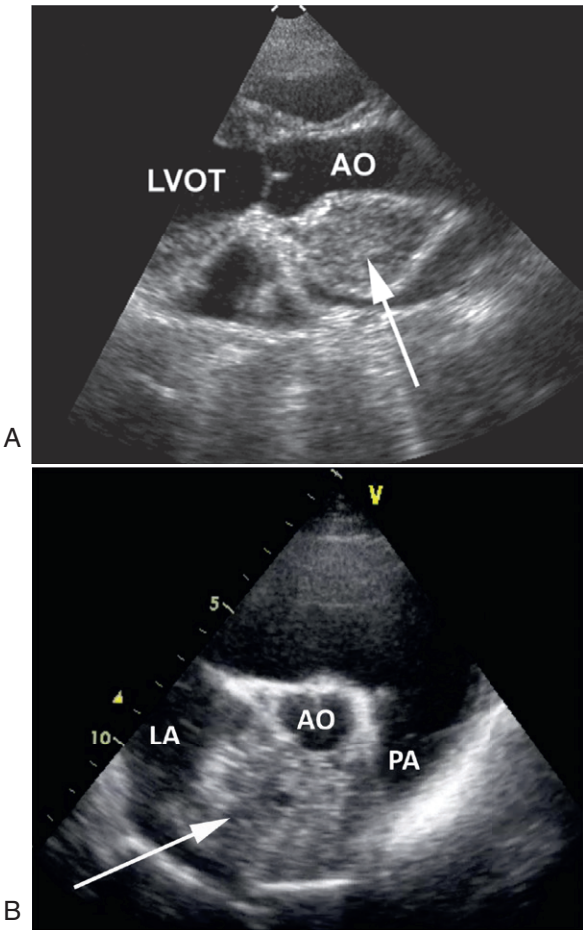


Fig. 11.5 Two-dimensional echocardiographic images recorded using right parasternal windows in two dogs with a heart base tumor. **(A)** A large round homogeneous mass *(arrow)* is seen adjacent to the aortic root *(AO)*. **(B)** A similar well-circumscribed mass is noted between the AO, pulmonary artery *(PA)*, and left atrium (*LA*). *LVOT*, Left ventricular outflow tract.

- Currently, for veterinary patients with cardiac tumors, advanced cross-sectional imaging can complement echocardiography and thoracic radiography by allowing better characterization of lesion origin, extent, and invasion or compression of adjacent structures, and in planning for potential tumor resection, debulking, radiation therapy, or stent placement.
- The use of ^{18}F-fluoro-2-deoxyglucose positron emission tomography-CT for staging and monitoring of dogs with HSA and other malignancies has been described. Although currently limited by availability and expense, this tool could improve tumor detection in dogs with pericardial effusion for which conventional imaging fails to identify these masses.

Pericardial Fluid Analysis

- Pericardial effusions in dogs are nearly always hemorrhagic or serosanguineous, sterile, and inflammatory, regardless of cause.
- Total nucleated cell count, red cell count, protein concentration, pH, and lactate concentration of pericardial fluid overlap extensively between the various neoplastic and nonneoplastic causes of pericardial effusion. Therefore these characteristics are rarely useful in establishing etiology.
- Cytologic examination is also typically unrewarding for distinguishing among potential causes of pericardial effusion. In fact, fluid analysis establishes an etiologic diagnosis in only 7.7% to 12.8% of cases. In one study, the diagnostic utility of cytologic examination was found to increase to 20.3% in cases with an effusate hematocrit <10%, reflecting a threefold increased chance of obtaining a diagnosis in these cases.
- Hemangiosarcoma and aortic body tumor (ABT), the most common neoplasms associated with pericardial effusions in dogs, are poorly exfoliating and therefore associated with a high incidence of false-negative cytologic diagnoses. Further, pericardial diseases that lead to effusion often result in dramatic mesothelial proliferation, with exfoliated reactive mesothelial cells that often bear characteristics that mimic malignancy. For these reasons, cytologic evaluation of pericardial effusion must be interpreted cautiously.
- Nonetheless, some of the less common causes of pericardial effusion, including infectious pericarditis and lymphosarcoma, are diagnosed primarily by pericardial fluid cytologic evaluation. Consequently, the author advocates submission of pericardial fluid for analysis in all cases, despite the generally low diagnostic yield of this test.
- Pericardial effusion should be cultured in cases in which an infectious cause is suspected based on cytologic examination.

Other Diagnostics

- Recently, transthoracic ultrasound-guided fine needle aspiration of cardiac masses with an anatomic location that permitted direct sampling was reported in six dogs. A histologic diagnosis was possible in all cases and no major complications were reported, suggesting that fine needle aspiration can safely provide a definitive diagnosis in certain cases, potentially allowing for more targeted therapy and prognostication.
- In one study, on average, dogs with cardiac HSA had greater circulating cardiac troponin I concentrations than dogs with idiopathic pericardial effusion, suggesting that this biomarker might be useful to differentiate between these etiologies when a mass is not identified on echocardiography. The results of another study suggested that plasma cardiac troponin I concentration >0.25 ng/mL in a dog with pericardial effusion is likely indicative of cardiac HSA. However, because dogs with infectious conditions were not included in either of these studies,

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the diagnostic accuracy of this biomarker in a "general population" of dogs with pericardial effusion remains untested.

Key Point

Cytologic examination of pericardial effusates is unreliable in distinguishing neoplastic compared with nonneoplastic (i.e., inflammatory) causes in most cases of pericardial effusion in dogs. However, submission of fluid is still recommended because diagnosis of some of the less common causes of pericardial effusion can still be reliably made.

INITIAL PATIENT STABILIZATION: PERICARDIOCENTESIS

- Pericardiocentesis—the mechanical removal of fluid from the pericardial space—is lifesaving in cases of pericardial effusion with significant hemodynamic compromise caused by cardiac tamponade. This procedure is performed to relieve cardiac tamponade and to obtain fluid samples for diagnostic purposes.
- Medical interventions are rarely successful in resolving signs of symptomatic pericardial effusion and should be used alone only while preparations for pericardiocentesis are made. Rapid intravenous fluid resuscitation can provide transient benefit by supporting systemic venous return, particularly in acute tamponade associated with hemorrhage. The exception to this is in dogs with hemopericardium caused by rupture of the left atrium, for which administration of intravenous fluids could precipitate or exacerbate cardiogenic pulmonary edema.
- The most serious potential complications of pericardiocentesis include death, or laceration or perforation of the myocardium or coronary vasculature. Additional risks include the development of life-threatening ventricular arrhythmias or pneumothorax.
- In one study of dogs undergoing pericardiocentesis for pericardial effusion of various etiologies, 20% required repeat pericardiocentesis within 24 hours. At the author's institution, the preference is to hospitalize cases for 12 to 24 hours following pericardiocentesis to monitor for signs of potential complications or rapid re-effusion. Some authors have described the safe placement of indwelling (i.e., for up to 3 days) pericardial catheters to facilitate repeated pericardial drainage in hospitalized dogs.

Approach to Pericardiocentesis

- The author's preferred approach is to restrain the animal in left lateral or sternal recumbency and to approach the pericardium from the right side. This technique avoids the major coronary arteries, reducing the risk of laceration, and provides a lung-free window through the cardiac notch.
- Severely affected dogs frequently tolerate pericardiocentesis without sedation; however, it might be necessary in some cases. At the author's institution, butorphanol (0.1–0.3 mg/kg, administered intravenously) is preferred when mild sedation is required.
- Continuous electrocardiographic monitoring should be performed during pericardiocentesis. Ventricular arrhythmias commonly occur because of contact between the catheter and epicardium and can require treatment. For this reason, one to two doses (2 mg/kg of body weight each) of 2% lidocaine can be pre-prepared for prompt intravenous administration if needed.
- The author prefers a 14- or 16-gauge over-the-needle catheter system to perform pericardiocentesis in dogs, although through-the-needle catheters are also effective. With the former system, two small side holes can be made near the tip of the catheter to minimize the chance of obstruction or aspiration of the myocardium against a single end hole. The catheter system is coupled to a large-volume syringe via an extension tube and a three-way stopcock.
- The right thorax is shaved, and using transthoracic ultrasound, the location providing the shortest distance between the pericardial space and skin without intervening lung tissue is chosen as the ideal site for entry. If ultrasound is not available, thoracic radiographs can be used to identify the ideal intercostal space. This usually corresponds to the fifth or sixth intercostal space, just ventral to the costochondral junction.
- The skin, intercostal muscles, and parietal pleura are infiltrated with 2% lidocaine, and the area is surgically prepared.
- A small stab incision is made in the skin (Fig. 11.6A), and the catheter system is advanced towards the pericardium, maintaining an orientation perpendicular to the body wall (Fig. 11.6B). During advancement, slight negative pressure is applied and maintained with the syringe and extension tube; this allows the operator to detect access to the pericardial space by observing fluid movement into the extension tubing and syringe.
- The catheter is then advanced over the needle into the pericardial space, with care taken to immobilize the stylet to avoid inadvertent laceration of the heart (Fig. 11.6C). The stylet is removed, the extension tube is connected to the catheter (Fig. 11.6D), and a small amount of fluid is aspirated (Fig. 11.6E).
- Pericardial fluid is typically hemorrhagic in appearance. For confirmation that the tip of the catheter is in the pericardial space and not within a cardiac chamber, an aliquot of aspirated fluid is placed in an activated clotting time tube. Blood will normally clot in an activated clotting time tube within 60 to 90 seconds. In contrast, hemorrhagic effusions in body cavities are rapidly depleted of clotting

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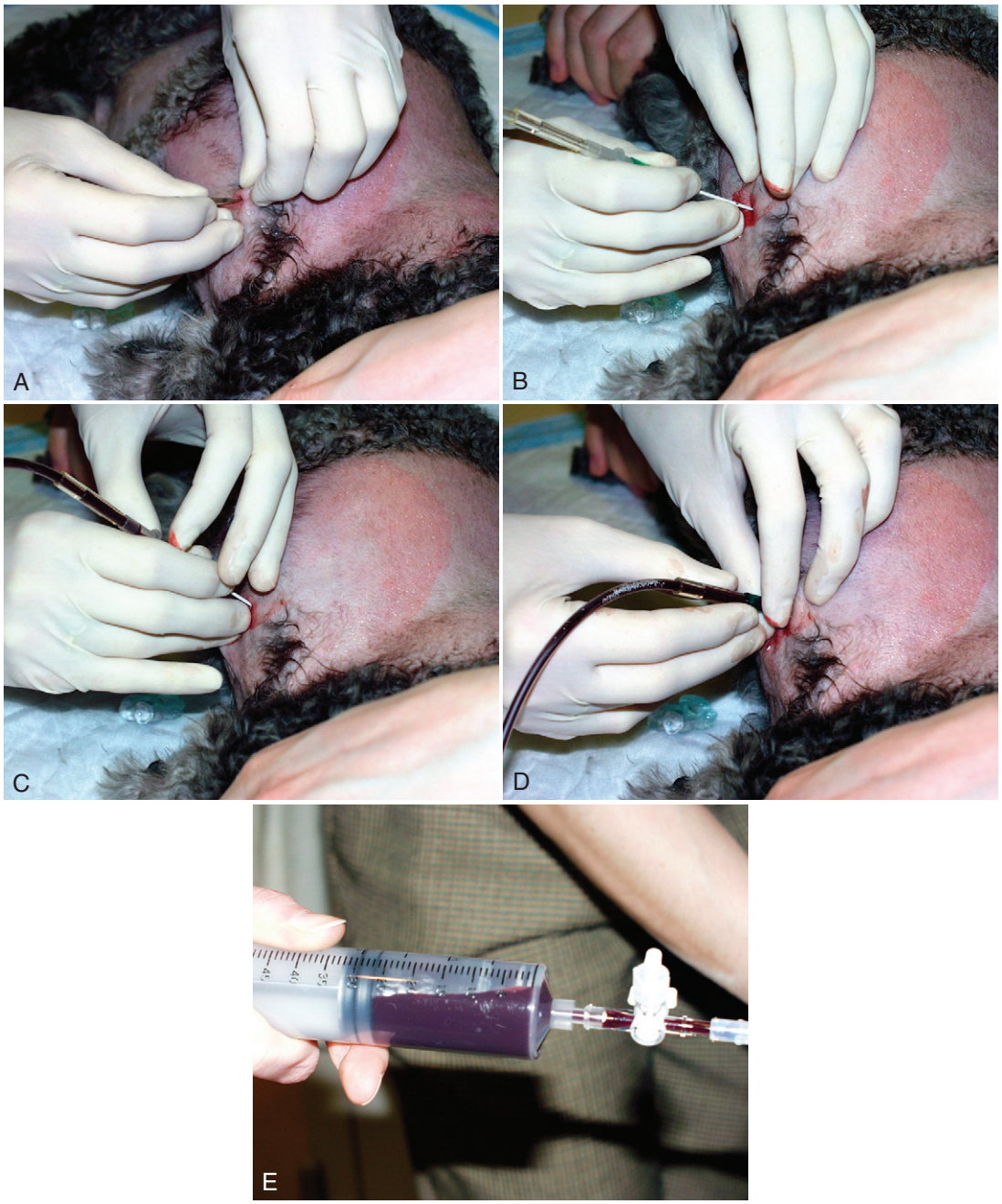


Fig. 11.6 Pericardiocentesis in a dog. **(A)** A small skin incision is made over the appropriate intercostal space after local anesthetic has been administered. **(B)** An over-the-needle catheter system with two side holes is advanced toward the pericardium. Slight negative pressure is applied with a syringe coupled to the needle by extension tubing. **(C)** Pericardial fluid flows into the extension tubing, and the catheter is carefully advanced over the needle into the pericardial space. **(D)** The extension tubing is removed from the needle and coupled to the catheter. **(E)** Pericardial effusion is aspirated.

factors and thrombocytes, meaning that pericardial fluid should not clot in such a tube. An alternative approach to confirm appropriate catheter placement involves instillation of a small volume of sterile agitated saline through the catheter and imaging with ultrasound. If no clots form within the activated clotting tube (or microbubbles confirm appropriate catheter placement), samples are collected for fluid analysis and culture, and the remaining pericardial fluid is aspirated. The catheter is then removed.

- In virtually all cases, pericardiocentesis results in rapid and marked hemodynamic improvement; this is manifest as improvement in clinical signs, pulse quality, and mucous membrane perfusion, and a decline in heart rate.

CHRONIC MANAGEMENT OF RECURRENT PERICARDIAL EFFUSION

The approach to the chronic management of pericardial effusion varies according to the underlying cause. Therefore the value of identifying likely cause by

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use of echocardiography, if possible, is emphasized. Treatment considerations for specific etiologies are considered later.

Conservative Management

- In 21% to 50% of dogs with idiopathic pericardial effusion (IPE), long-term resolution is achieved after a single pericardiocentesis.
- Intermittent pericardiocentesis can be used to palliate clinical signs in dogs with recurrent effusion. The reported median survival time of six dogs with IPE treated conservatively with repeated pericardiocentesis was 1834 days; in this group, a median of 2.5 (range, 2–11) treatments was required. Despite these results, there is concern for the eventual development of constrictive or effusive-constrictive pericarditis with this approach. Some authors, therefore, recommend pericardiectomy (surgical removal of the pericardial sac) in all dogs with recurrent pericardial effusion to avoid this potential complication.

Pericardiectomy

- Pericardiectomy is indicated in cases of recurrent pericardial effusion that are *not* associated with disorders of active bleeding (i.e., not associated with HSA, left atrial rupture, or hypocoagulable states). Significant survival benefit of pericardiectomy has been demonstrated in patients with IPE and pericardial effusion associated with ABT; however, survival time is not significantly improved in those with cardiac HSA treated with this approach alone.
- Removal of all or a portion of the pericardium decreases its surface area, thereby reducing fluid production when it directly participates. Furthermore, this procedure creates a connection between the pericardial and pleural spaces. Because of the larger resorptive surface area of the latter, fluid re-accumulation can be avoided or dramatically slowed.
- Direct examination during pericardiectomy allows the surgeon to identify any previously undetected masses in equivocal or presumed idiopathic cases. In addition, diagnostic samples of the pericardium and any identified masses can be obtained.
- The most common technique for pericardiectomy is the subphrenic (subtotal) technique, whereby the pericardium is transected just ventral to the phrenic nerves bilaterally. This is performed directly through a median sternotomy or lateral thoracotomy. Alternatively, thoracoscopic techniques have been described. Perioperative mortality rate for surgical subtotal pericardiectomy is approximately 13%.
- Less invasive approaches for pericardial resection or fenestration, including thoracoscopic techniques and balloon pericardiotomy, have been described in dogs with neoplastic and nonneoplastic pericardial effusion.
  - Thoracoscopic techniques provide theoretical advantages over open-chest approaches, including less tissue trauma and superior visualization (afforded by magnification and illumination), and less postoperative pain. Pericardial window techniques, by which a small (at least 3 cm × 3 cm) portion of the pericardium is excised, have been described; however, it appears that the creation of a window is likely inferior to subphrenic techniques for cases of IPE, as discussed below. Small patient size can limit thoracoscopic approach.
- Although reported in dogs, percutaneous balloon pericardiotomy is rarely utilized as this procedure has been associated with stoma closure and recurrence of pericardial effusion in many cases.

SPECIFIC CAUSES OF PERICARDIAL EFFUSIONS: EPIDEMIOLOGY, MANAGEMENT, AND PROGNOSIS

NEOPLASTIC CAUSES

Although cardiac tumors are relatively uncommon in dogs, neoplastic diseases are the most common causes of clinically significant pericardial effusion. Of these, cardiac HSA, chemodectoma (also referred to as ABT), mesothelioma, and ectopic thyroid carcinoma are reported with the greatest frequency. In general, dogs with neoplastic effusions tend to have a worse prognosis than dogs with effusions due to nonneoplastic causes. Cardiac tumors are not always associated with the presence of pericardial effusion; they can cause clinical signs by obstructing blood flow within the heart or great vessels, impairing myocardial function, or compressing adjacent structures. Here the focus is on cardiac tumors commonly associated with the development of pericardial effusion.

In general, the prognosis for cardiac neoplasia depends on the tumor type and whether useful treatment options exist and are pursued. Because definitive diagnosis is difficult to achieve in most cases, tumor location is frequently used to predict likely histologic diagnosis. This can be problematic, however, with one study reporting agreement of only 65% between presumptive (i.e., based on echocardiographic location) and definitive (i.e., necropsy/histopathologic) diagnoses. Although that study was limited by a small sample size (24 dogs) and likely bias toward cases with an unusual echocardiographic appearance, its findings serve as a practical reminder of the limitations of echocardiography.

Cardiac Hemangiosarcoma

Description and diagnostic features

- Hemangiosarcoma, a highly aggressive and malignant neoplasm of vascular endothelium or hematopoietic precursor cells, is the most commonly diagnosed cardiac tumor in the dog. In this species, it occurs with nearly tenfold greater incidence than ABT, the second most common cardiac neoplasm. Cardiac HSA also represents the most common cause of symptomatic canine pericardial effusion.

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- Cardiac HSA is most often associated with the right atrial free wall or right atrial appendage, and comprises the majority (88%) of masses affecting the right atrium. However, this tumor can also occur at other cardiac locations, including the heart base or ventricular myocardium, or as a diffuse, infiltrative tumor.
- These tumors produce pericardial effusion and cardiac tamponade when they rupture, rapidly bleeding into the pericardial space. Often the effusion is of small or moderate volume when clinical signs associated with cardiac tamponade manifest.
- Hemangiosarcoma is highly metastatic. Among dogs with cardiac HSA presenting to a referral center for signs attributable to cardiac tamponade, the prevalence of detectable, concurrent, noncardiac HSA is approximately 55%. Two necropsy-based studies of dogs dying or euthanized because of HSA-related pericardial effusion found similar metastatic rates of 68% to 75%.

Epidemiology

- Pericardial effusion due to cardiac HSA tends to affect older, large-breed dogs, with a median age at diagnosis of approximately 9 years (range, 3–13 years) and a mean body weight of 32 kg.
- Reported breed predispositions for HSA include Golden Retriever and German Shepherd dogs.
- No clear gender predilection for cardiac HSA has been reported, although within gender, intact dogs, particularly females, appear to be at greater risk for development of these tumors.

Treatment and prognosis

- Cardiac HSA is associated with a grave long-term prognosis.
- Many owners of dogs with cardiac HSA choose palliation with pericardiocentesis alone. Pericardiocentesis is predictably associated with marked clinical improvement; however, clinical signs of tamponade typically recur within days or weeks, often resulting in death or prompting euthanasia. Median survival time for dogs with pericardial effusion due to HSA, treated with either pericardiocentesis or pericardiectomy alone, is approximately 7 to 16 days.
- Pericardiectomy alone has not been clearly associated with an increased disease-free interval or survival benefit in dogs with cardiac HSA and pericardial effusion. This might be because removal of the pericardium increases the risk of exsanguination.
- Yunnan Baiyao, a Chinese herbal remedy with hemostatic properties, has been used to treat dogs with pericardial effusion due to HSA. In one retrospective case-control study of 67 dogs with right atrial masses and pericardial effusion, treatment with Yunnan Biayao, alone or with ε-aminocaproic acid, appeared to be safe. However, its use was not associated with increases in survival time, time to recurrence of clinical signs, or number of pericardiocenteses performed, when compared to pericardiocentesis alone.
- More aggressive approaches to the treatment of cardiac HSA have included various combinations of tumor resection, pericardiectomy, chemotherapy, and radiation therapy.
  - In general, surgical excision of HSA is considered the treatment of choice, when possible. Various authors have noted median survival times of 42 days to 4 months in dogs with right atrial HSA treated by mass resection alone.
  - Tumor resection followed by adjuvant chemotherapy might prolong survival in dogs with cardiac HSA. A retrospective study of 23 dogs with right atrial HSA in which tumor resection and pericardiectomy were performed reported a median survival time of 56 days in all dogs. However, the eight dogs receiving adjuvant chemotherapy lived significantly longer than those who did not, with median survival times of 175 days and 42 days, respectively. In that study, no dog lived greater than 7.6 months.
  - Another report retrospectively examined the use of doxorubicin-based chemotherapy protocols alone for the treatment of right atrial masses presumed to be HSA based on echocardiography. These patients did not undergo pericardiectomy or mass resection, and only one had histopathologic confirmation of tumor type. Median survival time in this sample was 139 days (range, 2–302). In a more recent study that compared outcomes in dogs with presumed HSA that were either treated with first-line doxorubicin ($n = 64$) or untreated ($n = 76$), median survival times were 119 days and 12 days, respectively.
  - The results of two small studies evaluating the use of radiotherapy alone or with medical therapy in dogs with pericardial effusion caused by right atrial tumors presumed to be HSA suggest potential clinical benefit.
    - In six dogs treated with a single (12 Gy) fraction, external-beam radiation therapy was associated with a MST of 79 days and a statistically significant decrease in the frequency of pericardiocentesis.
    - In another report of seven dogs (five of which had pericardial effusion, and none of which had CT evidence of pulmonary metastasis) treated with hypofractionated (i.e., 20–30 Gy as five daily fractions) intensity-modulated radiotherapy, weekly vinblastine, and daily oral propranolol, effusion resolved in all cases and median progression-free survival and MST were 290 and 326 days, respectively.
- Management of cardiac HSA should include consultation with a veterinary oncologist to take

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advantage of continually evolving and emerging treatment strategies.

Heart Base Tumors

Description and diagnostic features

- The term *heart base tumor* is used to designate any mass located at the base of the heart in association with the great vessels.
- Most heart base tumors in dogs are ABTs (also referred to as aortic body chemodectoma and nonchromaffin paraganglioma), although other tumor types are reported in this location. These include HSA, tumors arising from ectopic thyroid tissue, and mesothelioma, which account for up to 35%, 26%, and 17% of heart base tumors, respectively.
- ABTs are neuroepithelial in origin, arising from the chemoreceptor cells of the aortic bodies, small clusters of cells situated within the adventitia of the aortic root. These cells respond to alterations in blood oxygen and carbon dioxide tensions and pH by modifying respiration and blood pressure.
- ABTs tend to be slowly growing and locally expansive, although local invasion and metastasis do occur. In one study, metastasis was noted in 50% of cases at the time of necropsy, most commonly to the lungs, spleen, and liver.
- Clinical signs associated with ABT are typically related to the development of pericardial effusion or to compression of adjacent structures. ABT can be noted as incidental findings in patients undergoing echocardiography or thoracic radiography for other indications. These tumors are nonfunctional and so are not associated with cardiovascular disruption through mechanisms other than those described above.

Epidemiology

- Despite being the second most common cardiac tumor in dogs, the incidence of ABT is approximately tenfold lower than that of cardiac HSA, accounting for approximately 8% of all cardiac tumors.
- Brachycephalic breeds, especially the English Bulldog, Boxer, and Boston Terrier, are predisposed to the development of ABT, although dogs of nonbrachycephalic breeds can be affected as well. In various studies, brachycephalic breeds have accounted for 39% to 85% of dogs with ABT. It has been postulated that the chronic hypoxia experienced by dogs of these breeds might explain this predisposition; in both dogs and humans, chronic hypoxia is known to induce hyperplasia of the chemoreceptor organs.
- Some studies suggest that males of predisposed breeds might be at increased risk of developing ABTs.
- Median age at the time of diagnosis of ABTs is 9 years (range, 7–12).

Treatment and prognosis

- Complete surgical resection of heart base tumors is seldom possible because the tumors are highly vascular, invariably located close to major blood vessels, and usually extensive by the time of diagnosis.
- Palliation with pericardiectomy alone often results in prolonged survival with an excellent quality of life in dogs with ABT.
  - In one study, median survival times among dogs with biopsy-confirmed ABT was 730 days in those undergoing subtotal pericardiectomy at the time of surgical biopsy and only 42 days in those not treated with pericardiectomy.
  - Curiously, dogs experienced survival benefit regardless of whether pericardial effusion was present at the time of diagnosis. The authors suggested that pericardiectomy might prolong the time to onset of clinical signs, avoiding early euthanasia at the time of cardiac tamponade development. Indeed, 8 of the 10 dogs not undergoing pericardiectomy were euthanized because of persistent or progressive pericardial effusion.
- Additional therapies that might benefit dogs with HBT experiencing the volume-related compressive effects of these tumors include:
  - External-beam radiotherapy. A 2012 case report first described the use of radiation therapy for a dog with progressive cough related to a large ABT; improvement in the cough and a 50% reduction in tumor volume were noted 25 months after treatment, and the dog was alive 3.5 years after diagnosis despite the need for pericardiectomy and additional radiation therapy. Subsequently, three uncontrolled case series have evaluated outcomes in a total of 40 dogs with HBT treated with stereotactic or conventional fractionated radiotherapy. In the first series, measurable reduction of tumor volume was demonstrated in almost all cases. The second series evaluated symptomatic dogs ($n = 6$) or dogs with progressively enlarging masses ($n = 2$), and reported improvement in clinical signs in five of the six symptomatic dogs and an overall MST of 785 days from the first treatment. In the largest series ($n = 26$ dogs), a partial response or stable disease was noted in in 85% of cases, for a median duration of 333 days.
  - Chemotherapy with toceranib phosphate. A recent report detailing monotherapy with toceranib phosphate in an 8-year-old dog with right-sided heart failure caused by a large neuroendocrine tumor of the heart base described measurable decreases in tumor size and right-heart compression, and a survival time of 10 months postdiagnosis. In a retrospective uncontrolled multicenter study of 27 dogs affected by HBT—24 (89%) of which had clinical signs that ranged from lethargy to cranial vena cava syndrome, and 11 (41%) of which had pericardial

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effusion—treatment with toceranib phosphate alone (n = 14) or combined with other therapies was associated with clinical benefit (i.e., complete response, partial response or stable disease >10 weeks in duration) in 89% of dogs, with a median (range) progression-free interval of 240 days (93–584 days) in those for which it could be determined.
- Endovascular stent placement. Isolated reports have described the successful use of endovascular stents to palliate dogs with compression of the main or branch pulmonary arteries, or cranial vena cava, by large HBT.
- Radiofrequency ablation. The use of percutaneous ultrasound-guided radiofrequency ablation to treat five dogs with clinical signs related to cytologically confirmed ABT was recently described. In that series, no complications were reported and all dogs experienced resolution of clinical signs within 1 week and measurable decreases in tumor size within 1 month of the procedure. Although the follow-up period was relatively short, all dogs were alive and had stable tumor size 6 months postablation; one dog required a second procedure after developing recurrent ascites 8 months postablation.

Mesothelioma

Description and diagnostic features

- Mesothelioma, a malignant neoplasm that affects the epithelial cells lining the serosal surfaces of body cavities (including the pericardial space) is an important cause of pericardial effusion. The reported prevalence in dogs presented with pericardial effusion is 2.5% to 5%. In one study, mesothelioma was responsible for 21% of all neoplastic pericardial effusions.
- Mesothelioma represents a significant diagnostic challenge. Differentiation from IPE (see "Idiopathic Pericardial Effusion" section) is often difficult, even when histopathologic analysis of the pericardium is possible.
  - Mesothelioma typically manifests as a diffuse neoplasm, which complicates its differentiation from IPE based on echocardiographic findings. Less frequently (in one study, only 5 of 15 cases), it is associated with a discrete mass, most often located at the heart base.
  - As noted above, pericardial diseases that lead to effusion, including IPE, often result in mesothelial proliferation and reactivity, with mesothelial cells often bearing characteristics that mimic malignancy. In one study, three (43%) of seven dogs with an ultimate diagnosis of mesothelioma were misclassified as having IPE on initial biopsy.
  - A recent case report described the use of immunohistochemistry to distinguish between malignant mesothelioma, reactive mesothelial cells and adenocarcinoma in a patient with hemorrhagic pericardial effusion, suggesting that this technique could eventually aid in future diagnoses.
- The clinical course of a patient with mesothelioma can be helpful in determining the etiology of the effusion. The accumulation of significant amounts of unrelenting pleural effusion within 4 to 6 months of pericardiectomy should increase the index of suspicion for mesothelioma in a dog that previously received a diagnosis of IPE.

Epidemiology

- Mesothelioma is most common among older dogs of all sizes. Mean age among affected cases at the time of presentation was 8.6 years (range, 4–14) in one study, with both genders equally represented.
- Breed predispositions have not been widely suggested, although one report has described the eventual development of pericardial mesothelioma in five Golden Retrievers that received long-term treatment with intermittent pericardiocentesis for presumed IPE.

Treatment and prognosis

- The long-term prognosis for dogs with pericardial effusion caused by mesothelioma is grave.
- Pericardiectomy, with or without adjuvant chemotherapy, can be used to palliate cardiac tamponade in dogs with mesothelioma. Ultimately most dogs undergoing pericardiectomy die or are euthanized because of the development of severe, recurrent pleural effusion that requires frequent thoracocentesis.
- Adjuvant chemotherapy might prolong survival in dogs with mesothelioma, as suggested by the results of several investigations.
  - In one study of eight dogs with mesothelioma, for which a median survival time of 60 days (range, 15–300) was reported, only dogs receiving chemotherapy lived >120 days; the dog that survived the longest (300 days) was treated with pericardiectomy, doxorubicin, and intracavitary cisplatin.
  - In another study of five dogs with mesothelioma that underwent pericardiectomy, of which three also were treated with adjuvant chemotherapy, median survival time was 13.6 months.
  - More recently, the authors of two larger case series that describe outcomes in a total of 74 dogs with mesothelioma (approximately one-half of which had pericardial involvement) found chemotherapy to be the only treatment significantly and independently associated with survival; median survival times were 234 and 366 days in dogs receiving chemotherapy versus 25 and 74 days in dogs not receiving chemotherapy.

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Key Point

Pericardial mesothelioma represents both a diagnostic and therapeutic challenge. Unlike other cardiac tumors, mesothelioma does not typically form discrete tumor masses that are readily detectable with echocardiography. Even with pericardial biopsy and histopathologic analysis, the diagnosis can be elusive, and only after recurrent pleural or pericardial effusions result in mortality and postmortem examination is the diagnosis confirmed in many cases.

Other Cardiac and Pericardial Tumors

- Cardiac lymphoma, rhabdomyosarcoma, and fibrosarcoma with pericardial effusion have been reported in both dogs and cats.
- Cardiac lymphoma is diagnosed in approximately 1% of dogs with pericardial effusion. Among the various cardiac tumors, cardiac lymphoma is unique because cytologic evaluation of the pericardial fluid establishes the diagnosis in most cases (11 of 12 dogs in one report). Treatment with adjuvant chemotherapy, with or without pericardiectomy, might increase survival time; in one study of 12 dogs with pericardial effusion due to cardiac lymphoma, the median survival times of dogs receiving ($n = 5$) or not receiving ($n = 7$) multidrug chemotherapy were 157 days and 22 days, respectively.
- Other rare cardiac and pericardial tumors that have been reported in association with pericardial effusion include primary and secondary mast cell tumors, intrapericardial lipoma, and intracardiac paraganglioma.

IDIOPATHIC PERICARDIAL EFFUSION

Description and Diagnostic Features

- IPE, also referred to as idiopathic hemorrhagic pericardial effusion, idiopathic pericardial hemorrhage, and idiopathic pericarditis, is a diagnosis of exclusion, made in cases in which no intrapericardial masses are identified after thorough echocardiographic evaluation or exploratory surgery, and the results of ancillary tests such as pericardial fluid analysis fail to disclose etiology.
- As with any idiopathic disease, the diagnosis should be made with caution.
  - Small intrapericardial tumors can elude detection, especially in cases where echocardiography is performed following pericardiocentesis.
  - As discussed previously, mesothelioma should always be considered an important differential diagnosis for IPE.
- Pericardial histopathologic examination from dogs with IPE demonstrates changes consistent with pericarditis of undetermined etiology. Pericardial tissue from dogs with IPE can exhibit a spectrum of changes that include extensive fibrosis, mixed inflammatory cell infiltrate, and perivascular lymphoplasmacytic aggregates, nonspecific findings that are often indistinguishable from those seen in dogs with neoplastic pericardial effusion.
- An immune-mediated autoreactive process has been identified in certain cases of human idiopathic pericarditis; however, immunohistochemistry-based investigations in dogs have not clearly identified a similar process.

Epidemiology

- IPE is the second most common cause of hemodynamically significant pericardial effusion, accounting for 19% to 45% of cases.
- Middle-aged large or middle-aged giant dogs (mean body weight, 29–35 kg) appear to be predisposed to the development of IPE, with some, but not all, authors noting a male gender predilection.
- In general, dogs with IPE tend to be younger at the time of presentation than dogs with neoplastic pericardial effusion, with a median age of approximately 6 to 7.9 years (range, 2–14 years). In one report, there was no age difference between dogs with IPE and mesothelioma.
- Breeds that appear to be predisposed to IPE include the Golden Retriever, Saint Bernard, and Great Dane. Saint Bernards with symptomatic pericardial effusion are reportedly diagnosed at a younger age (mean, 2.6 years) than other commonly represented breeds.

Treatment and Prognosis

- In 21% to 50% of dogs with IPE, long-term resolution is achieved following a single pericardiocentesis. In the remainder, time to recurrence is variable but typically occurs within 1 year (median, 16 weeks; range, 1–250 weeks).
- In dogs with recurrent large-volume pericardial effusion due to IPE, intermittent pericardiocentesis has been used to palliate clinical signs. In one study that described the use of intermittent pericardiocentesis in six dogs managed conservatively for recurrent PE, a median survival time of 5 years was noted, with a median of 2.5 (range, 2–11) treatments required. Other authors have observed median survival times of 3 to 4.5 years in dogs treated with pericardiocentesis alone.
- Despite these encouraging results, many clinicians recommend treating first episodes of IPE with pericardiocentesis, followed by pericardiectomy in cases that develop recurrent effusion.
  - This recommendation is based on a desire to reduce the risk for recurrent life-threatening tamponade and the potential for development of constrictive or effusive-constrictive pericarditis, a serious complication that has been noted in association with long-standing IPE.
  - Furthermore, surgical pericardiectomy permits examination of thoracic and intrapericardial

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structures to rule out other causes of pericardial effusion, such as previously undetected tumors or foreign bodies. The prognostic importance of this was highlighted in a retrospective study of 18 dogs with presumed IPE that underwent thorascopic pericardiectomy and pericardioscopy. In that study, the MST (not reached) for dogs without pericardioscopic abnormalities was significantly longer than the MST (66 days) for dogs in which previously undetected masses, nodules, or adhesions were identified on pericardioscopy.

- Pericardiectomy appears to significantly improve survival times in dogs with IPE compared to conservative management. In most studies in which the use of this treatment was examined, median survival time could not be accurately ascertained because most dogs were still living at the time of writing. Among 13 dogs with IPE undergoing subtotal pericardiectomy in two reports, chances of survival were 100% at the end of the 2-year ($n = 8$ dogs) or 3-year ($n = 5$ dogs) study period.
  - Subtotal (subphrenic) techniques are likely superior to pericardial window techniques for the treatment of dogs with IPE, with significantly longer median survival time and disease-free intervals noted with the former. This might be due to the higher risk of inaccurate diagnosis (i.e., greater risk of missing a neoplastic cause due to less thorough surgical exploration) or "healing" of the pericardium to the epicardium (resealing the pericardial space and allowing recurrence of tamponade) with the window technique compared to subtotal pericardiectomy.

Key Point

Prognosis is very good in dogs with recurrent IPE that undergo pericardiectomy and survive the perioperative period.

- Nonsteroidal antiinflammatories, with or without colchicine, are routinely prescribed to humans with idiopathic pericarditis. Colchicine, when used as an adjunctive therapy, has proven effective in reducing the number of recurrences in these patients, although use of this drug can be limited in some by the development of gastrointestinal side effects. In severe cases or in patients who fail to respond to these measures, systemic corticosteroids can also be prescribed; however, in general, corticosteroids are avoided in the absence of known autoimmune disease because there is evidence that they might increase the incidence of relapse. The safety and efficacy of colchicine, nonsteroidal antiinflammatories, corticosteroids, and any other medical therapies in the management of IPE in small animals have yet to be established.

LEFT ATRIAL RUPTURE

Description and Diagnostic Features

- Left atrial rupture (also known as left atrial perforation, left atrial tear, and left atrial splitting) is an uncommon cause of pericardial effusion that occurs as a complication of severe, chronic degenerative valve disease. Affected animals have advanced mitral valve disease that includes significant left atrial dilatation.
- Pericardial effusion results from linear splits of the left atrial endocardial and myocardial layers, with dissecting hemorrhage and perforation of the epicardium.
- Affected patients have a history consistent with acute pericardial effusion, with most owners reporting an acute onset of generalized weakness or collapse. Additional clinical signs reported in these cases include dyspnea, tachypnea, gastrointestinal signs (i.e., vomiting, hematochezia), neurological signs (i.e., seizures, stupor, ataxia, strabismus, head tilt, circling), and inappetence. Sudden death can occur as well; in one study, 4 (30%) of 11 dogs with left atrial rupture had cardiopulmonary ($n = 3$) or pulmonary ($n = 1$) arrest.
- A loud left apical systolic murmur is usually apparent, even if muffling of the heart sounds is noted.
- Typical echocardiographic findings include small-to-moderate-volume pericardial fluid with a hyperechoic, mobile thrombus within the pericardial space, severe mitral insufficiency, and enlargement of the left atrium.
- Ruptured mitral valve chordae tendineae are frequently noted on necropsy in dogs with left atrial rupture; therefore it has been postulated that chordal rupture might be an important acute event that precipitates left atrial rupture through creation of a sudden rise in left atrial pressure. This observation might also help to explain the frequent concurrence of acute cardiogenic pulmonary edema in these patients at the time of diagnosis (43%–60% in three retrospective reports), which is unexpected in the setting of cardiac tamponade and in clear contrast to other causes of pericardial effusion.
- Left atrial rupture should be suspected in small-breed dogs with a history of advanced mitral valve disease or a heart murmur on physical examination that experience an acute episode of weakness, collapse, or cardiogenic shock and in which pericardial effusion, especially with evidence of blood clot formation, is present on echocardiography. In some cases, the dog might be recovering or even clinically normal at the time of evaluation, and relatively little pericardial fluid might be present.

Epidemiology

- Dogs affected by left atrial rupture are typically older, small dog breeds with chronic degenerative mitral valve disease.

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- Across three retrospective studies, the median age was 10.1 to 12 years (range, 5.8–19 years) and median body weight was 5.8 to 11 kg (range, 2.3–30 kg).

Prognosis and Treatment

- Reported survival times for dogs affected by left atrial rupture vary considerably. In general, the long-term prognosis for this condition is considered poor.
  - In a retrospective study of 14 dogs with left atrial rupture that were seen at a referral emergency service, only 5 dogs (36%) survived to discharge, with most of the nonsurvivors being dead at the time of arrival or euthanized during hospitalization. However, this study was likely affected by case selection bias, as five of the included dogs were identified through a necropsy database.
  - In contrast, the authors of a separate retrospective study of 11 dogs with left atrial rupture noted a median survival time of 203 days (range, 0–760 days), with only one dog not surviving the first 24 hours after diagnosis. Four of the 10 dogs surviving at least 24 hours did not require hospitalization and each of these 10 dogs lived at least 30 days. The authors noted the likelihood of selection bias in their results, as only dogs stable enough to undergo echocardiography were included. However, these findings illustrate that affected dogs can survive the hemodynamic insult of left atrial rupture.
  - More recently, in a case-controlled retrospective study of dogs with myxomatous mitral valve disease that did (n = 15) or did not (n = 15) experience left atrial rupture, median survival times were 52 days (range, 2–1699 days) and 336 days (range, 82–1147 days), respectively. In that study, control dogs were matched by body weight, age, stage of myxomatous mitral valve disease, and left ventricular and left atrial dimensions. Although there was no significant difference in survival times between groups, the investigators noted a high (33%) mortality rate in the first seven days after diagnosis in dogs with left atrial rupture; when these dogs were excluded, median survival time of the remaining dogs was 407 days (range, 30–1699 days). These results suggest a fair prognosis (i.e., comparable to that of severity-matched myxomatous mitral valve disease without left atrial rupture) in dogs with left atrial rupture that survive at least 1 week after the event.[26]
- For cases in which the patient is recovering or clinically normal after an episode of collapse, no specific therapy is necessary other than CHF medications if appropriate (i.e., for dogs with concurrent left-sided CHF). In these cases, complete resolution of both the pericardial effusion and the intrapericardial thrombus is often noted at recheck in 7 to 14 days. Some clinicians, including the author, consider the addition of an afterload-reducing agent (e.g., amlodipine) in recovered, normotensive patients in an effort to decrease afterload and left atrial pressure. However, this approach has not been systematically evaluated.
- Pericardiocentesis is required for cases with hemodynamic compromise due to tamponade. As the possibility of continued hemorrhage exists, blood transfusion and repeated pericardiocentesis can be necessary, with thoracotomy to remove larger clots from the pericardial space and to repair the left atrium in the most severe of cases; the prognosis in such instances is grave.

HYPOCOAGULABLE CONDITIONS

- Pericardial effusions secondary to coagulation disorders are rare causes of clinically significant tamponade.
- Two cases of pericardial effusion and cardiac tamponade secondary to anticoagulant rodenticide intoxication have been reported in the dog; both were successfully treated with pericardiocentesis, vitamin K_1 therapy, and supportive care.
- At the author's institution, coagulation parameters are routinely measured in patients with hemorrhagic pericardial effusion for which an intrapericardial mass is not identified with echocardiography.

INFECTIOUS CAUSES

- Bacterial and fungal infections are occasionally associated with pericardial effusions in dogs, accounting for up to 8.5% of cases of pericardial effusion in some parts of the United States.
- Many cases of septic pericardial effusion associated with bacterial pathogens arise or are thought to arise from intrapericardial foreign body penetration, usually by migrating foxtails (*Hordeum* species), porcupine quills, or perforating gastrointestinal foreign bodies (e.g., wooden skewers). Other cases have been reported after dog fights, extension of local disease, and hematogenous spread. In rare instances, pericardial abscess formation can also be noted. In contrast to most other causes of pericardial effusion, pericardial fluid cytologic and culture results are crucial for the diagnosis of infectious causes.
- In the largest series of bacterial pericarditis reported in dogs (five cases), treatment involved pericardiectomy and removal of foreign bodies, chest drainage, and antibiotic therapy for up to 6 months. All dogs recovered without complications, suggesting that dogs with bacterial pericarditis have a good prognosis when treated aggressively with a combination of surgical and medical therapy.
- Systemic coccidioidomycosis in dogs has been associated with the development of effusive-constrictive pericarditis. Coccidioidomycosis should be considered in young, large dog breeds with pericardial

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thickening and effusion that reside in or have a travel history that includes areas where the soil fungus *Coccidioides immitis* is endemic, such as the southwestern United States.
  - Treatment involves pericardiectomy (with or without epicardial excision), chest drainage, and antifungal therapy.
  - Based on limited published information, the prognosis for cases of coccidioidomycosis with pericardial involvement is guarded. In one case series that examined outcomes in 17 dogs treated with pericardiectomy and epicardial excision, perioperative mortality was 23.5%. However, in dogs that survived to discharge and for which follow-up was available, 82% survived at least 2 years after surgery.
- Other fungal organisms that have been reported as rare causes of pericarditis and pericardial effusion in dogs include *Aspergillus* spp., *Candida albicans*, and *Inonotus tropicalis* (an opportunistic basidiomycete).

MISCELLANEOUS CAUSES OF PERICARDIAL EFFUSION

- Right-sided CHF, uremia, systemic inflammatory conditions (e.g., steroid-responsive meningitis arteritis, systemic inflammatory response syndrome) and myocarditis are known to cause small-volume pericardial effusion in dogs; however, the effusion is typically not of sufficient volume to cause hemodynamic compromise.
- Iatrogenic pericardial effusion has been reported in association with cardiac perforation during cardiac catheterization in several dogs.
- A case of cholesterol-based pericardial effusion has been reported in a dog with hypothyroidism and cardiac tamponade.
- Chylopericardium and cardiac tamponade associated with severe tricuspid valve stenosis that resolved with successful balloon tricuspid valvuloplasty was reported in a 2-year-old Labrador Retriever.
- Intrapericardial granulation tissue, mimicking a tumor and likely representing a resolving hematoma or abscess, was reported in an 8-month-old dog with lethargy and increased respiratory effort.

PERICARDIAL EFFUSION IN THE CAT

- Hemodynamically significant pericardial effusion rarely occurs in cats. Instead, pericardial effusion in this species occurs most commonly in association with CHF, is of small volume, and does not produce cardiac tamponade. Consequently, these effusions are rarely the primary cause for presenting signs; in a study examining 146 cats with pericardial effusion, only 3 (2%) had effusions that were characterized as greater than mild.
- In addition to CHF, diseases associated with pericardial effusion in cats include:
  - Neoplastic diseases, including—most commonly—lymphoma and metastatic carcinoma. Thymoma, mesothelioma, primary and metastatic cardiac HSA, ABTs, and epicardial undifferentiated pleomorphic sarcoma are rare but reported causes of pericardial effusion in cats.
  - Uremia
  - Feline infectious peritonitis
  - Disseminated intravascular coagulation
  - Warfarin toxicity
  - Septic bacterial pericarditis due to hematologic or localized spread of organisms, a migrating grass awn, or a penetrating needle foreign body, have been reported in the cat
  - Aortic dissection
  - Feline hypereosinophilic syndrome
- IPE has not been reported in cats.

CONSTRICTIVE PERICARDIAL DISEASE

DESCRIPTION AND DIAGNOSTIC FEATURES

- Constrictive pericardial disease (also known as constrictive pericarditis) is a rare condition in small animals, thought in most cases to be a complication of chronic pericardial inflammation. The term *effusive-constrictive pericardial disease* has been used to distinguish those cases in which pericardial effusion is also present.
- Pericardial constriction occurs when a thickened, fibrotic pericardium impairs cardiac filling and limits total diastolic volume by creating a noncompliant "shell" in which the heart is encased. The hallmark of constrictive pericarditis is rapid early ventricular diastolic filling that abruptly halts when the limit of ventricular distensibility is reached, a point that is set by the constraining pericardium.
- Signs of right-sided CHF dominate the clinical picture of patients affected by constrictive pericardial disease, and these patients can also have tachypnea, weakness, syncope, weight loss, and diminished exercise tolerance.
- Constrictive pericardial disease is most often idiopathic, but it has also been reported with the following:
  - Infectious agents, including *C. immitis*, *Actinomyces* organisms, *Mycobacterium* organisms, and *A. niger*
  - Chronic, recurrent IPE or chylothorax
  - Metallic foreign bodies
  - Osseous metaplasia
  - Isolated pericardial HSA and pericardial mesothelioma
- Constrictive pericardial disease represents a significant diagnostic challenge. The disease is suspected in patients with signs of right-sided CHF in which other causes have been ruled out, particularly those

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in which the pericardium appears thickened and a small volume of pericardial effusion is present. Although echocardiographic findings (e.g., excessive respiratory variation in transmitral and transtricuspid inflow velocities, abnormalities of interventricular septal motion) can support the diagnosis, the most reliable proof of pericardial constriction is obtained by documenting characteristic abnormalities on invasive catheter-based hemodynamic studies.

EPIDEMIOLOGY

- Medium- to large-breed dogs of various ages seem to be predisposed to the development of constrictive pericardial disease. Mean age in one study was 6.6 years (range, 2.5–9.5 years).

Treatment and Prognosis

- Treatment of constrictive pericardial disease consists of surgical pericardiectomy. In many cases, the visceral pericardium (epicardium) is spared or only minimally affected, and removal of the pericardium is essentially curative. However, for those in which the visceral pericardium is also involved, as seems to be the case particularly in dogs with infectious forms of the disease, epicardial excision might also be required.
- In a study of 10 dogs treated for constrictive pericardial disease with pericardiectomy, 6 had complete resolution of clinical signs and long-term survival. In the remaining four, the most common cause of death in the postoperative period was pulmonary thromboembolism.
- In a case series that examined outcomes in 17 dogs with *C. immitis* treated with pericardiectomy and epicardial excision, perioperative mortality was 23.5%. However, in dogs that survived to discharge and for which follow-up was available, 82% survived at least 2 years after surgery.

MISCELLANEOUS PERICARDIAL DISEASES

PERITONEOPERICARDIAL DIAPHRAGMATIC HERNIA

Description and Diagnostic Features

- Peritoneopericardial diaphragmatic hernia (PPDH) is an uncommon condition defined by an abnormal communication between the peritoneal cavity and pericardial sac, which allows the movement of abdominal structures into the pericardial space. In affected animals, clinical signs are a manifestation of the compressive effects of the displaced abdominal organs on cardiopulmonary structures or of their entrapment, strangulation, or obstruction.
- In dogs and cats, PPDH is almost exclusively a congenital anomaly, likely caused by abnormal formation or incomplete fusion of the septum transversum during embryogenesis.
- Commonly herniated structures include the liver, gallbladder, omentum, and small intestine.
- Clinical signs in affected animals can be nonspecific in nature or can relate to the respiratory (more common in cats) or gastrointestinal (more common in dogs) systems. Tachypnea, dyspnea, exercise intolerance, vomiting, or anorexia are common clinical signs noted by owners. In up to 50% of cases, PPDH can be an incidental radiographic finding in apparently asymptomatic animals or those with seemingly unrelated clinical signs.
- Radiographic signs suggestive of PPDH include enlargement of the cardiac silhouette, indistinct diaphragmatic borders (diaphragmatic "discontinuity"), the presence of soft tissue or fat opacity between the heart and diaphragm, or gas-filled structures within the pericardial space.

Epidemiology

- Prevalences of up to 0.59% have been reported in cats and dogs. The prevalence of PPDH in a large referral population was significantly greater for cats than for dogs (0.062% and 0.015%, respectively).
- Affected animals are usually mature at the time of diagnosis, with median ages of 1 to 4 years and 1.2 to 2.5 years noted for cats and dogs, respectively.
- In retrospective studies of PPDH, cats of long-haired breeds (e.g., Himalayan and Persian) and Weimeraner dogs appear to be overrepresented. Domestic shorthair cats were underrepresented in two reports.
- Although no sex predilection has been reported in cats, some authors have noted a possible predisposition in male dogs.

Treatment and Prognosis

- Dogs and cats with clinical signs related to PPDH can undergo surgical herniorrhaphy, which is associated with resolution of clinical signs in 85% of cases. Perioperative mortality rates of 5.5% to 14% have been reported for this procedure.
- In a recent retrospective study of dogs and cats with PPDH treated with surgery ($n = 34$) or with medical management alone ($n = 24$), long-term survival was similar between treatment approaches. The authors of this study suggest that nonsurgical management of PPDH might therefore be appropriate for animals without clinical signs.

CONGENITAL INTRAPERICARDIAL CYSTS

- Congenital intrapericardial cysts have been reported sporadically in veterinary patients. In two of these cases, the histologic appearance was similar to that of human coelomic pericardial cysts, thought to be formed by abnormal fetal development of the pericardial mesoderm. However, in most veterinary cases, these structures appear to be cystic hematomas, either associated with PPDH or with a stalk

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of tissue that attaches them to the parietal pericardium. For this reason, it has been postulated that these cysts might be a result of omental or falciform fat entrapment during fetal development.
- Clinical signs in affected patients are similar to those noted in other pericardial diseases and are caused by cardiac tamponade caused by compression from the cyst itself and/or any associated pericardial effusion. Most affected dogs are young and presented with abdominal distension, decreased exercise tolerance, and syncope or collapse.
- Echocardiography can be used to identify a mass in the pericardial space that is often loculated in appearance. Pericardial effusion can be present as well.
- Surgical removal of these structures with pericardiectomy is typically associated with resolution of clinical signs and an excellent long-term prognosis in affected animals. Percutaneous centesis of cystic fluid might be required to stabilize the patient before surgery, and advanced imaging (e.g., computed tomography) can provide information to guide surgical planning.

CONGENITAL AND ACQUIRED PERICARDIAL DEFECTS

- Partial or complete congenital absence and acquired defects of the pericardium have been reported in veterinary patients. It is believed that most cases are clinically silent, with most reported instances identified incidentally on necropsy. However, there are rare descriptions of dogs with clinical signs attributable to herniation of cardiac structures, typically through partial pericardial defects.
- In dogs with aneurysmal dilatation of the left or right auricular appendage, whose antemortem clinical findings have been described in three separate reports, pericardial defects with extrapericardial hernation were suspected. Most of these cases were identified incidentally.
- Reported presentations in clinically affected patients with a confirmed pericardial defect include:
  - A 2-year-old Labrador Retriever with clinical signs of CHF, in which approximately 80% of the left ventricle was herniated into the pleural space through a foramen-like defect, with incarceration and acute myocardial infarction.
  - A 14-year-old Maltese dog with frequent syncope, focal atrial tachycardia, and herniation of the left auricular appendage through a defect.

SUMMARY AND CONCLUSIONS

- Chief complaints, histories, and physical examination findings in dogs with pericardial effusions are diverse and often vague.
- Thoracic radiography and electrocardiography can be helpful to diagnose the presence of pericardial effusion; however, these techniques are relatively insensitive. Echocardiography is considered the most sensitive and specific diagnostic tool for the diagnosis and semiquantification of pericardial effusion.
- Most symptomatic pericardial effusions in dogs are neoplastic in origin, with cardiac HSA representing the most common cause.
- Echocardiography is the diagnostic test of choice for the detection of cardiac tumors. This test should be performed before pericardiocentesis if the patient's clinical condition permits.
- In most patients with hemodynamically significant pericardial effusion, pericardiocentesis is necessary for initial stabilization.
- Fluid analysis and cytologic evaluation are rarely helpful in determining the underlying etiology of pericardial effusion; nonetheless these tests are warranted because they can provide a diagnosis in patients with cardiac lymphoma or infectious pericarditis.
- Depending on the underlying cause, the prognosis for dogs with pericardial effusion varies from grave to excellent. Pericardiectomy is the cornerstone of therapy in most cases of recurrent pericardial effusion that have prolonged survival.

Frequently Asked Questions

IS THERE A ROLE FOR THE USE OF DIURETICS IN THE TREATMENT OF PERICARDIAL EFFUSION?

Because many dogs with pericardial effusion show signs of right-sided CHF, it might seem that diuretic agents would be indicated to mobilize accumulated fluid. However, their use is contraindicated in patients with hemodynamically significant pericardial effusion because these agents significantly reduce preload, a determinant of cardiac output on which these animals heavily rely. In this setting, diuretics can significantly worsen systemic hypotension and increase the risk of cardiogenic shock and azotemia. The exception to this rule is in dogs with left atrial rupture associated with advanced degenerative mitral valve disease; these dogs frequently have respiratory difficulty associated with concurrent left-sided CHF and can benefit from cautious diuretic therapy. After pericardiocentesis, fluid accumulation in the abdominal and thoracic cavities due to right-sided CHF will typically spontaneously resolve without the need for diuretic therapy.

WHY DO DOGS WITH PERICARDIAL EFFUSION HAVE PULSUS PARADOXUS?

Pulsus paradoxus refers to the phasic change in peripheral arterial pulse quality that corresponds to the patient's phase of respiration. Specifically, it is defined as a decline in systemic arterial pressure of >10 mm Hg during inhalation; therefore dogs demonstrating pulsus paradoxus have stronger pulse quality during exhalation and weaker or absent palpable pulses during inhalation. Pulsus paradoxus is due to the limitation to cardiac filling imposed by the presence of significant pericardial effusion. When the patient inhales, intrathoracic pressure falls, and venous return to the thorax and right side of the heart is augmented. As the right-sided

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heart is constrained by the presence of the pericardial effusion, right-sided heart filling occurs at the expense of left-sided heart volume. This phenomenon causes left-sided heart volume and systemic cardiac output to fall during inhalation, resulting in poor systemic arterial pulse quality. The opposite effect occurs in exhalation, during which the quality of the systemic arterial pulse increases.

WHAT ARE THE PRIMARY CLINICAL SIGNS IN A DOG WITH CLINICALLY SIGNIFICANT PERICARDIAL EFFUSION?

Commonly dogs with significant pericardial effusion have three cardinal signs, historically referred to as Beck's Triad. These include muffled heart sounds, weak femoral pulses, and jugular venous distension. Careful examination of dogs suspected to have pericardial effusion usually reveals the presence of all three components. Inspection of the jugular veins sometimes necessitates shaving the hair from the patient's jugular groove to properly visualize the vein. Inspection of the jugular vein should be done with the patient standing. Jugular venous distension is often overlooked, but it is a quick and reliable way to help distinguish cardiac from noncardiac causes of abdominal effusion.

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Chapter

12

Congenital Heart Disease

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INCIDENCE

- The incidence of congenital heart disease in dogs has been reported to be 0.46% to 1.6%. The actual incidence is likely higher as some defects result in neonatal death and are unreported.
- The most common congenital heart defects in dogs include pulmonic stenosis (PS), patent ductus arteriosus (PDA), aortic stenosis, ventricular septal defect (VSD), and mitral valve dysplasia (Table 12.1). Several breed predispositions have been identified (see Appendix 1).
- Less common defects in dogs include atrial septal defect (ASD), tricuspid valve dysplasia, cor triatriatum dexter, double-chambered right ventricle, mitral stenosis, and tetralogy of Fallot. Rare defects include persistent truncus arteriosus, tricuspid stenosis, double-outlet right ventricle, and transposition of the great vessels.
- Congenital heart disease is less common in cats than in dogs. The reported incidence is 0.13% to 0.2%. No consistent breed or gender predilections have been adequately demonstrated.
- The most common congenital heart defects in cats include VSD, AV valve dysplasia, PS, aortic stenosis, ASD, tetralogy of Fallot, PDA, and atrioventricular septal defect (formerly endocardial cushion or atrioventricular canal defect).
- Less common defects in cats include double-chamber right ventricle, double-outlet right ventricle, and endocardial fibroelastosis.

HEREDITARY ASPECTS

- Congenital heart diseases are the most common type of heart disease in young dogs and cats but are occasionally diagnosed in adult animals.
- Congenital heart defects are generally recognized in the young animal and usually represent a heritable trait or a defect that originated during gestation. A primary genetic mutation or environmental influences may exist, resulting in significant variation of disease severity.
- The following are four common forms of congenital heart disease that have been shown to be inherited in dogs:
  - PDA in the Poodle
  - Subaortic stenosis (SAS) in the Newfoundland
  - Tetralogy of Fallot in the Keeshond
  - PS in the Beagle

DIAGNOSIS

- In general, a congenital heart defect is suspected when a heart murmur is detected in a young dog or cat (Table 12.2).
- Other supporting clinical features may include the following:
  - Failure to thrive
  - Cyanosis
  - Collapse or seizure
  - Intolerance to activity or exercise
  - Jugular venous distention
  - Electrocardiographic (ECG) abnormalities
  - Radiographic evidence of cardiac enlargement
  - Abnormal pulse quality (e.g., bounding, weak)
- A tentative diagnosis can often be made based on the results of a complete cardiovascular physical examination, routine ECG, and radiography (see Table 12.2).
- The specific diagnosis is confirmed in most cases by echocardiography and rarely by cardiac catheterization, angiography, or oximetry. Information obtained from the echocardiogram is also helpful in determining the severity of the defect, especially if Doppler techniques are used (measurement of blood velocities within the heart).

THERAPY

- Advances since the early 1990s allow for the treatment of certain stenotic lesions by balloon valvuloplasty, which is a technique in which a small balloon located at the end of a cardiac catheter is used, and transcatheter occlusion of PDA with an occluding device. Surgical correction or palliation is possible for certain defects.
- Medical treatment is primarily for the control or prevention of complications, such as congestive heart failure (CHF), arrhythmias, and endocarditis, rather than for correction of the defect. Medical therapy is discussed briefly in this chapter.

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Table 12.1 Classification of Congenital Defects According to Pathophysiology

CANINE	FELINE
Defects Primarily Causing Volume Overload	**Defects Primarily Causing Volume Overload**
Systemic to pulmonary (left-to-right) shunting	
Common	Common
Patent ductus arteriosus	Ventricular septal defect
Ventricular septal defect	Patent ductus arteriosus
Uncommon	Atrial septal defect
Atrial septal defect	Atrioventricular septal defect
Atrioventricular septal defect	Uncommon
(Pseudo) truncus arteriosus	Truncus arteriosus
Valvular regurgitation	Valvular regurgitation
Common	Common
Mitral dysplasia	Mitral dysplasia
Tricuspid dysplasia	Tricuspid dysplasia
Uncommon	
Pulmonic insufficiency	
Aortic insufficiency	
Defects Primarily Causing Pressure Overload	**Defects Primarily Causing Pressure Overload**
Common	Common
Pulmonic stenosis	Dynamic subaortic stenosis
Subaortic stenosis	Uncommon
Uncommon	Pulmonic stenosis
Valvular aortic stenosis	Pulmonary artery branch stenosis
Coarctation and interruption of the aorta	Fixed subaortic stenosis
Cor triatriatum dexter	Valvular aortic stenosis
	Cor triatriatum dexter
	Cor triatriatum sinister
Defects Primarily Causing Cyanosis	**Defects Primarily Causing Cyanosis**
Common	Common
Tetralogy of Fallot	Tetralogy of Fallot
Uncommon	Atrioventricular septal defect
VSD	Uncommon
PDA	Pulmonary to systemic shunting VSD
Tricuspid atresia/right ventricular hypoplasia	Pulmonary to systemic shunting PDA
Double-outlet right ventricle	Double-outlet right ventricle
Transposition of the great vessels	Truncus arteriosus
Truncus arteriosus	
Aorticopulmonary window	
Miscellaneous Cardiac and Vascular Defects	**Miscellaneous Cardiac and Vascular Defects**
Common	Common
Peritoneopericardial diaphragmatic hernia	Peritoneopericardial diaphragmatic hernia
Persistent right aortic arch	Endocardial fibroelastosis
Persistent left cranial vena cava	Uncommon
Uncommon	Persistent right aortic arch
Endocardial fibroelastosis	
Pericardial defects	
Anomalous pulmonary venous return	
Double aortic arch	
Retroesophageal left subclavian artery	
Situs inversus	

PDA, Patent ductus arteriosus; *VSD*, ventricular septal defect.

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Table 12.2 Auscultatory Findings in Congenital Heart Disease

LESION	TIMING	FEATURES	POINT OF MAXIMUM INTENSITY	COMMENTS
Atrial septal defect	Systolic (diastolic)	Ejection (diastolic rumble)	Left base	Systolic murmur ends before S2, which is usually split; murmur (or murmurs) caused by relative pulmonic (tricuspid) stenosis
(Sub) aortic stenosis	Systolic[a]	Ejection (crescendo-decrescendo)	Left base	Often nearly as loud at the right base; diastolic murmur of aortic regurgitation may also occur
Mitral valve dysplasia[b]	Systolic	Regurgitant (holosystolic or pansystolic)	Left apex	May radiate widely
Patent ductus arteriosus	Continuous	Machinery	Left base	Murmur peaks at S2, often radiates to right base and thoracic inlet
Pulmonary hypertension (Eisenmenger syndrome)	None (systolic)	Split S2 (regurgitant)	Left base	Accentuated and split S2; systolic murmur of tricuspid regurgitation or blowing, decrescendo diastolic murmur of pulmonic regurgitation may also occur
Pulmonic stenosis	Systolic	Ejection (crescendo-decrescendo)	Left base	Occasional systolic ejection sound; blowing, decrescendo diastolic murmur of pulmonic regurgitation may occur
Tetralogy of Fallot	Systolic	Ejection (crescendo-decrescendo)	Left base	Murmur due to pulmonic stenosis; may be soft or absent with pulmonary artery hypoplasia
Tricuspid valve dysplasia[b]	Systolic	Regurgitant (holosystolic or pansystolic)	Right midprecordium	Often low pitched and rumbling
Ventricular septal defect	Systolic[a]	Regurgitant (holosystolic or pansystolic)	Right base	Often higher pitched and more cranially located than tricuspid regurgitation; may also be loud at left base

[a]At times, a diastolic murmur of aortic regurgitation may also be present.
[b]Mitral stenosis and tricuspid stenosis are rare but may cause diastolic murmurs over the affected valve and ventricle.
From Ettinger SE, Feldman EF: *Textbook of veterinary internal medicine*, ed 7, St. Louis, MO, 2010, Saunders.

INNOCENT (FUNCTIONAL) MURMUR

- Not all young dogs and cats with heart murmurs have congenital heart disease. Innocent or functional murmurs are created by mild turbulence within the heart and great vessels, and usually diminish in intensity or resolve by 4 to 5 months of age. The following characteristics of innocent murmurs help differentiate them from pathologic murmurs.
  - Innocent murmurs are systolic in timing, usually occurring early in systole and of short duration (ejection type). They are "soft" (i.e., grade 2 or less in intensity) and often have a low pitched, vibrating, or musical quality.
  - Innocent murmurs are usually loudest along the left sternal border and are poorly transmitted. Their intensity may vary with changes in position, heart rate, or phase of respiration, during exercise, and from examination to examination.
  - The most important characteristic of the innocent murmur is that it is heard in the absence of any other demonstrable evidence of cardiovascular disease (e.g., lack of clinical signs or radiographic abnormalities).
  - More intense systolic murmurs (grade 3 or greater) and diastolic murmurs are indicative of cardiac disease and should prompt further diagnostics.

SPECIFIC DEFECTS

PULMONIC STENOSIS

- PS is the most commonly reported congenital defect in the dog, making up 20% to 32% of all congenital heart diseases. PS is most often caused by fusion of the pulmonic valve leaflets at the valvular level. In Bulldogs and Boxers, stenosis of the pulmonary artery can occur at the subvalvular level because of an anomalous origin of the left coronary artery. Supravalvular PS is uncommon.

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PATHOPHYSIOLOGY

- The consequences of this defect are in direct proportion to the severity of the obstruction. The major clinical manifestations are secondary to pressure overload of the right ventricle.
- Hemodynamically, valvular PS results in a pressure gradient across the stenotic valve because of obstruction to right ventricular outflow (Fig. 12.1). The severity of the lesion is directly related to this pressure gradient. Right ventricular hypertrophy is almost always present; the degree varies with the severity of stenosis.
- Turbulence associated with the increase in velocity of blood across the stenotic valve is the cause of the poststenotic dilation in the main pulmonary artery segment.
- The valvular lesion may be characterized by fusion of the valve leaflets, dysplasia of the valvular apparatus, or both.
- Depending on the severity of the right ventricular hypertrophy, some patients will have a dynamic, infundibular stenosis in addition to the fixed valvular stenosis.

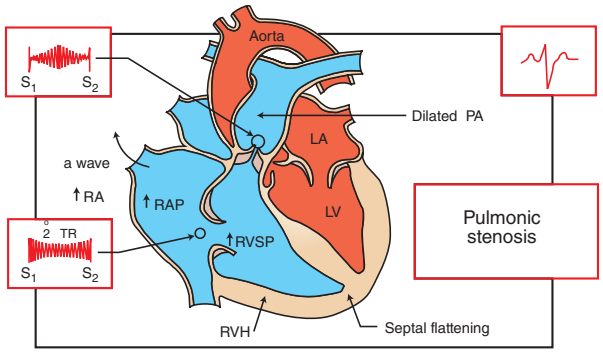


Fig. 12.1 Pathophysiology and genesis of clinical signs in pulmonic stenosis (PS). *LA*, left atrium; *LV*, left ventricle; *PA*, pulmonary artery; *RAP*, right atrial l pressure; *RVH*, right ventricular hypertrophy; *RVSP*, right ventricular systolic pressure. (Modified from Fox PR, Sisson D, Moise NS, eds: *Textbook of canine and feline cardiology: principles and clinical practice*, ed 2, Philadelphia, PA, 1999, WB Saunders.)

DIAGNOSIS

Etiology and Breed Disposition

- PS occurs more often in the dog than in the cat.
- The defect is more common in the following breeds: Airedale Terrier, Beagle, Boykin Spaniel, Boxer, Bull Mastiff, Chihuahua, Cocker Spaniel, English Bulldog, French Bulldog, Samoyed, Schnauzer, and West Highland White Terrier.
- A recessive inheritance pattern is suspected, though environmental and genetic modifiers influence disease presentation.

History and Clinical Signs

- Animals with mild or moderate PS are usually symptom free, and many live apparently normal lives with mild disease.
- Animals with PS that have symptoms also have clinical signs of fatigue secondary to low cardiac output. Exercise-induced syncope may occur because of the limitation in cardiac output imposed by the stenotic valve. Decompensation and signs of right-sided heart failure may occur. Approximately, 35% of dogs with severe stenosis have clinical signs.
- Severe right ventricular hypertrophy can result in myocardial hypoxia and ventricular arrhythmias.

PHYSICAL EXAMINATION

PALPATION

- Animals with moderate-to-severe PS have easily palpable right ventricular impulses. A precordial thrill is usually present at the left lower third or fourth intercostal space.
- Jugular venous distention and pulsations may be present if tricuspid regurgitation or CHF is present.

AUSCULTATION

- A systolic, ejection murmur is heard best at the left lower third or fourth intercostal space. If the murmur is severe, it may radiate over the cranial thorax and be audible at the thoracic inlet.
- A split second heart sound (split S2) due to delayed closure of the pulmonic valve may also be present.
- It can be difficult to differentiate between the murmurs of subaortic/aortic stenosis and PS.

DIAGNOSTIC TESTING

ELECTROCARDIOGRAM

- Signs of right ventricular hypertrophy are present in the majority of cases. There are deep S waves present in leads I and II, and a right shift of the mean electrical axis in the frontal plane.
- Ventricular arrhythmias may be present.

THORACIC RADIOGRAPHS

- The primary radiographic features (Figs. 12.2 and 12.3) include right atrial and ventricular enlargement and enlargement of the main pulmonary segment. Decreased pulmonary vascularity with pulmonary arteries smaller than normal is detected in severe cases.

ECHOCARDIOGRAPHY

- Echocardiography is highly sensitive in the diagnosis and determination of severity of PS.
- Characteristic findings include the following:
  - Hypertrophy of the right ventricular wall and interventricular septum. Because of increased pressures within the right ventricle, the septum is typically flattened (Fig. 12.4). Right ventricular dilation may also be seen.

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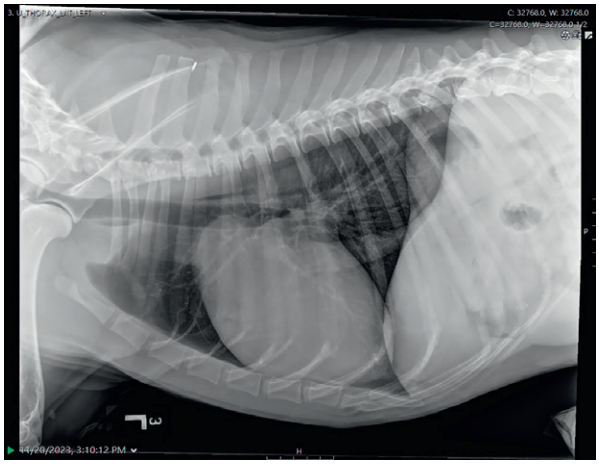


Fig. 12.2 Pulmonic stenosis (left lateral projection) in a dog. There is increased sternal contact of the right side of the heart because of right-sided heart enlargement. There is a bulge in the region of the main pulmonary artery (hat sign).

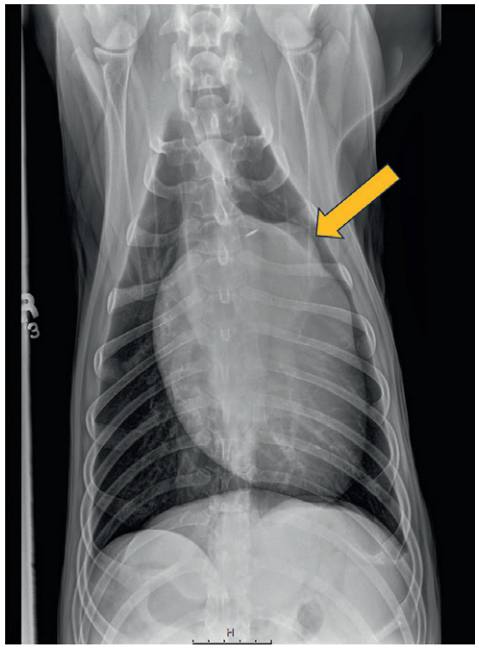


Fig. 12.3 Pulmonic stenosis (dorsoventral projection) in a dog. There is marked right-heart enlargement and prominence of the main pulmonary artery (*arrow*).

- In cases of valvular stenosis, the pulmonic valve cusps may demonstrate variable degrees of fusion and thickening (Fig. 12.4). Fibrous adhesions at the sinotubular junction and pulmonary artery hypoplasia may be present in some cases. A poststenotic dilation of the main pulmonary artery is usually evident.
- Doppler echocardiography allows measurement of blood flow velocity through the stenosis. The pressure gradient across the stenosis can be derived readily from the velocity with the modified Bernoulli equation: pressure gradient between RV and pulmonary artery (mm Hg) = 4 × velocity of PA flow $(m/s)^2$.
- Doppler echocardiography thereby provides a sensitive and noninvasive method to determine the severity of stenosis by estimating the pressure gradient across the obstruction: mild (<50 mm Hg), moderate (50–80 mm Hg), and severe (>80 mm Hg).

OTHER DIAGNOSTIC TECHNIQUES

- Cardiac catheterization and angiography may be performed before an interventional procedure such as balloon valvuloplasty. Selective coronary angiography or computed tomography (CT) angiography is required to rule out the presence of an anomalous left main coronary artery in English Bulldogs, French Bulldogs and Boxers, if surgery or balloon valvuloplasty is considered.

DIFFERENTIAL DIAGNOSIS

- Atrial septal defect: The right ventricular volume overload associated with a left-to-right shunting atrial septal defect may result in a systolic ejection murmur over the pulmonic valve. Such murmurs are usually softer than those associated with PS.
- Innocent or physiologic murmurs: These murmurs are usually much softer than typical PS murmurs and do not typically persist beyond 6 months of age.

NATURAL HISTORY

- Uncomplicated survival to adulthood is common in dogs with mild-to-moderate PS. In general, the natural history correlates with disease severity (pressure gradient) and the degree of right ventricular hypertrophy. Dogs with moderate-to-severe PS may have signs of right-sided CHF, cardiac arrhythmias, syncope with exertion, or sudden death (rare). Concurrent tricuspid valve dysplasia increases the risk of the development of CHF.

THERAPY AND PROGNOSIS

- The need for treatment and the prognosis are dependent on the severity of the defect as determined by Doppler echocardiography or cardiac catheterization. Cases with mild stenosis do not require treatment and have a favorable prognosis. Those with severe stenosis are candidates for an interventional procedure and may have a poor prognosis if uncorrected. Intermediate cases should be followed to monitor for progression of the stenosis.

SURGERY

- The method of surgical correction depends on the type of stenosis present and its severity.
- Some forms of PS may be palliated by circumventing the stenosis with a conduit or by placing a

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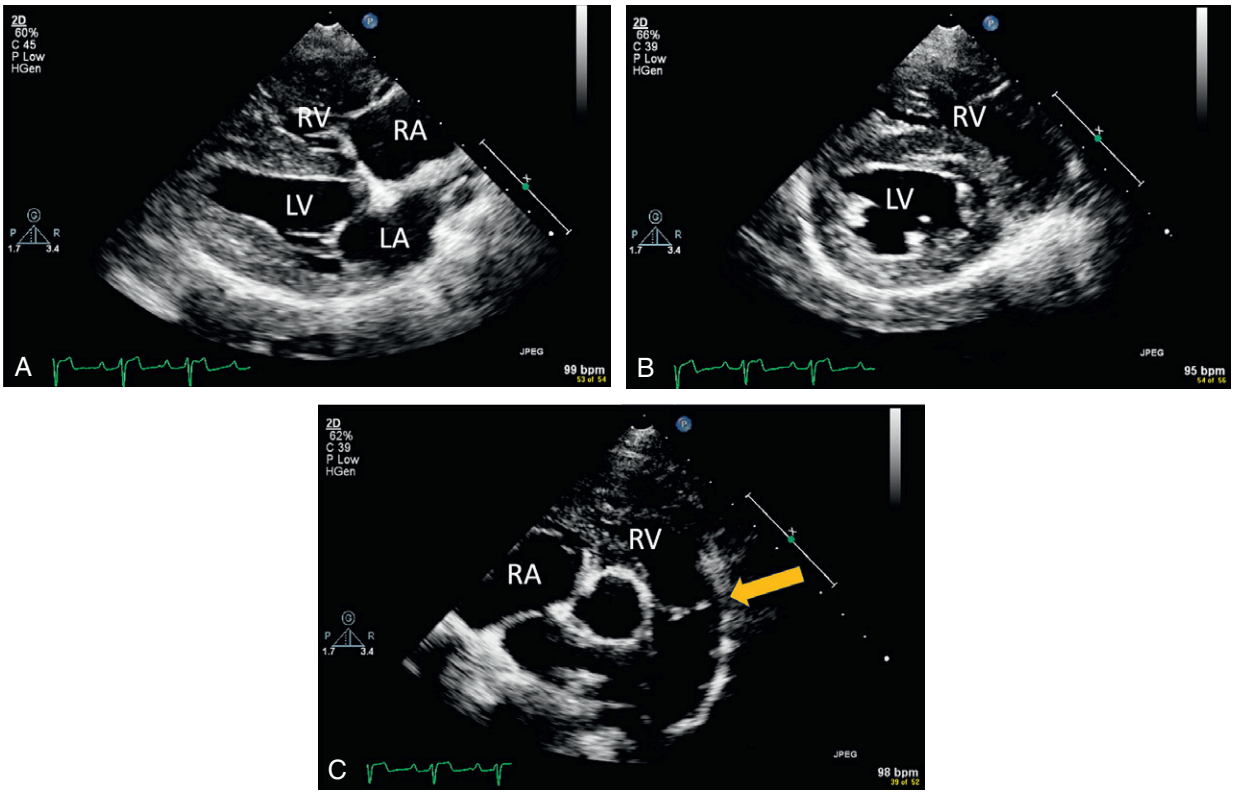


Fig. 12.4 Two-dimensional (2D) echocardiogram obtained from a Boxer with severe pulmonic stenosis (PS). **(A)** The right parasternal long-axis view shows severe thickening of the right ventricle (*RV*) and mild dilation of the right atrium (*RA*). **(B)** The right parasternal short-axis view of the ventricles shows slight flattening of the interventricular septum. **(C)** The right parasternal short-axis view of the heart base shows thickening and fusion/doming of the pulmonic valve leaflets (*arrow*). Note the severely thickened right ventricle *(RV)*. *LV*, Left ventricle; *LA*, left atrium.

patch graft in young animals with valvular PS. In general, open surgical correction is not commonly performed.

BALLOON VALVULOPLASTY

- During balloon valvuloplasty, a special cardiac catheter is guided from the jugular or femoral vein through the stenotic valve. A balloon situated at the end of the catheter is inflated to break open the stenosis. Balloon valvuloplasty is most effective for valvular stenosis when valves are thin and fused and hypoplasia of the annulus is not present.
- This procedure is generally recommended for patients with moderate-to-severe stenosis, particularly those patients with symptoms such as syncope or decreased exercise capacity.
- Balloon valvuloplasty is generally contraindicated in cases of subvalvular PS due to a coronary artery anomaly because avulsion of the coronary artery from the aortic root can occur during balloon inflation.
- Use of a high-pressure balloon may be necessary in patients with pulmonary valve dysplasia and/or a hypoplastic annulus. Additionally, high-pressure balloon valvuloplasty should be considered in dogs that fail to respond to conventional balloon dilation.
- In general, balloon valvuloplasty results in a significant (40%–60%) decrease in the pressure gradient and is maintained in over 60% of patients. Restenosis occurs in 18%–38% of dogs. The long-term effects on survival improvement have not been definitively determined.

TRANSPULMONARY STENT IMPLANTATION

- Transpulmonary stent implantation is a newer treatment approach for dogs with PS that fail to respond to balloon valvuloplasty or dogs with features that make them poor candidates for balloon valvuloplasty (e.g., highly dysplastic valves, anomalous coronary arteries).
- Stent implantation is technically challenging compared to balloon valvuloplasty. However, early experience suggests that this procedure is effective.

Key Point

French Bulldogs, English Bulldogs, and some Boxers may have an anomalous left main coronary artery arising from a single right coronary artery associated with the stenosis that precludes surgical or catheter-based intervention because of the risk of avulsion of the left coronary artery and subsequent death.

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MEDICAL MANAGEMENT

- Signs of right-sided CHF should be treated as described in Chapter 15.
- Beta-blocker therapy (atenolol 1–2.5 mg/kg PO q12h) is routinely administered to patients with moderate-to-severe right ventricular hypertrophy in an effort to decrease myocardial oxygen consumption and to suppress ventricular arrhythmias.

PATENT DUCTUS ARTERIOSUS

- In fetal circulation, the ductus arteriosus serves to shunt maternally oxygenated blood into the aorta, thereby bypassing the nonfunctional lungs. Shortly after birth, several factors contribute to closure of the ductus. Pulmonary vascular resistance drops, vasodilatory prostaglandin levels decrease, and oxygen tension increases, resulting in a marked increase in pulmonary blood flow and vasoconstriction of the ductus. After closure by vasoconstriction, the ductus is permanently closed by fibrous contracture, which produces the ligamentum arteriosum. Failure of the ductus to close is termed *patent ductus arteriosus (PDA)*.

PATHOPHYSIOLOGY

- The consequences of a PDA depend primarily on the diameter of the ductus and the pulmonary vascular resistance, with the large majority of PDA cases resulting in left-to-right shunting.
- Failure of ductal closure is due to an abnormal amount of elastic fibers compared with contractile smooth muscle fibers (extension of the noncontractile wall structure of the aorta into the ductus arteriosus). Varying amounts of normal smooth muscle causes varying degrees of ductal closure; this creates a structure that ranges from a funnel-shaped ductus that narrows on the pulmonary artery side of the ductus (Fig. 12.5) to a tubelike structure without narrowing.
- Blood flow through the ductus is dependent on the relative resistances of the systemic and pulmonary circulation. When pulmonary vascular resistance is normal, blood continually shunts from the aorta (high resistance) into the pulmonary circulation (low resistance). Shunting in this fashion (systemic to pulmonary) is referred to as left-to-right and represents the most common pattern in PDA (Fig. 12.6). If pulmonary vascular resistance increases and exceeds systemic vascular resistance, blood will shunt from the pulmonary artery into the aorta (so-called right-to-left or reversed PDA).
- With a very small diameter ductus, the volume of blood shunted is small, and there may be no hemodynamic effects. In most cases there is significant shunting and one of two major consequences occurs. In cases of persistent left-to-right shunting, volume overloading of the left atrium and left ventricle results in eventual left-sided congestive heart failure. In the rare case of a very large-diameter ductus (sometime referred to as nonresistive), excessive pulmonary blood flow induces dramatic increases in pulmonary vascular resistance, pulmonary hypertension, and shunt reversal (Eisenmenger syndrome), resulting in right-to-left shunting, systemic hypoxemia to the caudal half of the body, and differential cyanosis. If it is going to occur, shunt reversal takes place within the first 6 months of life. Long-term effects of reversed PDA include polycythemia, activity intolerance, and organ failure, as opposed to CHF, which is rare.

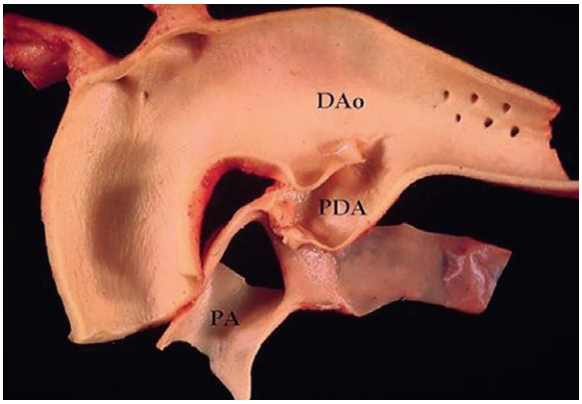


Fig. 12.5 Postmortem specimen, from a young German Shepherd, of the transverse and proximal descending aorta (*DAo*) on top, the pulmonary artery (*PA*) branches ventrally, and a left-to-right shunting patent ductus arteriosus (*PDA*) between them. The aortic end of the PDA is wide open. The PA end is partially constricted. This results in a funnel shape to the PDA. (From Kittleson MD, Kienle RD: *Small animal cardiovascular medicine*, St. Louis, MO, 1998, Mosby.)

DIAGNOSIS

ETIOLOGY AND BREED DISPOSITION

- PDA occurs in both the dog and cat, with higher frequency in the dog. There is also a higher frequency in the female.
- Breeds predisposed or at increased risk include Bichon Frisé, Chihuahua, Cocker Spaniel, Collie, English Springer Spaniel, Keeshond, Labrador Retriever, Maltese, Newfoundland, Poodle, Pomeranian, Shetland Sheepdog, and Yorkshire Terrier.
- The defect has been shown to be inherited in the Miniature Poodle, transmitted as a polygenic trait.
- The German Shepherd often exhibits a large tubular ductus that is prone to shunt reversal and is unamenable to closure with catheter-based occlusion devices.

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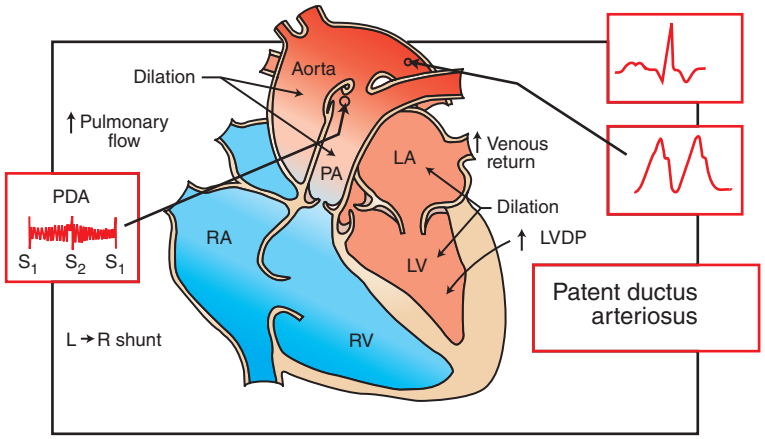


Fig. 12.6 Pathophysiology and genesis of clinical findings in patent ductus arteriosus (*PDA*). *LA*, Left atrium; *LV*, left ventricle; *LVDP*, left ventricular developed pressure; *PA*, pulmonary artery; *RA*, right atrium; *RV*, right ventricle. (Modified from Fox PR, Sisson D, Moise N, eds: *Textbook of canine and feline cardiology: principles and clinical practice*, ed 2, Philadelphia, PA, 1999, WB Saunders.)

HISTORY AND CLINICAL SIGNS

- Clinical signs are related to the degree and direction of shunting and may range from none to severe CHF in left-to-right PDA, or signs of hypoxia and polycythemia in right-to-left PDA.
- Cats seldom display signs of cardiac failure until decompensation is advanced and life threatening.

PHYSICAL EXAMINATION

PALPATION

- Bounding or "water-hammer" arterial pulses are usually present in animals with left-to-right shunting PDA. This type of pulse represents a widened pulse pressure secondary to the loss of diastolic pressure through the ductus and elevation of systolic blood pressure from the volume-overloaded left ventricle. Usually the larger the ductus (and therefore the shunt), the more prominent the arterial pulse.
- In most left-to-right PDA cases, a precordial thrill can be palpated over the cranial left-heart base, and the left apical impulse is prominent. In cases with right-to-left shunting, there is no precordial thrill and the right apical impulse is more prominent.

GENERAL

- Caudal or differential cyanosis is an important physical finding in animals with right-to-left shunting PDA. Owing to the location of the ductus (distal to the arteries supplying the head and forelimbs), cyanosis is limited to the caudal half of the body. Differential cyanosis is best appreciated by examining caudal mucous membranes of the prepuce or vulva as compared with mucous membranes of the mouth.
- Polycythemia (packed cell volume greater than 60%) is usually present in cases with right-to-left shunts but can be absent in animals younger than a year of age.

AUSCULTATION

- A continuous machinery-type murmur is a hallmark of left-to-right shunting PDA. The murmur is loudest in mid-to-late systole and gradually decreases in intensity through diastole. In cases with a very small diameter ductus, this characteristic murmur is restricted to the cranial left-heart base and may be missed if auscultation is limited to the apex. The systolic component of the murmur is usually quite prominent at the cardiac apex; however, a systolic murmur secondary to mitral regurgitation because of annular dilation as the heart enlarges is also possible.
- In cases with a right-to-left shunting PDA, there is no murmur associated with the shunt; however, a split second heart sound (split S2) or diastolic murmur of pulmonic insufficiency as a result of pulmonary hypertension can be present. A murmur of tricuspid regurgitation may be present in some cases. Because right-to-left PDAs start as large left-to-right PDAs that gradually reverse, there is initially a typical continuous murmur early in life, followed by loss of the diastolic component of the continuous murmur and then loss of the systolic component.

DIAGNOSTIC TESTING

ELECTROCARDIOGRAM

ECG abnormalities are present in most cases of PDA and include evidence of left ventricular and left atrial enlargement such as the following:

- Tall R waves are present in leads II and aVF (>3.0 mV).
- P mitrale (widened P wave) is often present.
- Supraventricular arrhythmias (including atrial fibrillation) and ventricular arrhythmias may occur in association with CHF.
- Animals with pulmonary hypertension may show evidence of right ventricular hypertrophy such as

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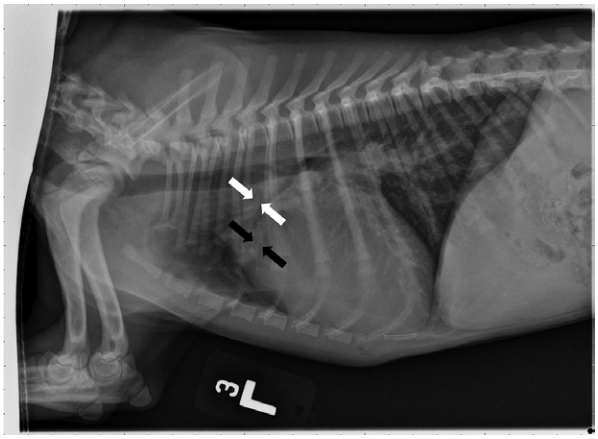


Fig. 12.7 Patent ductus arteriosus (lateral projection) in a 4-month-old puppy. There is generalized cardiomegaly with left atrial enlargement, engorgement of the right cranial lobar artery (*white arrows*), and engorgement of the right cranial lobar vein (*black arrows*).

deep S waves in leads I and II and a right shift of the mean electrical axis.

THORACIC RADIOGRAPHS

- The radiographic signs of PDA (Figs. 12.7 and 12.8) vary considerably with the volume of blood being shunted, the age of the animal, and the degree of cardiac decompensation.
- Dorsoventral radiographs often reveal three prominences along the left cardiac silhouette
  - The aneurysmal bulge of the aortic arch
  - The enlarged pulmonary outflow tract
  - The enlarged left atrial appendage
- Mild-to-moderate left ventricular and left atrial enlargement are usually present.
- Overcirculation of the pulmonary vasculature can often be visualized.
- Severe cardiomegaly, pulmonary venous congestion, and pulmonary edema will be present when there is CHF.
- Right ventricular enlargement and prominent tortuous pulmonary arteries are seen in animals with severe pulmonary hypertension and right-to-left PDA.

ECHOCARDIOGRAPHY

- Echocardiographic changes reflect the volume-overloaded state of the left side of the heart and include left ventricular and left atrial dilation. Left ventricular wall motion (contractility) may be normal or mildly decreased. In most cases, the ductus can be visualized. Transthoracic or transesophageal echocardiography of the ductal shape is an essential diagnostic tool when contemplating catheter-based occlusion of the PDA.
- Shunting of left-to-right blood flow through the ductus can be detected by spectral Doppler echocardiography and can be visualized with color-flow Doppler echocardiography.

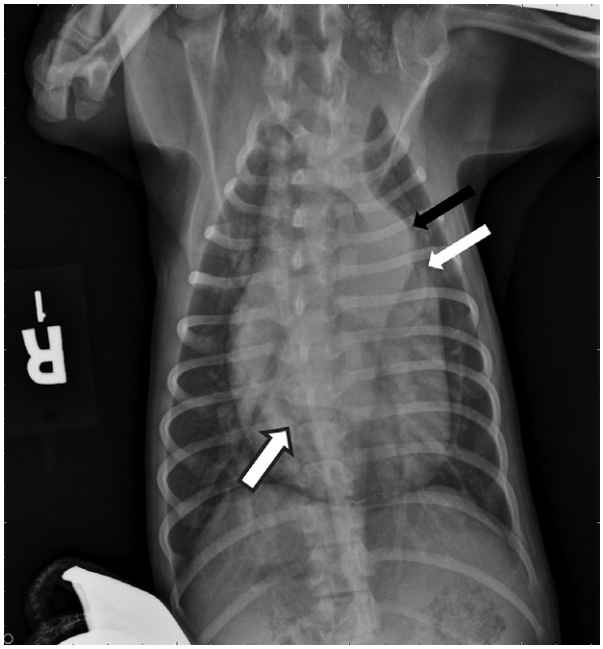


Fig. 12.8 Patent ductus arteriosus (dorsoventral projection) in a 4-month-old Poodle. There is generalized heart enlargement, an aneurysm-like bulge of the descending aorta (*black arrow*), a bulge of the main pulmonary artery (*white arrow*), and enlargement of the left atrium (double-density sign) (*open arrow*).

- In cases with pulmonary hypertension and shunt reversal, right ventricular hypertrophy is evident along with enlargement of the main pulmonary artery; Doppler echocardiography may reveal pulmonic insufficiency.
- Contrast echocardiography can be used to confirm the presence of a right-to-left shunting PDA. In this technique, agitated saline is injected into a peripheral vein during echocardiographic examination of the abdominal aorta to detect right-to-left shunting of blood. The absence of intracardiac shunting with the presence of microbubbles within the abdominal aorta is diagnostic of reversed PDA.

CARDIAC CATHETERIZATION AND ANGIOCARDIOGRAPHY

- Cardiac catheterization and angiocardiography are rarely needed for diagnosis but may be performed to characterize and measure the ductus before catheter-based occlusion.
- The diagnosis of a left-to-right PDA can be made by injecting contrast media into the aortic arch. The simultaneous filling of the main pulmonary artery and the aorta is diagnostic for PDA. Likewise a main pulmonary artery contrast injection can be used to confirm the diagnosis of a right-to-left shunt (simultaneous filling of the main pulmonary artery and aorta).
- Nonselective angiocardiography is performed by injecting contrast material through a large-diameter venous catheter and may be used to support the

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diagnosis of a right-to-left shunting PDA; however, this technique is of limited value in the diagnosis of left-to-right shunts.

DIFFERENTIAL DIAGNOSIS

- Echocardiography or angiocardiographic studies may be used to differentiate PDA from other congenital cardiac defects. Two cardiac defects that can resemble PDA are as follows:
  - Aorticopulmonary window
    - A round or oval communication between the aorta and the main pulmonary artery close to their origin from the heart base. This rare defect usually results in severe pulmonary hypertension and might require angiography or transesophageal echocardiography for differentiation from PDA.
  - Concurrent aortic stenosis and insufficiency
    - The systolic murmur of aortic stenosis and the diastolic murmur of aortic insufficiency combine to mimic the continuous murmur of PDA. Echocardiography easily differentiates this condition from PDA.

NATURAL HISTORY

- Mortality is high in affected, untreated dogs; approximately 64% die before 1 year of age if left untreated.
- Constant volume overload of the left atrium and left ventricle results in chamber dilation, myocardial dysfunction, and arrhythmias.
  - Left-sided cardiac strain and pulmonary congestion lead to CHF.
  - Sudden death following exertion has been observed in young affected dogs.
- When there is right-to-left shunting, progressive polycythemia and hypoxemia occur. Survival to 3 to 5 years of age is not uncommon. Phlebotomy is indicated if the packed cell volume is elevated to greater than 65% and symptoms are present.

THERAPY AND PROGNOSIS

- Because of the high risk of CHF, the vast majority of left-to-right shunting PDA cases should be either surgically ligated or occluded minimally invasively.
- In symptom-free animals with left-to-right shunting PDA, surgical correction of the ductus should be performed as soon as possible. Current surgical ligation success rates are >95%.
- In cases with mild-to-moderate left-sided heart failure, resolution of pulmonary edema must precede anesthesia and surgery.
- In cases with severe heart failure, patient stabilization should be attempted prior to surgery; however, these patients are poor anesthetic candidates. The presence of myocardial failure and atrial fibrillation are negative prognostic indicators.

SURGICAL LIGATION

- Shunting through the ductus may be obviated by surgical ligation. This procedure involves surgically opening the chest and tying suture around (ligating) the ductus to prevent flow through the vessel.

ADVANTAGES

- Best approach for patients with a large PDA with tubular shape (e.g., German Shepherds).
- May be the best approach for very small dogs or cats that are too small for catheter-based occlusion.

DISADVANTAGES

- Requires thoracotomy and hospitalization for 48 to 72 hours with postoperative analgesia.
- Success depends on the experience of the surgeon.
- Correction of PDA is contraindicated when right-to-left shunting is present. Acute right ventricular failure and death will occur because the patent ductus functions as a relief valve for the right ventricle.

CATHETER-BASED OCCLUSION

- A minimally invasive, catheter-based occlusion device can be used for treatment of PDA. This procedure uses delivery of a coil or ductal occluding device into the lumen of the ductus resulting in embolization of the ductus.
- Coils or occlusion devices are selected based on the approximate size of the ductus (as determined by angiography or echocardiography). The device is typically delivered from a catheter passed up the femoral artery and into the ductus from the aorta side (Fig. 12.9), or less commonly through a transvenous approach that accesses the ductus from the pulmonary artery side. Multiple coils may be needed for full occlusion of the ductus, whereas only a single ductal occlusion device (e.g., Amplatz canine ductal occluder or Amplatzer vascular plug) is used.

ADVANTAGES

- A less invasive procedure with a shorter hospitalization period and reduced postoperative morbidity as compared with surgical ligation.
- Success rate very similar to ligation by an experienced surgeon.

DISADVANTAGES

- Might be difficult to catheterize the femoral artery of very small dogs.
- Ineffective in patients with a large, tubular PDA (e.g., PDA in German Shepherds).
- Requires fluoroscopy or transesophageal echocardiography.
- Includes possible complications of embolization of coils or occlusion device, or incomplete closure.

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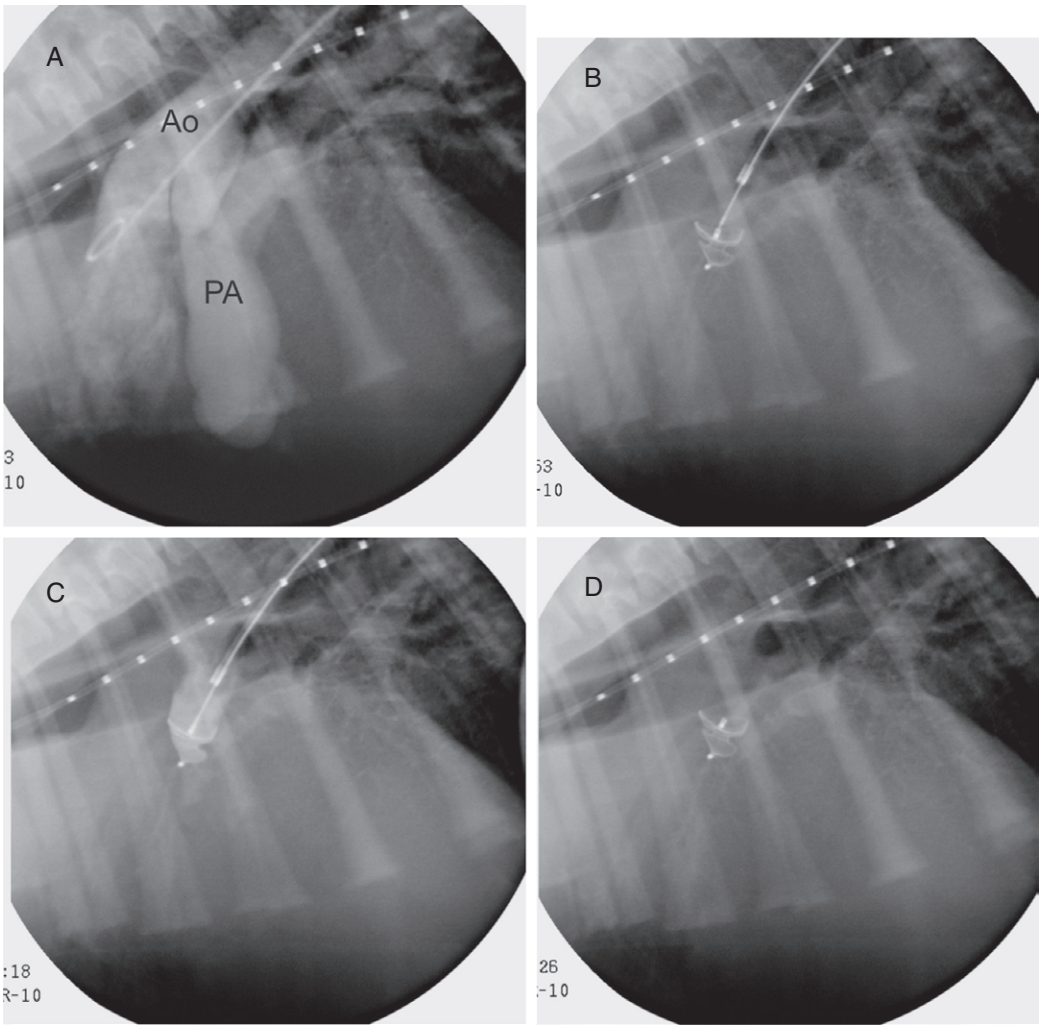


Fig. 12.9 Serial fluoroscopic images and angiocardiograms obtained during a patent ductus arteriosus (PDA) catheter-based device occlusion procedure. **(A)** Angiogram reveals funnel-shaped PDA and left-to-right shunting from the aorta (*Ao*) to the pulmonary artery (*PA*). **(B)** Catheter-based device is positioned across the PDA. **(C)** Angiogram reveals occlusion of the PDA by the device. **(D)** Device is disconnected from the catheter and remains properly positioned across the PDA.

MEDICAL MANAGEMENT

- In cases with CHF before correction, or in cases deemed unacceptable anesthetic-surgical candidates, medical management of CHF is indicated (see Chapter 15). In cases with advanced CHF, medical therapy might be continued beyond closure, especially if severe mitral regurgitation or myocardial failure is present.
- There are no drugs currently effective for closing the patent ductus in dogs and cats.
- In cases with right-to-left PDA, sildenafil is prescribed to manage pulmonary hypertension.

SUBAORTIC STENOSIS

ANATOMY

- Aortic stenosis is a narrowing or reduction of the left ventricular outflow tract (LVOT) at the subvalvular level, fusion of the aortic valve leaflets at the valvular level, or stenosis of the aorta at the supravalvular level. The subvalvular form (SAS) is the most common form in the dog. With this defect, a fibrous band or ring located just below the aortic semilunar valves impedes left ventricular systolic ejection and causes left ventricular concentric hypertrophy.
- Valvular and supravalvular stenosis are relatively rare in the cat and dog. Valvular aortic stenosis is most commonly reported in the Bull Terrier.
- Some patients demonstrate a dynamic SAS associated with systolic anterior motion of the anterior mitral valve leaflet. This condition has been described in patients with fixed aortic stenosis/SAS, mitral valve dysplasia, hypertrophic cardiomyopathy, and other conditions that cause hypertrophy of the interventricular septum.
- Some breeds (Boxers, Bull Terriers, Golden Retrievers) have mildly decreased LVOT areas that

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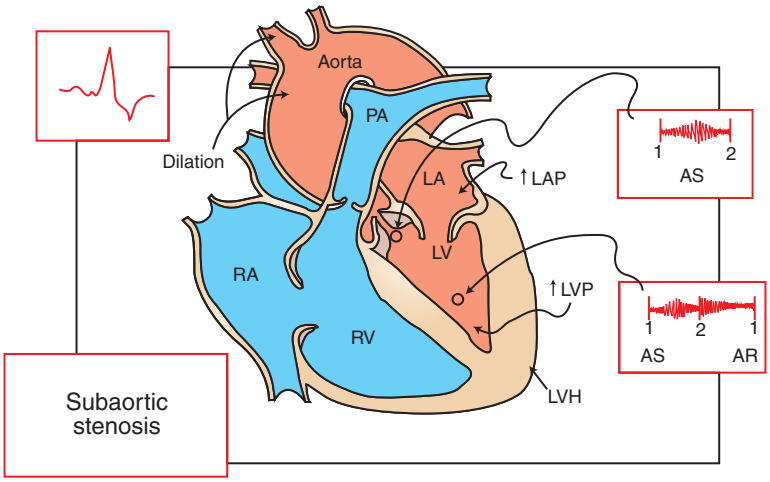


Fig. 12.10 Pathophysiology and genesis of clinical signs in congenital subaortic stenosis (SAS). *AS*, Aortic stenosis; *AR*, aortic regurgitation; *LA*, Left atrium; *LAP*, Left arterial pressure; *LV*, left ventricle; *LVH*, left ventricular hypertrophy; *LVP*, left ventricular pressure; *PA*, pulmonary artery; *RA*, right atrium; *RV*, right ventricle. (From Fox PR, Sisson D, Moise NS, eds: *Textbook of canine and feline cardiology: principles and clinical practice*, ed 2, Philadelphia, PA, 1999, WB Saunders.)

result in mild elevations of transvalvular blood velocity without other structural abnormalities.

PATHOPHYSIOLOGY

- Stenosis of the LVOT results in pressure overload and concentric hypertrophy of the left ventricle (Fig. 12.10). As blood is forced through the stenotic area, velocity increases, resulting in turbulence, a systolic ejection murmur, and a poststenotic dilation of the aorta. The velocity of blood through the lesion is directly proportional to the severity (i.e., cross-sectional area of the LVOT) of disease. Left ventricular hypertrophy results in increased myocardial oxygen demand and ischemia, as well as diastolic dysfunction.
- Myocardial hypertrophy, decreased capillary density, and increased wall tension all contribute to produce myocardial hypoxia/ischemia. Focal areas of myocardial infarction and fibrosis, particularly involving the papillary muscles, small coronary vessels, and subendocardium, are common in patients with severe stenosis. These cases are prone to sudden death, presumably from fatal ventricular arrhythmias induced by hypoxia.
- Left-sided CHF is uncommon.
- The presence of aortic stenosis or SAS increases the risk of the development of infective endocarditis.

DIAGNOSIS

ETIOLOGY AND BREED PREDISPOSITION

- An autosomal dominant inheritance pattern has been identified in the Newfoundland and is associated with mutation of the *PICALM* gene.
- Other commonly affected breeds include Bouvier de Flandres, Boxer, English Bulldog, German Shepherd, German Shorthair Pointer, Golden Retriever, Great Dane, Rottweiler, and Samoyed.
- Bull Terriers are predisposed to valvular aortic stenosis characterized by thickened and calcific aortic valve leaflets and a hypoplastic aortic valve annulus.

HISTORY AND CLINICAL SIGNS

- In mildly to moderately affected cases, there are often no clinical signs, and the suspicion of aortic stenosis occurs when a murmur is detected during routine examination.
- In severe cases, owners may report failure to thrive, exercise intolerance, collapse, and labored breathing. In some cases, the first clinical sign is sudden death.

PHYSICAL EXAMINATION

PALPATION

- Animals with aortic stenosis can have a strong left ventricular precordial impulse, and those with severe stenosis usually have a palpable precordial thrill over the aortic valve area. Femoral pulses are often weak and late rising (pulsus parvus et tardus).

AUSCULTATION

- SAS causes a systolic ejection-type murmur heard best over the left fourth intercostal space at the level of the costochondral junction (aortic valve area). Frequently the murmur radiates up the carotid artery and can be auscultated in the midcervical area. Occasionally the murmur may be loudest in the right third or fourth intercostal space midway up the thorax. Rarely a concurrent diastolic murmur of aortic insufficiency is present.

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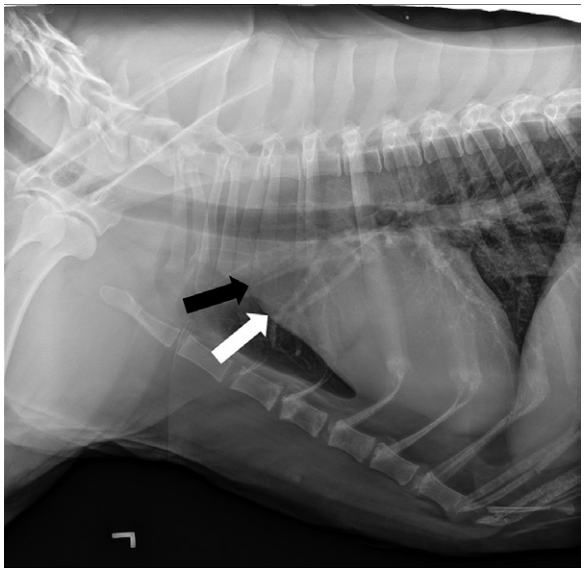


Fig. 12.11 Aortic stenosis (lateral projection). There is enlargement of the aortic arch cranial to the heart base (*arrows*).

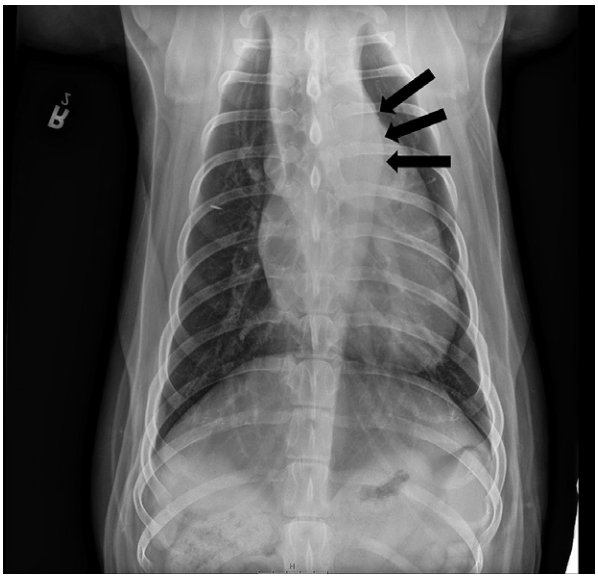


Fig. 12.12 Aortic stenosis (dorsoventral projection). There aortic arch is prominent (*arrows*) and associated with widening of the cranial mediastinum.

DIAGNOSTIC TESTING

ELECTROCARDIOGRAM

- The ECG may be normal. Alternatively there may be evidence of left ventricular hypertrophy (tall R waves in lead II and lead aVF [>3.0 mV]).
- Ventricular or supraventricular arrhythmias are occasionally detected.
- ST segment depression or elevation at rest or after exercise is occasionally detected in severe cases and is indicative of myocardial ischemia.

THORACIC RADIOGRAPHS

- Radiographic changes tend to parallel severity of the stenosis. Characteristic features include enlargement of the aortic arch (poststenotic dilation) and prominent left ventricle (Figs. 12.11 and 12.12).
- Left atrial enlargement is rare but can be present in advanced cases with concurrent mitral insufficiency.

ECHOCARDIOGRAPHY

- Echocardiography is the most sensitive noninvasive method to visualize the narrowed LVOT and secondary cardiac changes. In most cases, there is significant left ventricular hypertrophy. In severe cases, the papillary muscles and myocardium become hyperechoic secondary to ischemia and fibrosis.
- In a similar manner as PS, Doppler echocardiography allows measurement of blood flow velocity through the stenosis and provides data regarding severity. Pressure gradients <50 mm Hg are considered mild, 50 to 80 mm Hg are considered moderate, and >80 mm Hg are considered severe.

Key Point

The differentiation between patients with trivial to mild stenosis and normal patients with a physiologic acceleration of transaortic velocity can be difficult, if not impossible. A diagnosis of trivial to mild subaortic/aortic stenosis based solely on elevations of transaortic velocity can be problematic; the diagnosis should include a combination of the following: accelerated transvalvular velocities, turbulent blood flow, visualization of an anatomical lesion, concurrent aortic insufficiency, or a distinct "step up" in velocity across a discreet region of the outflow tract. Most examiners consider LVOT velocities >2.25 m/s indicative of SAS.

CARDIAC CATHETERIZATION

- Cardiac catheterization and angiography are occasionally used when multiple cardiac defects are suspected.
- Angiography illustrates the stenosis and poststenotic dilation. Pressure measurements obtained during selective catheterization are used to determine the location and pressure gradient across the stenosis.

NATURAL HISTORY

- The clinical manifestations of SAS are variable. Up to 70% of dogs with severe stenosis may die suddenly during the first 3 years of life. Cardiac arrhythmias are common in affected dogs. In others, CHF may develop later in life. Dogs with SAS are predisposed to infective endocarditis or the aortic valve, which is often a life-threatening complication. Other affected individuals may demonstrate symptoms such as syncope or exercise intolerance.

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THERAPY AND PROGNOSIS

SURGERY

- Surgical resection of the SAS lesion requires cardiopulmonary bypass and is not indicated for patients with mild stenosis. Surgery in cases of severe SAS has not been associated with improved long-term survival and is rarely performed because of the expense and morbidity associated with the procedure.

BALLOON DILATION

- Balloon valvuloplasty is infrequently performed and most often contemplated in cases with a discrete, membrane-like stenosis. Special cutting and high-pressure balloons can be used. Significant reductions in the pressure gradient across the stenosis can be achieved immediately after balloon dilation; however, restenosis is likely, and balloon dilation has not been consistently associated with improved long-term outcome.

MEDICAL MANAGEMENT

- Because of the lack of effective interventional techniques, medical management forms the basis of therapy in most cases of SAS. A beta-blocker (atenolol most commonly) is used to reduce myocardial oxygen demand. They might also be of some benefit in reducing the frequency of arrhythmias. Renin-angiotensin-aldosterone antagonists or inhibitors such as spironolactone or angiotensin-converting enzyme inhibitors are occasionally prescribed in an attempt to reduce myocardial remodeling or fibrosis. The benefits of medical therapy are largely theoretic in nature and have not been consistently shown to improve long-term outcome.
- Antibiotics should be administered when bacteremia is suspected or likely (e.g., dental procedures) to reduce the chance of infective endocarditis.
- Diuretic therapy is indicated in cases of CHF.
- Positive inotropes such as pimobendan or digoxin are contraindicated in SAS and in other forms of outflow obstruction and should not be used.

VENTRICULAR SEPTAL DEFECT

ANATOMY

- The interventricular septum separates the left ventricle and the right ventricle. It is muscular at the apex and tapers to a membranous portion at the heart base near the origin of the aorta. VSDs may occur in any area of the septum but are most commonly located in the membranous portion (so-called perimembranous VSDs).
- VSDs may occur as isolated defects, may coexist with concurrent defects (e.g., PDA and atrial septal defect), or may be a component of more complex cardiac anomalies (e.g., tetralogy of Fallot).

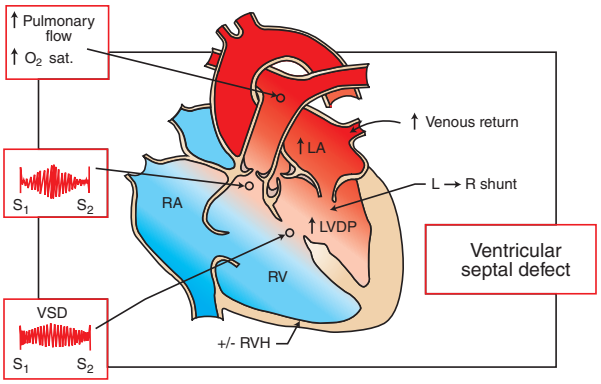


Fig. 12.13 Pathophysiology and genesis of clinical findings in ventricular septal defect. *LA*, Left atrium; *LVDP*, left ventricular developed pressure; *RA*, right atrium; *RV*, right ventricle; *RVH*, right ventricular hypertrophy. (Modified from Fox PR, Sisson D, Moise NS, eds: *Textbook of canine and feline cardiology: principles and clinical practice*, ed 2, Philadelphia, PA, 1999, WB Saunders.)

PATHOPHYSIOLOGY

- The two major factors that determine the consequences of a VSD are the cross-sectional area of the VSD and the relative pressures (or resistances) of the right or left ventricles and pulmonary and systemic circulations, respectively. When left and right ventricular pressures and vascular resistances are normal, blood will shunt left to right, as the left ventricular systolic pressure is roughly five times greater than right ventricular systolic pressure (Fig. 12.13). In the typical VSD (left-to-right shunting perimembranous defect), most of the blood is shunted into the right ventricular outflow tract, or the main pulmonary artery. This shunt creates a large volume overload to the pulmonary circulation, the left atrium, and the left ventricle. Thus, when pulmonary resistance remains relatively normal, despite the left-to-right shunting, the right ventricle is spared most of the effects of the shunting.
- Small left-to-right shunting VSDs may be of no hemodynamic significance, although they may predispose the animal to the development of endocarditis. In rare cases, some small defects close spontaneously.
- Moderately sized defects may allow significant shunting and produce clinical signs of left-sided CHF.
- Very large defects may create a functional single ventricle with equilibration of left and right ventricular pressures. If right ventricular resistance increases (e.g., pulmonary hypertension), right ventricular pressure will increase proportionately, thereby decreasing the shunt volume. If right ventricular pressure increases to a point where it exceeds left ventricular pressure, blood will shunt right to left, and signs of generalized cyanosis and polycythemia may develop.

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DIAGNOSIS

ETIOLOGY AND BREED PREDISPOSITION

- Breed studies in the Keeshond have shown the defect to be polygenic.
- The English Bulldog appears to have a higher incidence than other breeds, but the defect is seen in many purebred and mixed-breed dogs.

HISTORY AND CLINICAL SIGNS

- The clinical course of animals with VSD is variable and, to a large extent, dependent on the size of the defect and the ventricular pressures. Small defects may cause little or no functional disturbances and may cause no clinical signs. Moderate-to-large defects usually produce signs of left-sided failure subsequent to the chronic left-sided volume overload. In cases that develop pulmonary hypertension, signs of right-sided failure, cyanosis, and possibly polycythemia will predominate.

PHYSICAL EXAMINATION

PALPATION

- In many cases, a precordial thrill is present at the right-sided heart base (third to fourth intercostal spaces, just above the costochondral junctions). Volume overloading of the left ventricle often accentuates the left-sided impulse. In cases with significant pulmonary hypertension, precordial thrills are unusual, and the right apical impulse is accentuated.

AUSCULTATION

- Typically the murmur is harsh, holosystolic, and heard best at the right sternal border, at the second through fourth intercostal spaces.
- A murmur of functional PS may be heard over the pulmonic valve area. This is a result of the extra volume of blood passing through the pulmonic valve and not related to a structural valve problem.
- A split second heart sound (split S2) occasionally is present because of a slightly prolonged right ventricular ejection time.
- When the defect causes destabilization of an aortic valve cusp, a diastolic murmur of aortic insufficiency may be present in addition to the systolic murmur. This combination is referred to as a to-and-fro murmur rather than a continuous murmur.
- In cases with progressive pulmonary hypertension, elevations in right ventricular resistance may result in net right-to-left shunting. With very large defects, severe pulmonary hypertension, or both, a murmur may be absent because of the lack of a pressure differential across the septum.

DIAGNOSTIC TESTING

ELECTROCARDIOGRAM

- ECG abnormalities usually parallel the degree of hemodynamic compromise.
- The ECG may be normal when a small defect is present.
- The ECG may show evidence of left, right, or biventricular enlargement, depending on hemodynamic consequences of the shunt.
- Right bundle branch block or a splintered QRS complex indicative of a ventricular conduction abnormality may also be observed.

THORACIC RADIOGRAPHS

- Small defects usually do not cause radiographic changes.
- Left ventricular enlargement, left atrial enlargement, and increased pulmonary artery prominence are seen with larger defects resulting in significant left-to-right shunting.
- Right ventricular enlargement accompanied by prominent tortuous pulmonary arteries may be present in cases of pulmonary hypertension and right-to-left shunting.

ECHOCARDIOGRAPHY

- The diagnosis of VSD can usually be confirmed by routine echocardiography. Typical findings for a moderate-to-large shunt include left ventricular and left atrial dilation and a defect in the interventricular septum with two-dimensional (2D) echocardiography. Distinguish the septal dropout normally present in the membranous septum from a VSD. In most VSD cases, the interventricular septum is slightly blunted just apical to the defect, whereas the septum tapers gradually in normal animals.
- Contrast echocardiography will confirm the presence of a right-to-left shunting VSD. Agitated saline is rapidly injected into a peripheral vein, resulting in the appearance of microbubbles (contrast) within the heart. In the absence of a right-to-left shunt, all contrast remains right sided. In right-to-left shunting VSD, microbubbles will be seen traversing the defect.
- Doppler echocardiography will confirm the shunting of blood through the defect (Fig. 12.14).

Key Point

- High-velocity flow across the VSD and normal transpulmonic valvular velocities typically indicate that the defect is associated with a small shunting volume. These "restrictive" shunts are often hemodynamically insignificant and are unlikely to be associated with clinical signs.
- Low-velocity flow across the VSD and increased transpulmonic valvular velocities typically indicate that the defect is associated with a large shunting volume. These "unrestrictive" shunts are often hemodynamically significant and are likely to be associated with clinical signs.
- Low-velocity flow across the VSD and decreased transpulmonic valvular velocities typically indicate development of pulmonary hypertension and net right-to-left shunting.

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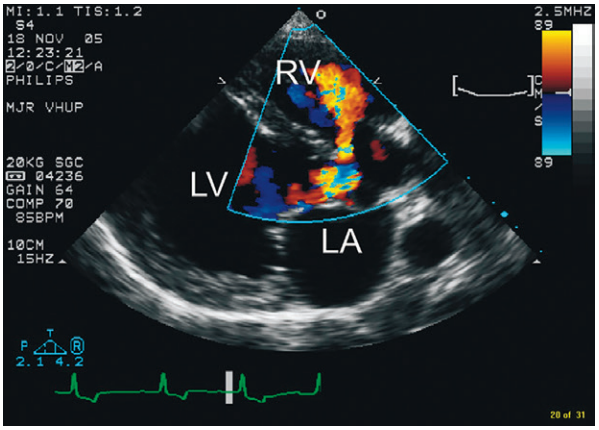


Fig. 12.14 Right parasternal long-axis, two-dimensional echocardiogram obtained from a 9-week-old Golden Retriever puppy with a right-sided systolic murmur. Note the aliased color-flow pattern crossing the interventricular septum. This finding is consistent with a left-to-right shunting ventricular septal defect. *LA*, Left atrium; *LV*, left ventricle; *RV*, right ventricle.

OTHER DIAGNOSTIC PROCEDURES

- Cardiac catheterization and determination of oxygen saturation of blood from individual cardiac chambers may be used to confirm shunting. Left-to-right shunts produce a step up in oxygen saturation in the right ventricle compared with the right atrium.

THERAPY AND PROGNOSIS

- The prognosis and need for therapy are dependent on the severity of the defect. Small defects are usually well tolerated with little to no effect on longevity. Larger septal defect with significant shunting might require surgical or catheter-based device closure or eventual medical therapy for CHF. The proximity of the VSD to the aortic valve increases risk of infectious endocarditis, and antibiotic prophylaxis before procedures likely to produce bacteremia (e.g., dental cleaning) is recommended.

SURGERY OR CATHETER-BASED DEVICE CLOSURE

- Open heart techniques to surgically close or patch the VSD are curative; however, these techniques are available at a very limited number of locations and are often prohibitively expensive. Palliative reduction of the shunt can be accomplished by pulmonary artery banding, a technique resulting in elevation of the right ventricular systolic pressure. As right ventricular pressure increases, the shunt volume decreases, and the pulmonary circulation is spared the deleterious effects of chronic volume overload. Catheter-based closure of the VSD with an occlusion device is reported but technically difficult because of the proximity of the VSD to the aortic valve. A hybrid approach combining the use of surgery and catheters/devices to VSD closure has also been described.

MEDICAL MANAGEMENT

- In patients with significant shunting, medical management of CHF may be required. Treatment should be tailored to the type and degree of failure (see Chapter 15). In cases of right-to-left VSD, the long-term consequences of hypoxemia and polycythemia are managed with activity restriction and periodic phlebotomy.

PROGNOSIS

- Prognosis is excellent for animals with small defects or for those with surgically corrected defects. Cases with moderate-to-large defects have a variable clinical course and prognosis depending on shunt volume.

TETRALOGY OF FALLOT

- Tetralogy of Fallot is a cyanosis-producing defect and results from a combination of PS, VSD, right ventricular hypertrophy, and varying degrees of dextroposition and overriding of the aorta. The right ventricular hypertrophy is secondary to the obstruction in right ventricular outflow. The PS may be valvular, infundibular, or both.

PATHOPHYSIOLOGY

- The hemodynamic consequences of tetralogy of Fallot depend primarily on the severity of the PS and the size of the VSD.
- The direction and magnitude of the shunt through the septal defect are dependent on the degree of right ventricular obstruction. If the PS is mild and right ventricular pressures are only modestly elevated, then blood will shunt primarily from left to right. Pathophysiologically, these cases are similar to VSD cases with pulmonary artery banding (i.e., the mild right ventricular obstruction protects the pulmonary vasculature from excessive shunting).
- When PS is severe, the elevated right ventricular pressures will result in right-to-left shunting. Consequences include reduced pulmonary blood flow (resulting in fatigue and shortness of breath) and generalized cyanosis (resulting in weakness).
- Because of the shunting of venous blood into the aorta and consequent hypoxemia, the kidneys are stimulated to release erythropoietin. Chronic elevations in erythropoietin result in polycythemia. The increased blood viscosity associated with polycythemia can have significant hemodynamic effects, resulting in sludging of blood and poor capillary perfusion. Animals with severe polycythemia may experience seizures.

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DIAGNOSIS

ETIOLOGY AND BREED PREDISPOSITION

- Breeds predisposed to tetralogy of Fallot include the English Bulldog, Keeshond, Miniature Poodle, Miniature Schnauzer, and Wire-Haired Fox Terrier. This defect has also been recognized in other canine breeds and in cats.

HISTORY AND CLINICAL SIGNS

- Typical historical features include stunted growth, exercise intolerance, cyanosis, collapse, and seizure activity.

PHYSICAL EXAMINATION

PALPATION

- A precordial thrill may be felt in the third left intercostal space near the costochondral junction.

AUSCULTATION

- In most cases, a murmur of PS is present. The intensity of the murmur is attenuated when severe polycythemia is present.

DIAGNOSTIC TESTING

ELECTROCARDIOGRAM

- A right ventricular enlargement pattern is usually present.
- Arrhythmias may be present.

THORACIC RADIOGRAPHS

- Variable right-sided heart enlargement is present.
- Pulmonary vessels are undersized, and the main pulmonary artery is often diminished.

ECHOCARDIOGRAPHY

- Echocardiography will confirm the diagnosis. Contrast echocardiography will usually demonstrate right-to-left shunting at the level of the VSD. Flow through the defect can also be determined by Doppler echocardiography.

CARDIAC CATHETERIZATION

- Selective angiocardiography of the right ventricle demonstrates simultaneous filling of the aorta and pulmonary artery in cases with right-to-left shunts. Oximetry can be performed to help quantify the degree of right-to-left shunting.

THERAPY AND PROGNOSIS

SURGERY

- Surgical correction of tetralogy of Fallot is not commonly performed because of the attendant mortality and expense. Palliative surgical options include the modified Blalock-Taussig anastomosis, wherein a vascular graft is placed between the left subclavian artery and pulmonary artery to increase pulmonary blood flow. This procedure is generally effective in reducing signs of pulmonary hypoperfusion and systemic hypoxia. In some cases, palliation can be provided by reducing the severity of the PS with balloon valvuloplasty.

MEDICAL MANAGEMENT

- Nonselective beta-adrenergic blockade (e.g., propranolol) has been used to reduce the dynamic component of right ventricular outflow obstruction and to attenuate beta-adrenergic-mediated decreases in systemic vascular resistance, which increase the magnitude of right-to-left shunting during activity.
- Polycythemia should be controlled by periodic phlebotomy. When the packed cell volume exceeds 68%, intervention is indicated. Up to 20 mL/kg of blood can be removed and replaced with a crystalloid solution such as lactated Ringer solution or saline.
- If the required frequency of phlebotomy is poorly tolerated by the patient hydroxyurea, an oral myelosuppressive agent can be used. However, administration of this drug requires close patient monitoring with periodic complete blood and platelet counts.

ATRIOVENTRICULAR VALVE DYSPLASIA

- Congenital malformation of the mitral and tricuspid valves has been reported in dogs and cats. Malformation of the valve may result in a range of hemodynamic consequences, including valvular regurgitation, mitral or tricuspid stenosis, and dynamic left ventricular outflow obstruction.
- Congenital malformation of the mitral valve complex (mitral valve dysplasia) is a common congenital cardiac defect in the cat. In addition, several canine breeds are predisposed, including Bull Terriers, German Shepherds, and Great Danes. Tricuspid valve dysplasia has been shown to have a genetic basis in Labrador Retrievers.

ANATOMY

- Mitral valve dysplasia most commonly results in valvular insufficiency and systolic regurgitation of blood into the left atrium.
- Any component of the atrioventricular valve complex (valve leaflet, chordae tendineae, papillary muscles) may be malformed. Often more than one component is defective. A wide spectrum of valvular malformations has been described: thickening of the valve leaflets; incomplete separation of the valvular structures from the ventricular wall; shortening or elongation, thickening, and fusion of the chordae tendineae; and malpositioning or malformation of the papillary muscles.

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PATHOPHYSIOLOGY

- Malformation of the atrioventricular valve complex can result in significant valvular insufficiency. In the case involving the mitral valve, chronic mitral regurgitation leads to volume overload to the left heart, which results in dilation of the left ventricle and atrium in the same manner that occurs in chronic degenerative valvular disease (see Chapter 7).
- When mitral regurgitation is severe, cardiac output decreases, resulting in signs of cardiac failure.
- Dilation of the left-sided chambers predisposes affected animals to arrhythmias. Severe atrial enlargement increases the risk of tachyarrhythmias such as atrial fibrillation.
- In some cases, malformation of the mitral valve complex causes a degree of valvular stenosis in addition to insufficiency.
- If the tricuspid valve is involved, then atrial and ventricular enlargement secondary to volume overload with subsequent systemic venous hypertension and right-sided CHF is a likely outcome, if regurgitation is severe.

DIAGNOSIS

HISTORY AND CLINICAL SIGNS

- Clinical signs correlate with the severity of the defect. Affected animals usually display signs of left-sided heart failure, including weakness, cough, and exercise intolerance if the mitral valve is affected. Alternatively signs of right-heart failure (abdominal distention associated with ascites) may be present if the tricuspid valve is affected.

PHYSICAL EXAMINATION

PALPATION

- Affected animals may have a precordial thrill over the left cardiac apex (mitral valve) or right cardiac apex (tricuspid valve).

AUSCULTATION

- A holosystolic or pansystolic murmur of mitral regurgitation is most prominent at the left cardiac apex. A diastolic heart sound (gallop) is present in some cases. The murmur of tricuspid regurgitation is heard most clearly at the right cardiac apex. Some patients with clinically significant tricuspid regurgitation may have "silent" regurgitation.

DIAGNOSTIC TESTING

ELECTROCARDIOGRAM

- Atrial arrhythmias (atrial premature contractions, atrial fibrillation) are common.
- Evidence of left atrial enlargement (widened P waves) and left ventricular enlargement may be present.
- Splintered QRS complexes are common ECG findings in cats and dogs with tricuspid valve dysplasia. Right-sided heart enlargement patterns may also be present.

THORACIC RADIOGRAPHS

- The most prominent abnormality with mitral valve dysplasia is severe left atrial enlargement. Left ventricular enlargement is also present. Pulmonary veins may be distended, and evidence of left-sided CHF may be present.
- Right atrial and ventricular enlargement may be evident if severe tricuspid valve regurgitation is present. Distention of the caudal vena cava may be noted if right-sided CHF is present.

ECHOCARDIOGRAPHY

- Echocardiography demonstrates malformation of the mitral valve complex (fused chordae tendineae and thickened, immobile valve leaflets) and left atrial and ventricular dilation.
- Doppler echocardiography demonstrates severe mitral regurgitation. If present, mitral stenosis can be identified.
- The echocardiographic findings with tricuspid valve dysplasia mirror those changes seen with mitral valve dysplasia and only involve the right side of the heart instead (Fig. 12.15).

THERAPY AND PROGNOSIS

- Surgical valve repair is not commonly performed because of the attendant mortality and expense. Catheter-based balloon valvuloplasty (or a hybrid approach) has been reported in patients with AV valve stenosis.
- Prognosis for severely affected animals with clinical signs is poor. Mildly affected animals may remain free of clinical signs for several years. Therapy for progressive CHF is detailed in Chapter 15.

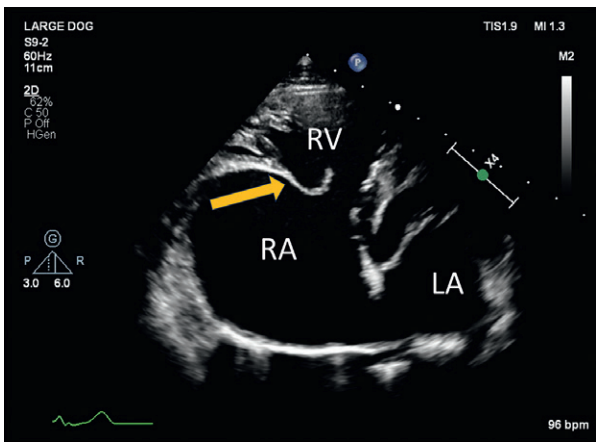


Fig. 12.15 Echocardiogram from a dog with tricuspid valve dysplasia. Note the severely thickened tricuspid valve leaflets (*arrow*). *LA*, Left atrium; *RA*, right atrium; *RV*, right ventricle.

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Frequently Asked Questions

WHICH IS THE BEST APPROACH FOR TREATMENT OF PDA: SURGICAL LIGATION OR CATHETER-BASED DEVICE OCCLUSION?

Both approaches have been demonstrated to be effective and safe when performed by experienced individuals; therefore, the best approach is the approach that is most readily available (there are geographical limitations for device occlusion because there are a limited number of individuals trained to perform catheter-based interventional procedures).

DOES THE INTENSITY OF A CARDIAC MURMUR ASSOCIATED WITH CONGENITAL HEART DISEASE CORRELATE WITH THE SEVERITY OF THE DISEASE?

In general, the intensity of the murmur roughly approximates the severity of the defect; however, there are some exceptions. Patients with small, restrictive VSDs often have very loud systolic murmurs but are unlikely to be clinically affected. Regarding defects that cause obstruction to ventricular outflow, the presence of myocardial failure can decrease the intensity of the murmur. Some patients with tricuspid valve dysplasia have "silent" regurgitation that can only be detected by echocardiography or angiocardiography. Patients with reversed PDA have severe cardiovascular derangement but often have no audible cardiac murmurs.

WHAT IS THE MOST IMPORTANT DIAGNOSTIC TEST FOR A PATIENT SUSPECTED OF HAVING A CONGENITAL HEART DEFECT?

Echocardiography is the most important part of the evaluation of a patient suspected of having a congenital heart defect. 2D echocardiography provides an anatomic image of the cardiac structures that can be observed in real time for assessment of structural and functional abnormalities. Along with 2D echocardiography, M-mode echocardiography can be used to quantify cardiac dimensions and function. Doppler echocardiography is a critical part of the echocardiographic evaluation of a patient with a cardiac murmur. Color-flow Doppler allows for rapid detection of abnormal flow patterns, and spectral Doppler can be used for quantification of blood flow characteristics such as velocity.

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Chapter 13

Cardiovascular Effects of Systemic Diseases

Donald Paul Schrope and Carl D. Sammarco

Many systemic diseases are capable of profoundly affecting cardiovascular structure and function (Box 13.1). The veterinarian may detect cardiovascular abnormalities as the predominant clinical sign, or systemic disease manifestations may overshadow cardiac abnormalities. Although cardiovascular manifestations may sometimes be of no clinical significance, at other times they may constitute the major medical concern. Detection of cardiovascular involvement may be based on clinical signs, radiographic changes, electrocardiographic (ECG) or echocardiographic abnormalities, or laboratory findings. Emphasis in this chapter is given to diseases having the greatest cardiovascular effects or incidence in veterinary practice. The focus of discussion is the cardiovascular effects and their treatment.

ENDOCRINE DISEASES

HYPERTHYROIDISM

- Hyperthyroidism is the most common systemic disturbance to affect cardiac function in cats and is rare in dogs. Hyperthyroid heart disease can be a severe problem, leading to heart failure in some animals.
- Hyperthyroidism is a disease of middle-aged to geriatric cats (range 4–22 years; mean 13 years) that is generally caused by a thyroid adenoma. Clinical evidence of thyrotoxic heart disease is unusual before the age of 6 years.
- There is a declining reported incidence of cardiovascular complications from hyperthyroidism. This change is probably related to earlier diagnosis, as measurements of T_4 concentrations are now a common component of geriatric screening profiles.
- Thyroid tumors in dogs are generally rare and nonfunctional adenocarcinomas that do not cause hyperthyroidism. Iatrogenic hyperthyroidism can result from over supplementation of thyroxin in hypothyroid dogs.

Cardiac Pathophysiology

Direct effects

- Positive inotropic effects (i.e., increased contractility) result from an increased sodium-potassium exchanging adenosine triphosphatase (Na^+, K^+-ATPase) activity, increased mitochondrial protein synthesis, and increased synthesis and enhanced contractile properties of myosin. Thyroid hormone favors the production of the alpha heavy chain myosin isoenzyme, which has the fastest ATPase activity but is less efficient at converting ATP to mechanical energy. Thyroid hormones increase the number of L-type calcium channels and expression of sodium calcium ATPase, which improves the recycling of calcium by the sarcoplasmic reticulum. Both systolic and diastolic functions are altered.
- Positive chronotropic effects (i.e., increased heart rate) result from an increased rate of sinoatrial firing, decreased threshold of atrial activation, and shortened refractory period of the conduction tissue. The effect on atrial activation may increase the risk for atrial arrhythmias.

Indirect effects

- The effect of thyroid hormones on the adrenergic system is controversial. The number of myocardial beta-adrenergic receptors and their affinity are increased in hyperthyroid animals. However, this does not always explain or result in an increased responsiveness to catecholamines. Post-receptor effects of thyroid hormone are probably mediated by changes in intracellular G protein populations that result in an enhanced response to adrenergic agonists. Circulating catecholamine levels are normal.
- Hyperthyroidism results in an increased metabolic rate and, consequently, an increased tissue oxygen demand. The need for increased tissue perfusion and oxygen delivery requires greater cardiac output. Peripheral vasodilation increases tissue perfusion and decreases afterload. As blood volume increases, increased venous return to the heart and increased preload result. The combination of increased preload, increased contractility, increased heart rate, and decreased afterload results in increased cardiac output. The chronic volume overload and the high metabolic rate can result in heart failure even though the cardiac output is still greater than normal. This is referred to as high-output heart failure.
- Hypertension is a potential sequela of hyperthyroidism in cats, with a prevalence in newly diagnosed hyperthyroid cats ranging from 9% to 23% in recent reports. This result occurs despite peripheral

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Box 13.1 Classification of Important Systemic Disorders That Affect the Heart

ENDOCRINE
- Thyroid gland
  - Hyperthyroidism
  - Hypothyroidism
- Adrenal gland
  - Hypoadrenocorticism
  - Hyperadrenocorticism
  - Pheochromocytoma
- Pituitary
  - Acromegaly (hypersomatotropism)
- Pancreas
  - Diabetes mellitus (hyperglycemia)

METABOLIC
- Hyperkalemia
- Hypokalemia
- Hypercalcemia
- Hypocalcemia
- Hypermagnesemia / Hypomagnesemia
- Hypoglycemia
- Uremia
- Anemia

NEOPLASTIC AND INFILTRATIVE HEART DISEASES

PHYSICAL AND CHEMICAL AGENTS
- Hyperpyrexia (heat stroke)
- Hypothermia
- Carbon monoxide
- Toad poisoning
- Oleander toxicity
- Chocolate (theobromine) toxicity
- Doxorubicin cardiotoxicity

INFECTIOUS/INFLAMMATORY MYOCARDIAL DISEASES
- Lyme Disease
- Chaga's Disease (Trypanosmiasis)
- Parvovirus

MISCELLANEOUS
- Systemic lupus erythematosus (SLE)
- Neurologic disease / Neurogenic cardiomyopathy
- Gastric dilatation-volvulus complex (GDV)
- Pancreatitis
- Traumatic myocarditis

vasodilation and probably reflects the increased stroke volume and heart rate that increases cardiac output. Blood pressure is a function of cardiac output and systemic vascular resistance. Approximately 20% of hyperthyroid cats have systemic hypertension after treatment, with median time from treatment to subsequent development of hypertension of 5.3 months. Approximately one-third of these cats had a serum creatinine level greater than 2 mg/dL, and two-thirds had normal kidney function. The mechanism of this posttreatment hypertension is unknown.

- The combination of volume and pressure overload alters myocardial protein synthesis and degradation, which results in myocardial hypertrophy and chamber dilation. These changes are most notable in the left side of the heart. In rare cases, hyperthyroid cats with congestive heart failure have a cardiomyopathy of overload that resembles dilated cardiomyopathy (DCM).

Key Point

The net effects of hyperthyroidism on the cardiovascular system are enhanced cardiac contractility, tachycardia, cardiomegaly, left ventricular hypertrophy, high cardiac output, systemic hypertension, and occasionally high-output heart failure.

Diagnosis

History and physical examination

- Historical findings and clinical signs include weight loss, polyphagia, unkempt hair coat, polydipsia, diarrhea, nervousness, hyperactivity, vomiting, tremor, polyuria, lethargy, aggression, decreased appetite, weakness, episodic panting, and bulky and foul-smelling stool.
- Cardiovascular abnormalities that may be present on physical examination include tachycardia, premature heart beats, a gallop sound, an apical systolic murmur, accentuated heart sounds, forceful precordial beat, bounding femoral pulses, jugular venous distention, and dyspnea when stressed.
- Noncardiac findings can include a low body condition score, a palpable thyroid gland, hyperactivity, dehydration, easily stressed, small kidneys, depression, weakness, and ventral flexion of the neck.

Electrocardiography

- ECG findings in hyperthyroid cats include sinus tachycardia, right bundle branch block, tall R waves in lead II, left anterior fascicular block, ventricular premature complexes (VPCs), atrial premature complexes (APCs), atrial tachycardia, or atrial fibrillation. Ventricular tachycardia, ventricular bigeminy, ventricular preexcitation, and atrioventricular (AV) block have also been described.
- Sinus tachycardia and high R-wave voltage resolve with treatment of hyperthyroidism. APCs and VPCs may decrease in frequency or disappear. Conduction disturbances may or may not resolve. If arrhythmias or R-wave amplitude changes persist after hyperthyroidism is controlled, the patient should be evaluated for idiopathic cardiomyopathy or systemic hypertension.

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Radiography

- Mild-to-severe cardiomegaly may be seen.
- Evidence of congestive heart failure (pulmonary edema or pleural effusion) is reported in fewer than 5% of cases.

Echocardiography

- Common findings with hyperthyroidism include left atrial enlargement, left ventricular free wall and septal hypertrophy, hyperkinetic left ventricular free wall and septum, left ventricular dilation (eccentric hypertrophy), and increased fractional shortening.
- A dilated form of cardiomyopathy occasionally develops and is characterized by left atrial and ventricular dilation and a low fractional shortening.

Clinical pathology

- Elevated serum values for alkaline phosphatase, aspartate aminotransaminase, and alanine aminotransaminase are common.
- Mild azotemia is present in approximately 34% of cases.
- The thyroxine (T_4) value is usually elevated and is the diagnostic screening test of choice; however, thyroid hormone levels fluctuate during the day and may dip into the normal range in mildly hyperthyroid animals. Additional diagnostic tests may occasionally be needed to confirm the diagnosis of hyperthyroidism in cats with normal resting T_4 values on repeated assessment. These include measuring the free T_4 level and performing a triiodothyronine (T_3) suppression test.
- Cardiac biomarkers *N*-terminal pro-B-type natriuretic peptide (NT-ProBNP) and troponin I (cTnI) levels are elevated in approximately one-half of hyperthyroid cats. These tests cannot be used to differentiate cats with hyperthyroidism from cardiomyopathy; however, levels generally return to normal after 3 months of effective therapy for hyperthyroidism.

Specialized diagnostic tests

Blood pressure determination

- Systemic hypertension was present in 9% to 23% of newly diagnosed hyperthyroid cats in recent reports.
- Approximately 20% of hyperthyroid cats have systemic hypertension within 6 months of initiating treatment for hyperthyroidism.

Thyroid imaging

- Thyroid imaging with radioactive technetium-99 m will identify the affected gland(s). This imaging study is recommended before surgery in any hyperthyroid cat with nonpalpable thyroid glands because the thyroid glands can migrate into the chest.

Key Points

Hyperthyroidism should be ruled out in older cats with elevated NT-ProBNP or cTnI levels before ascribing the elevations to cardiomyopathy. Blood pressure should be monitored for up to 6 months after initiation of antithyroid therapy.

Therapy

Management of hyperthyroidism

- Radioactive iodine is curative and does not require anesthesia or surgery. In most instances it is the safest and most effective treatment.
- Surgical treatment effectively cures the condition and eliminates the need for chronic medication. Medical management generally is instituted to stabilize the patient before surgery. The most frequent complication of surgery is transient postoperative hypocalcemia.
- Medical management generally involves administering methimazole orally or as a transdermal formulation in pluronic lecithin organogel. Carbimazole, an alternative to methimazole, is available in Europe. These drugs inhibit the formation of thyroid hormones but do not inhibit or block the release of already formed T_3 and T_4. Thus serum thyroid levels do not drop acutely.
- Inorganic iodine therapy is rarely used. Iodine blocks thyroid hormone synthesis and release, and thyroid levels rapidly drop. Rapid reduction of thyroid levels may benefit patients with severe congestive heart failure. Preoperative use may decrease the vascularity of the thyroid gland. Administer one dose of methimazole before the iodine so that the iodine is not incorporated into the new thyroid hormone.
- Sodium or calcium ipodate blocks the conversion of T_4-T_3. Ipodate is not effective in all cats and should be considered a treatment of last resort.

Management of arrhythmias

- The expedient way to manage tachyarrhythmias is to start methimazole therapy. Even before they become euthyroid, many arrhythmias improve or are abolished. In this fashion, ancillary therapy is usually unnecessary.
- Severe tachyarrhythmias (i.e., atrial tachycardia, multiform VPCs, ventricular tachycardia) in patients without heart failure are best managed with beta-adrenergic blockers such as atenolol (3.125–6.25 mg per cat PO q12h) or propranolol (2.5–7.5 mg per cat PO q8h). Higher-than-normal doses of beta-adrenergic blockers may be needed because of an increased rate of metabolism and elimination in hyperthyroid animals. Supraventricular arrhythmias in cats with asthma should be managed with diltiazem (7.5 mg per cat PO q8h) rather than a beta-blocker.

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- Supraventricular arrhythmias in patients with congestive heart failure can be managed with digoxin, beta-blockers, or diltiazem. Exercise caution when using beta-blockers in the presence of congestive heart failure because these agents depress cardiac contractility and may acutely exacerbate heart failure. Cats with hyperthyroidism almost always have increased contractility, so worsening of congestive heart failure with beta-blockade is rare. Ideally, congestion should be treated and controlled with diuretics before a beta-blocker is prescribed. Diltiazem also depresses contractility but less so than beta-blockers. The vasodilative effects of diltiazem usually offset its negative inotropic effects.

Management of hypertension

- Usually accomplished with control of the hyperthyroid state. The use of beta-blockers (e.g., atenolol), angiotensin-converting enzyme (ACE) inhibitors (e.g., enalapril, benazepril), or amlodipine are occasionally indicated.

Management of congestive heart failure

- Diuretics are used to control pulmonary edema and pleural effusion.
- Enalapril or benazepril also may be helpful in refractory cases.
- Treat arrhythmias as discussed previously.
- Pimobendan may be useful in cases where chronic hyperthyroidism results in DCM.

Prognosis

- The prognosis for remission of sinus tachycardia, voltage changes, arrhythmias, and congestive heart failure is generally favorable when the hypermetabolic state is controlled.
- Systemic hypertension develops in 20% of cats following treatment of hyperthyroidism; therefore blood pressure monitoring is recommended. Renal insufficiency can also be unmasked after control of the disease.
- The prognosis is poor if DCM has developed secondary to chronic overload of the heart (rare).

HYPOTHYROIDISM

- Hypothyroidism is a common endocrinopathy in dogs and is rarely encountered as a naturally occurring condition in cats. It is most commonly diagnosed in middle-aged dogs (22% are 2–3 years old; 32% are 4–6 years old; 22% are 7–9 years old). Many breeds are affected, and there appears to be a breed predilection in the Golden Retriever, Doberman Pinscher, Dachshund, Irish Setter, Miniature Schnauzer, Great Dane, Poodle, and Boxer. There is no gender predilection. Hypothyroidism is rare in cats and generally limited to postoperative cases after surgical removal of unilateral or bilateral functional thyroid tumors.
- The cardiac effects of hypothyroidism are generally the opposite of those found in hyperthyroidism. The cardiac effects of hypothyroidism rarely have severe consequences. However, hypothyroidism can aggravate heart failure and complicate the management of cardiac patients.

CARDIAC PATHOPHYSIOLOGY

- Cardiac contractility is decreased as a result of decreased Na^+, K^+-ATPase activity, decreased mitochondrial protein synthesis, and decreased synthesis of contractile properties of myosin.
- Cardiac conduction is decreased as a result of a decreased rate of sinoatrial firing, increased threshold of atrial activation, and prolonged refractory period of the conducting tissue. Enhanced vagal tone in hypothyroidism also slows the heart rate and accentuates sinus arrhythmias.
- Thyroid hormone plays an important role in lipid and cholesterol metabolism. Hypothyroidism predisposes animals to increased levels of cholesterol and, to a lesser extent, triglycerides. Although rare in dogs, hypercholesterolemia can result in atherosclerosis and myocardial infarction. Lipid abnormalities resolve with thyroid hormone supplementation.
- Hypothyroidism decreases the metabolic rate, thus lessening the required cardiac output. Correcting hypothyroidism increases the workload on the heart; remember this when treating animals in congestive heart failure.
- Hypothyroidism decreases digoxin clearance, predisposing patients to digoxin toxicity.

Key Point

Evaluate the thyroid status in any dog in congestive heart failure, particularly those with inappropriately slow heart rates.

DIAGNOSIS

History and Physical Examination

- Historical findings in hypothyroid animals include alopecia and other dermatologic problems, lethargy, weakness, depression, infertility, and diarrhea.
- Cardiovascular abnormalities noted on physical examination can include muffled heart sounds, weak apex heartbeat, weak pulses, and bradycardia.
- Noncardiac physical findings can include symmetric alopecia, seborrhea, myxedema, pyoderma, hypothermia, depression, and lameness.

Electrocardiography

- ECG changes in patients with hypothyroidism reflect decreased automaticity of pacemaker tissue and depressed conduction. Changes that reflect decreased automaticity may include sinus bradycardia, pronounced sinus arrhythmia, and atrial and ventricular arrhythmias. Changes that reflect

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depressed conduction may include AV block, widened QRS complexes, and inverted T waves. The most common findings reported in dogs are sinus bradycardia and inverted T waves.
- Low-voltage QRS complexes are frequently seen in hypothyroid animals.
- Atrial fibrillation in giant-breed dogs occasionally accompanies hypothyroidism, usually in association with DCM. Supplementation with thyroid hormone rarely causes conversion to sinus rhythm.

Diagnostic Imaging
- Radiograph findings are generally normal.
- Echocardiography may reveal mild to marked decreases in indices of cardiac contractility and mild chamber enlargement. Decreased contractility is documented by an increase in left ventricular internal diameter during systole, prolongation of the pre-ejection period (PEP), a decrease in left posterior wall thickness during systole, and a decrease in septal wall thickness during systole and diastole. There is also often a decrease in aortic root diameter, decrease in velocity of circumferential shortening, and a decrease in fractional shortening.
- Always check the thyroid status when depressed contractility is noted in an animal of a breed not predisposed to DCM.

Clinical Pathology
- The hemogram may reveal a low-grade nonregenerative anemia.
- The most common abnormality on the serum chemistry profile is hypercholesterolemia and occasionally hypertriglyceridemia.
- A normal serum T_4 value generally is sufficient to rule out hypothyroidism. Unfortunately a low serum T_4 does not confirm the diagnosis. Concurrent systemic ailments and numerous drugs can cause depressed serum T_4 values in animals with normal thyroid function (euthyroid sick syndrome) (Box 13.2). Making a diagnosis in these cases requires integration of the history, physical findings, and other blood test results, or confirmation by specialized diagnostic tests.
- Serum T_3 values are less reliable than T_4 values at diagnosing hypothyroidism.

Specialized Diagnostic Tests
- The thyroid-stimulating hormone (TSH) response test has been the "gold standard" in veterinary medicine for diagnosing hypothyroidism. Animals with hypothyroidism have low baseline T_4 values and fail to increase their T_4 values above a predetermined level following administration of TSH. Animals with euthyroid sick syndrome will also have low baseline T_4 levels, but following TSH administration, their T_4 values will increase above an absolute cutoff value. Unfortunately TSH is expensive and often difficult to obtain.
- Free T_4 by equilibrium dialysis (ED) as a diagnostic test of canine hypothyroidism has a sensitivity of 98% and specificity of 92%, compared with a total T_4 sensitivity of 90% and specificity of 90%. The free T_4 by ED should be low in a hypothyroid animal.
- As a diagnostic test for canine hypothyroidism, canine TSH levels have a sensitivity of 75% and specificity of 90%. The TSH level should be high in a hypothyroid animal.
- When the endogenous TSH is high and the free T_4 by ED is low, the diagnostic accuracy approaches 100%.

Box 13.2 Some Drugs and Diagnostic Agents That Can Alter Basal Serum Thyroid Hormone Concentrations in Humans and Possibly Dogs

DECREASE T_4 AND/OR T_3	INCREASE T_4 AND/OR T_3
Amiodarone (T_3)	Amiodarone (T_4)
Androgens	Estrogens
Cholecystographic agents	5-Fluorouracil
Diazepam	Halothane
Dopamine	Insulin
Flunixin	Narcotic analgesics
Furosemide	Radiopaque dyes (ipodate)
Glucocorticoids	Thiazides
Heparin	
Imidazole	
Iodide	
Methimazole	
Mitotane	
Nitroprusside	
Penicillin	
Phenobarbital	
Phenothiazines	
Phenylbutazone	
Phenytoin	
Primidone	
Propranolol	
Propylthiouracil	
Radiopaque dyes (ipodate) (T_3)	
Salicylates	
Sulfonamides (sulfamethoxazole)	
Sulfonylureas	

From Feldman EC, Nelson RW: *Canine and feline endocrinology and reproduction*, ed 3, St. Louis, 2004, WB Saunders.

THERAPY
- Treatment of hypothyroidism with L-thyroxine is simple and inexpensive.
- Treatment in patients with congestive heart failure is more complicated. Thyroid supplementation will

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increase the metabolic rate and demand on the heart. If the failing heart is unable to meet these demands, heart failure will worsen. Therefore implement thyroid hormone supplementation gradually in patients with cardiac disease, especially if they are already in failure. Start with one-fourth of the standard dose and increase it by one-fourth dose weekly. It should be emphasized that hypothyroidism rarely causes heart failure but may significantly worsen cardiac function in an animal with underlying heart disease.

- The risk of digoxin toxicity is increased in hypothyroid states. Monitor hypothyroid animals closely for signs of digoxin toxicity and consider assessing thyroid status in any animal with normal renal function that has digoxin toxicity while receiving reasonable doses of digoxin. Ideally the digoxin level on a sample obtained 8 hours after a dose should be between 0.5 and 1 ng/mL.

PROGNOSIS

- There are few complications in the treatment of hypothyroidism.
  - Impaired myocardial function improves following thyroid supplementation.
  - Resolution of atrial fibrillation has been reported in one dog after correction of hypothyroidism. This response is rare.

HYPOADRENOCORTICISM (ADDISON DISEASE)

- Hypoadrenocorticism is a potentially life-threatening endocrinopathy that is uncommon in dogs and rare in cats. The disease is more common in female dogs and usually occurs in young to middle-aged animals. Approximately 76% of cases occur before 7 years of age with an age range of 5 weeks to 16 years.
- Primary hypoadrenocorticism (Addison disease) results from destruction of the zona glomerulosa and zona fasciculata of the adrenal gland. These areas of the adrenal gland produce aldosterone and cortisol, respectively. Destruction is generally immune mediated but also can occur secondary to infection, neoplasia, infarction, and drugs such as o,p′-dichlorodiphenyldichloroethane (o,p′-DDD, mitotane).
- Secondary hypoadrenocorticism results from decreased production or release of adrenocorticotropic hormone (ACTH) from the pituitary gland. Secondary adrenal insufficiency may occur following acute withdrawal of long-term, high-dose glucocorticoid therapy that has suppressed the hypothalamic-pituitary-adrenal axis. These patients have normal mineralocorticoid activity, so electrolyte values are normal. Cortisol values are depressed.
- Adrenal destruction occurs gradually; therefore basal hormone levels remain normal. It is only when the animal is stressed, and the adrenal reserve is inadequate that signs develop. Eventually the destruction proceeds to the point that basal hormone levels are depressed, and clinical signs are present without stress.

CARDIAC PATHOPHYSIOLOGY

Cardiovascular Effects of Cortisol

- Cortisol is important in maintaining vascular integrity and responsiveness to catecholamines. Cortisol deficiency predisposes animals to hypotension.
- Cortisol has a positive inotropic effect on the heart.
- Cortisol affects the distribution and elimination of body water.
- Cortisol is important in helping the body deal with stress. This is why so many Addisonian crises follow stress and why glucocorticoid supplementation must be increased during periods of stress.

Cardiovascular Effects of Aldosterone

- Aldosterone acts on the distal renal convoluted tubule and collecting duct to enhance sodium retention and potassium elimination. Aldosterone deficiency results in hyponatremia and hyperkalemia.
- Hyponatremia decreases plasma osmotic pressure, causing fluid shifts out of the vascular compartment. This contributes to hypovolemia and hypotension.
- Hyperkalemia alters cardiac conduction resulting in numerous arrhythmias. The cardiac effects of hyperkalemia are aggravated by low sodium, low calcium, and acidosis.

Key Point

Net cardiovascular effects of hypoadrenocorticism are hypovolemia, systemic hypotension, altered cardiac conduction, and depressed myocardial function. These changes can be fatal if not rapidly diagnosed and aggressively treated.

DIAGNOSIS

History and Physical Examination

- A history of waxing and waning of clinical signs, with more severe signs during periods of stress, generally is present.
- Patients often have a history of anorexia, vomiting, diarrhea, depression, weakness, and cardiovascular collapse. Other signs include weight loss, polyuria, polydipsia, melena, and shivering.
- Transient improvement after intravenous (IV) fluid therapy or corticosteroid administration followed by subsequent relapse days or weeks later can occur.
- Findings on physical examination include depression, weakness, dehydration, hypothermia, slow

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capillary refill time, shaking, bradycardia, and weak femoral pulses.

Electrocardiography

- ECG changes vary with the magnitude of the hyperkalemia and are aggravated by hyponatremia, hypocalcemia, and acidosis. The following ECG findings are seen with experimentally induced hyperkalemia (Fig. 13.1):
  - Peaking of the T wave with a narrow base is the earliest ECG abnormality and may be observed when serum K^+ exceeds 5.7 to 6.0 mEq/L.
  - P-wave shape is altered, its amplitude reduced, intraatrial conduction delayed, and PR interval prolonged when serum K^+ exceeds 7.0 mEq/L.
  - P waves become unrecognizable (atrial standstill) at plasma K^+ levels greater than 8.5 mEq/L.
  - The QRS widens uniformly at plasma K^+ levels greater than 9 mEq/L.
  - Sinoatrial exit block or junctional rhythms with and without escape complexes may be present.
  - Ventricular fibrillation and ventricular asystole may occur at plasma K^+ levels greater than 10 mEq/L.
  - In clinical cases, ECG changes do not correlate as closely with potassium concentrations as they do in published experimental studies.
  - The rhythm with atrial standstill secondary to hyperkalemia is generally slow and may be regular or irregular. It is a sinoventricular rhythm. The sinus node continues to fire, and impulses are transmitted to the AV node and ventricles by internodal pathways. P waves are absent because atrial myocytes are not activated.

Key Point

Hyperkalemia is the most common cause of atrial standstill. Atrial standstill also occurs with fibrous replacement of atrial muscle cells secondary to severe atrial distention (valve disease, cardiomyopathy) in dogs and cats, with cardiac arrest, and secondary to an atrial myopathy in dogs, a condition most commonly seen in English Springer Spaniels.

Radiographs

- Approximately 80% of untreated dogs have one or more radiographic abnormalities. Abnormalities in order of reported frequency include narrow caudal vena cava, microcardia, narrow cranial lobar pulmonary artery, and microhepatica. These changes are the result of hypovolemia and rapidly resolve with steroid and volume replacement.

Clinical Pathology

- A low-grade normocytic, normochromic nonregenerative anemia is present in 25% of cases but may be masked by the effects of hemoconcentration. Eosinophilia and lymphocytosis may also be present.
- A serum chemistry profile frequently reveals azotemia, hyponatremia, and hyperkalemia. A sodium/potassium ratio of less than 27 supports hypoadrenocorticism but is not pathognomonic for the disease. Hypercalcemia and hypoglycemia can also be present.
- Mild-to-moderate metabolic acidosis is common during a crisis.
- It is unusual for a dog with untreated Addison disease to have a urine specific gravity greater than 1.030, and it is often less than 1.020.
- In patients with secondary hypoadrenocorticism, sodium and potassium levels are normal. The other abnormalities described above may be present.

Specialized Diagnostic Tests

- A tentative diagnosis is frequently obtained from the history and results of the complete blood count and serum chemistry profile. However, there are numerous causes of hyperkalemia, and acid-base and electrolyte changes associated with severe gastrointestinal disease and renal failure can cause an abnormal sodium/potassium ratio that would erroneously suggest Addison disease.
- An ACTH response test is helpful in confirming Addison disease and essential for documenting secondary hypoadrenocorticism.

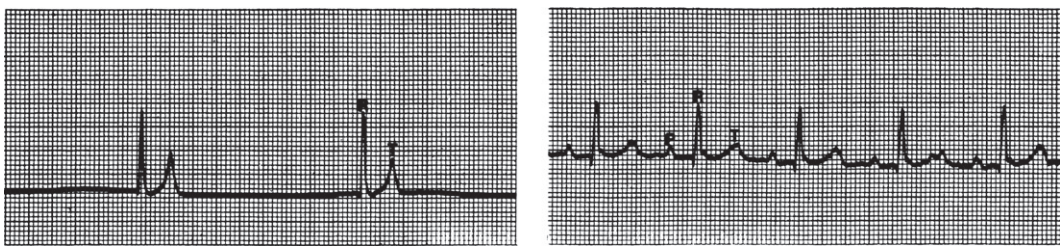


Fig. 13.1 **(A)** Hyperkalemia in a dog in cardiovascular collapse, consistent with an Addisonian crisis. P waves are absent (atrial standstill), and T waves are tall and peaked. Serum potassium was 8.4 mEq/L. **(B)** After institution of therapy, P waves are present, and the T wave is of smaller amplitude. Serum potassium is now 4.8 mEq/L. (From Tilley LP: *Essentials of canine and feline electrocardiography: interpretation and treatment*, ed 3, Malvern, 1992, Lea & Febiger.)

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THERAPY

Addisonian Crisis Therapy

- Patients that have cardiovascular collapse and atrial standstill require aggressive therapy. Therapy is directed at rapidly correcting hypovolemia, correcting rhythm disturbances, correcting electrolyte imbalances, and replacing mineralocorticoids and glucocorticoids.
- Volume replacement, and correction of hyponatremia and hypochloremia, is initially achieved with rapid infusion of 0.9% saline at 60 to 80 mL/kg/h for 1 to 2 hours and then at a modified rate according to patient needs. The ACTH response test can be performed while saline is being administered.
- Glucocorticoid deficiency is corrected with either methylprednisolone sodium succinate (1–2 mg/kg IV) or dexamethasone sodium phosphate (2–4 mg/kg IV) and then repeated every 2 to 6 hours. After the initial high dose of a rapidly acting, water-soluble glucocorticoid has been administered, glucocorticoid therapy can be continued with dexamethasone at a dosage of 0.05 to 0.1 mg/kg q12h added to the IV fluids. Once the patient is stable, switch to oral prednisone. It should be noted that prednisolone interferes with cortisol assays, so ideally it should be started after the ACTH response test is performed. Dexamethasone has no mineralocorticoid effects and does not interfere with the cortisol assay.
- Mineralocorticoid deficiency is initially corrected (after the ACTH response test is performed) with desoxycorticosterone pivalate (DOCP) at 2.2 mg/kg IM or fludrocortisone acetate at 0.02 mg/kg/day PO if the dog is not vomiting.
  - Hyperkalemia is corrected in several different ways:
  - Saline infusion results in rapid dilution of potassium and is often the only treatment needed.
  - Severe hyperkalemia associated with significant cardiac conduction abnormalities often requires more aggressive therapy. Treatment options include sodium bicarbonate (1–2 mEq/kg) given slowly IV over 5 minutes *or* a combination of regular insulin (0.5 U/kg IV) followed by dextrose added to fluids (1 g per unit insulin given). Dextrose can also be administered alone as it will stimulate endogenous insulin secretion.
  - If life-threatening cardiac arrhythmias are noted, administer calcium gluconate (10%) at 0.5 to 1.5 mL/kg IV slowly over 10 to 15 minutes. Monitor the ECG while administering calcium. Calcium will temporarily correct rhythm disturbances by countering the effect of potassium on the conduction tissue. Calcium has no effect on serum potassium levels. Calcium overdose can also cause severe cardiac disturbances; therefore careful monitoring is required.

Maintenance Therapy

- Mineralocorticoid replacement is initiated with either daily oral administration of fludrocortisone acetate or intramuscular injections of DOCP every 25 days. Doses—and dose interval for DOCP—are adjusted as needed to maintain normal electrolyte balance. Therapy must be individualized to the patient.
- Glucocorticoid replacement is achieved with either prednisone or cortisone. Some patients receiving fludrocortisone acetate or DOCP do not need glucocorticoid supplementation except during periods of stress.

Prognosis

- The prognosis is excellent with therapy.
- Many dogs appear to do better with a small daily maintenance dose of prednisone or cortisone. During illness or other stressful periods, however, larger doses of glucocorticoids are necessary to avoid relapse into acute adrenal crisis.

HYPERADRENOCORTICISM (CUSHING DISEASE)

- Hyperadrenocorticism is a common endocrinopathy in older dogs and is rarely reported in cats. The syndrome is associated with excessive levels of cortisol that result from excess pituitary ACTH, or excess ACTH from ectopic nonendocrine tumors, functional adrenocortical adenoma, or carcinoma. Iatrogenic Cushing syndrome occurs with excessive glucocorticoid administration.
- Pituitary-dependent hyperadrenocorticism accounts for 80% to 85% of cases of hyperadrenocorticism. It is a disease of middle-aged and older dogs (usually older than 6 years), although a few cases have been diagnosed in dogs younger than 1 year. There is no sex predilection. Poodles, Dachshunds, and Beagles may be at increased risk.
- Dogs with functional adrenocortical tumors tend to be older, ranging from 6 to 16 years of age with a mean age of 11 years. Females are affected more frequently. German Shepherds, Toy Poodles, Dachshunds, and Terriers are most commonly affected.
- Hyperadrenocorticism rarely produces significant cardiac disease; however signs and side effects of hyperadrenocorticism can mimic cardiac disease. Systemic effects of hyperadrenocorticism can also exacerbate the underlying cardiac disease.

PATHOPHYSIOLOGY

Systemic Hypertension

- Hypercortisolism increases vascular resistance by increasing smooth muscle sensitivity to catecholamines and increasing production of angiotensinogen. Mineralocorticoid properties of cortisol

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enhance the renal resorption of sodium, and secondary fluid retention results in an increased vascular volume. Hypertension is present in 57% to 82% of dogs with Cushing disease.
- Systemic hypertension may cause myocardial hypertrophy.
- Many affected dogs have coexisting AV valvular insufficiency and its associated cardiac changes. Hypertension might exacerbate underlying cardiac problems.

Pulmonary Thromboembolism
- Patients with hyperadrenocorticism are predisposed to pulmonary thromboembolism, with most cases diagnosed while the animal is being treated for Cushing disease. Factors causing thromboembolism with Cushing disease include obesity, high hematocrit, hypertension, and increased levels of clotting factors.
- Pulmonary thromboembolism should be suspected in any Cushingoid patient with a history of an acute onset of dyspnea or cyanosis.

Panting and Dyspnea
- Altered ventilation mechanics are often present owing to weakness in the muscles of respiration, increased thoracic fat deposition (decreasing chest wall compliance), and increased diaphragmatic abdominal pressure resulting from adipose tissue and hepatomegaly. Mild respiratory distress, or rapid respiratory rate at rest, often results.
- Many Cushingoid dogs have variable degrees of lower airway disease or pulmonary parenchymal disease.
- The triad of mitral/tricuspid insufficiency, respiratory disease, and Cushing syndrome may create intractable dyspnea caused by cardiopulmonary failure.

DIAGNOSIS

History and Physical Examination
- The major historical features are systemic manifestations of hypercortisolism; they include polydipsia, polyuria, polyphagia, panting, alopecia, anestrus, exercise intolerance, and lethargy.
- Findings on physical examination may include abdominal enlargement, hepatomegaly, muscle weakness, testicular atrophy, symmetric alopecia, skin atrophy, hyperpigmentation, calcinosis cutis, bruising, and obesity.

Electrocardiography
- ECGs show no characteristic changes. ECG evidence of left ventricular enlargement is often present in Cushingoid dogs with mitral valvular insufficiency.

Diagnostic Imaging
- Hypercortisolism causes changes on thoracic radiographs that include calcification of the tracheal and bronchial rings and osteoporosis of the thoracic vertebrae. Metastatic pulmonary lesions are seen infrequently with adrenal tumors.
- Radiographic changes associated with pulmonary thromboembolism include hypoperfusion of the infarcted lung lobes, overcirculation within the normal lung lobes, pleural effusion, and blunting and thickening of the pulmonary arteries. Thoracic radiographs may have normal findings.
- Echocardiography is rarely part of the standard workup of Cushing disease. Cushing disease though has been associated with the development of left ventricular hypertrophy in dogs independent of systolic blood pressure.

Clinical Pathology
- Excessive production of cortisol may result in neutrophilia, lymphopenia, eosinopenia, thrombocytosis and mild erythrocytosis (females).
- Chemistry abnormalities include fasting hyperglycemia, high serum alkaline phosphatase (sometimes extremely high), high alanine aminotransferase, hypercholesterolemia, lipemia, and low blood urea nitrogen.
- Urinalysis shows specific gravity less than 1.015 and often less than 1.008. Proteinuria, glycosuria, and bacterial cystitis are sometimes noted.

Specialized Diagnostic Tests
- A urine cortisol/creatinine ratio is a quick screening test for Cushing disease. This test has a high sensitivity but low specificity for Cushing disease.
- The low-dose dexamethasone suppression test (LDDST) is the screening test of choice unless iatrogenic hyperadrenocorticism is suspected.
- The ACTH response test is the screening test of choice if iatrogenic hyperadrenocorticism is suspected.
- If there is no suppression on a LDDST, measurement of canine ACTH (cACTH) or abdominal ultrasound is recommended to differentiate between pituitary-dependent Cushing disease and an adrenal tumor.
- A high-dose dexamethasone suppression test can be considered if there is no suppression on the LDDST, and cACTH and abdominal ultrasound are not options.
- Abdominal ultrasound, computed tomography (CT) scan, and magnetic resonance imaging (MRI) can be used to try to differentiate pituitary-dependent Cushing disease from an adrenal tumor and to better characterize the extent of the disease.

THERAPY

Pituitary-Dependent Hyperadrenocorticism
- Mitotane and trilostane are the two main drugs used for the treatment of pituitary-dependent hyperadrenocorticism.

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- Mitotane selectively, and usually reversibly, destroys the zona fasciculata (cortisol) and zona reticularis (sex hormones) of the adrenal gland. The zona glomerulosa (aldosterone) is occasionally affected.
- Trilostane inhibits adrenal steroidogenesis through enzyme inhibition and may affect aldosterone and cortisol synthesis.
- Ketoconazole can be prescribed in animals that do not tolerate mitotane or trilostane. Ketoconazole blocks an enzymatic step that is necessary to produce cortisol.
- Hypophysectomy is rarely performed in the United States.
- Radiation therapy can be used to treat pituitary macroadenomas.

Adrenocortical Tumor

- Surgical removal of the affected adrenal gland(s) is recommended if possible.
- If the tumor is inoperable or metastasis is identified, mitotane is the medical therapy of choice as it has a chemotherapeutic effect. Very high doses of mitotane are usually required in patients with adrenal tumors. Trilostane can be used on dogs that do not tolerate mitotane. Many dogs with adrenal tumors respond poorly to medical management.
- Replacement steroid therapy is necessary both during and after surgery.
- Management of pulmonary embolism and systemic hypertension, if present, is undertaken through standard therapeutic protocols (see Chapters 10 and 15).

PROGNOSIS

- Excellent prognosis is confirmed with resectable, benign adrenal tumors.
- Nonresectable or metastatic adrenal adenocarcinomas have a poor prognosis.
- Therapy with mitotane or trilostane for pituitary-dependent hyperadrenocorticism has potential side effects, but most treated dogs respond well to therapy. The average survival is 2 years, with dogs living longer than 6 months usually dying of causes unrelated to hyperadrenocorticism.
- Systemic hypertension generally resolves with control of hypercortisolism.

PHEOCHROMOCYTOMA

- Pheochromocytomas are catecholamine-producing tumors derived from chromaffin cells.
- These tumors of adrenal medullary origin are uncommonly detected in dogs. They are typically seen in old dogs (mean age, 11 years, with a range of 1–16 years) and extremely rarely in cats.
- In addition to important effects on the cardiovascular system, catecholamines have significant metabolic effects, stimulating glycogenolysis and gluconeogenesis.

CARDIAC PATHOPHYSIOLOGY

- The cardiovascular effects of pheochromocytomas result from the alpha-1, beta-1, and beta-2 effects of norepinephrine and epinephrine. Stimulation of alpha- and beta-adrenergic receptors generally causes opposite effects. The dominant effect varies with relative receptor density and activation thresholds. For example, in vascular smooth muscle, alpha-adrenergic effects predominate. Thus hypertension results when both alpha- and beta-adrenergic receptors are stimulated.
- Beta-1 adrenergic effects include sinus tachycardia, increased cardiac conduction velocity, and increased contractility.
- Beta-2 adrenergic effects on the cardiovascular system include venous and arteriole vasodilation.
- Alpha-1 adrenergic effects on the cardiovascular system include venous and arteriole vasoconstriction. Approximately 50% of dogs with pheochromocytomas are hypertensive at the time of testing.
- Myocardial injury and coronary vasoconstriction may result from catecholamine excess. Multifocal cardiomyocyte necrosis with contraction bands, interstitial fibrosis, cardiomyocyte degeneration, myocardial hemorrhage, and lymphohistiocytic myocarditis have been reported in dogs. Cardiac lesions were detected in approximately 15% of the cases examined.

DIAGNOSIS

History and Physical Examination

- Historical observations include weakness, collapse, anorexia, vomiting, weight loss, panting, dyspnea, lethargy, diarrhea, whining, pacing, polyuria, polydipsia, shivering, and epistaxis.
- Abnormalities noted on physical examination include lethargy, tachypnea and dyspnea, arrhythmias, systolic murmur, pulmonary crackles, tachycardia, weak pulse, pale mucous membranes, and muscle wasting.
- Other physical examination findings are emaciation, peripheral edema, ascites, and abdominal mass.
- Many symptoms and physical findings are reported to be episodic because of the episodic release of catecholamines by the tumor.

Electrocardiography

- Nonspecific ST-segment and T-wave changes may be noted.
- Arrhythmias can occur, especially VPCs and paroxysmal ventricular tachycardia. Complete AV block has been reported in several dogs.
- Consider 24-hour ambulatory ECG monitoring (Holter monitoring) to detect intermittent arrhythmias associated with episodic catecholamine release.

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Radiography
- Generalized cardiomegaly and pulmonary edema can develop, probably owing to sustained chronic hypertension.
- Adrenal tumors can be identified in one-third of the cases.

Echocardiography
- Ventricular hypertrophy is usually present, most commonly the left ventricle. Both concentric and eccentric hypertrophies have been reported.

Clinical Pathology
- No consistent abnormalities are present on the hemogram or chemistry profile.
- Plasma catecholamines and demonstration of elevated 24-hour urinary excretion of vanillylmandelic acid, total metanephrines, and fractionated catecholamines support the diagnosis.

Special Diagnostic Tests
- Abdominal ultrasound may be helpful as a localizing procedure. The presence of an adrenal mass on ultrasound with no evidence of Cushing disease is highly suggestive.
- Angiography can be used in some situations to evaluate adrenal masses invading the caudal vena cava.
- CT, nuclear imaging, and provocative testing to induce hypertension (with histamine, tyramine, and glucagon) or hypotension (with phentolamine) may be useful but rarely are practical or available in general practice.
- Blood pressure determination reveals systemic hypertension in approximately 50% of cases. As catecholamine release is often episodic, the patient should be rechecked several times if the blood pressure is initially normal.

THERAPY
- Alpha- and beta-adrenergic blocking drugs may help control hypertension and arrhythmias, respectively. Always start with an alpha-adrenergic blocker such as phenoxybenzamine (0.2–1.5 mg/kg PO q12h). If a hypertensive crisis is identified, administer phentolamine 0.02 to 0.1 mg/kg IV, followed by IV administration (0.02–0.1 mg/kg IV prn). If tachyarrhythmias remain a problem, then add a beta-blocker such as propranolol (0.2–1 mg/kg PO q8h) or atenolol (0.25–1 mg/kg PO q12 to 24 h). The inclusion of a beta-blocker without alpha blockade can result in severe hypertension.
- Surgical tumor removal is the only definitive treatment. This should be attempted after medical stabilization in patients without metastasis.

PROGNOSIS
- Dogs with inoperable lesions have a poor prognosis. Long-term alpha- and beta-adrenergic blockers may be used in these instances.

HYPERSOMATOTROPISM (ACROMEGALY) IN CATS
- Acromegaly is a syndrome associated with high levels of growth hormone (hypersomatotropism).
- In cats, acromegaly occurs secondary to a pituitary tumor. It is an uncommon endocrinopathy that is seen in primarily male, middle-aged to old cats (mean age 10 years; median age 9 years; range 4–17 years).
- In dogs, hypersomatotropism is associated with progestogen treatment or endogenous progestogens that are produced during diestrus. It does not cause clinically significant cardiac problems in dogs. The syndrome is seen in females and is reversible with discontinuation of progestogen therapy or resolves spontaneously with the end of diestrus.

CARDIOVASCULAR PATHOPHYSIOLOGY
- The trophic effect of growth hormone results in generalized organomegaly, including the heart. Cardiac changes are those of myocardial hypertrophy, interstitial fibrosis, myocytolysis, and intramural arteriosclerosis. Heart failure is a common sequela of acromegaly in cats.
- Systemic hypertension is seen frequently in humans with acromegaly. Systemic hypertension is sometimes present in cats with acromegaly and is usually accompanied by evidence of renal insufficiency.

DIAGNOSIS
History and Physical Examination
- Most cats have a history of polyuria, polydipsia, upper respiratory stridor, and weight gain.
- Cardiac abnormalities noted on physical examination may include a systolic murmur, gallop sound, and, less commonly, signs of congestive heart failure (dyspnea, cyanosis, muffled heart sounds, or crackles).
- Other abnormalities include hepatomegaly, nephromegaly, large head, arthritis, prognathism, pot belly, large tongue, and circling.

Cardiac Tests
- ECG abnormalities have not been observed in these patients.
- Radiographs of the thorax usually demonstrate cardiomegaly, and less commonly, pulmonary edema or pleural effusion.
- The echocardiogram generally reveals hypertrophy of the intraventricular septum and left ventricular free wall.

Clinical Pathology
- The hemogram is generally unremarkable, but some cats have erythrocytosis.
- All cases have hyperglycemia and glucosuria. Most cats also have hyperproteinemia, azotemia,

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and hyperphosphatemia. Hypercholesterolemia, high alanine aminotransferase, high serum alkaline phosphatase, and ketonuria are seen less frequently.

Specialized Diagnostic Tests

- A commercially available validated growth hormone assay is not currently available for cats.
- Insulin-like growth factor I (IGF-1) levels are elevated in most cases. One large study involving cats with high IGF-1 concentrations revealed that 90% of them had documented pituitary tumors evident on CT scans. This test is commercially available.
- CT can be used to identify the pituitary tumor, and in advanced cases, bony and soft tissue changes.
- Procollagen propeptide concentrations in serum are elevated in cats with hypersomatotropism.
- Blood pressure should be evaluated in any suspected or confirmed cases.

THERAPY

- Treatment options include hypophysectomy, radiation therapy, and medical management.
- Hypophysectomy has been performed successfully and resulted in remission of concurrent diabetes mellitus in 80% of treated cats. Perioperative and postoperative mortality is reported at 16%.
- Radiation therapy is difficult to obtain, and the effect is unpredictable.
- Medical management with common somatostatin analogs (e.g., octreotide) and dopamine agonists (e.g., bromocriptine) is generally unsuccessful. Pasireotide (a high-affinity analogue of multiple somatostatin receptor subtypes) has been shown to successfully decrease growth hormone and IGF-1 levels in cats but is expensive and requires twice daily injections.
- Supportive medical care includes high doses of insulin for insulin-resistant diabetes mellitus and diuretics for congestive heart failure.

PROGNOSIS

- Survival in cats with acromegaly ranges from 4 to 60 months (generally 1.5–3 years) from time of diagnosis.
- Most cats die or are euthanized owing to severe congestive heart failure, renal failure, or expanding pituitary tumor.

Note: Hypersomatotropism without acromegaly has been associated with hypertrophic cardiomyopathy in some cats. A total of 31 cats with hypertrophic cardiomyopathy were shown to have growth hormone levels that were four times the control levels. None of the cats that were examined postmortem had pituitary tumors, and none demonstrated hyperinsulinism or diabetes mellitus.

DIABETES MELLITUS

- Diabetes mellitus causes devastating cardiovascular problems in humans. Recent studies reported hypertension (defined as a pressure >160 mm Hg systolic, 100 mm Hg diastolic, and 120 mm Hg mean) in 46% of 50 dogs with diabetes. Subclinical reduction of myocardial contractility has also been reported. A retrospective case-controlled study revealed a significantly greater risk (10.4 times greater) for the development of heart failure in cats with diabetes than in normal controls.
- Hypersomatotropism (acromegaly) has been reported in cats with diabetes (prevalence estimated at 18%–32%) and could contribute to cardiovascular complications in this species.

CARDIAC PATHOPHYSIOLOGY

- Systemic hypertension in dogs with diabetes is associated with the duration of disease and an increased urine albumin to creatinine ratio. The severity of mean and diastolic hypertension is correlated with the duration of disease. In humans, the severity of hypertension correlates with the degree of glycemic control. This association has not been reported in dogs. Hypertension is rarely reported in cats with diabetes. The cause of hypertension is not known but may relate to changes in vascular compliance secondary to changes in lipid profile, generalized glomerular hyperfiltration, and immune-mediated microangiopathy affecting the basement membrane.
- Cardiac autonomic neuropathy has been observed in poorly regulated dogs with diabetes; suppression of sympathetic activity was demonstrated by decreases in both plasma norepinephrine concentrations and heart rate variability (LF component).
- Diabetic cardiomyopathy occurs in humans and is associated with myocardial hypertrophy, fibrosis, microvascular disease, and glycoprotein accumulation. Myocardial changes result in diastolic and systolic dysfunction. In the absence of coronary microvascular disease and systemic hypertension, diabetic cardiomyopathy in humans is a mild condition. In alloxan-induced diabetes in dogs, mild reductions in fractional shortening and left ventricular ejection fraction have been reported along with increased aortic stiffness and increased collagen type 1 and type 3 protein content. Diastolic changes have been mild and inconsistent. Functional coronary artery hyperemia is attenuated, and the balance between coronary blood flow and myocardial metabolism is impaired in alloxan-induced diabetic dogs.

Key Point

Decreased cardiac performance in dogs with diabetes mellitus is mild and unlikely to cause clinical problems unless accompanied by other forms of heart disease, such as DCM.

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DIAGNOSIS

History and Physical Examination

- No cardiovascular signs associated with diabetes.
- Polyuria, polydipsia, polyphagia with weight loss, cataracts (dogs)

Electrocardiography

- No changes related to diabetes.

Radiography

- No changes related to diabetes.

Clinical Pathology

- Fasting hyperglycemia, hypercholesterolemia, hypertriglyceridemia, increased alanine aminotransferase activity, and increased alkaline phosphatase activity.
- Glucosuria, variable ketonuria; sometimes proteinuria, microalbuminuria, and bacteriuria.
- High fructosamine and glycosylated hemoglobin.

Special Diagnostic Tests

- Evaluation of systemic blood pressure may reveal hypertension.
- Echocardiography may demonstrate mild reduction in fractional shortening, increase in PEP, decrease in left ventricular ejection time (LVET), and increase in PEP/LVET.

THERAPY

- Treat hypertension with an ACE inhibitor (e.g., enalapril or benazepril), amlodipine, or a beta-blocker (see Chapter 15).
- Manage diabetes with insulin. Optimizing glycemic control should reduce the risk of secondary cardiovascular complications.

PROGNOSIS

- Cardiac complications with diabetes mellitus in dogs and cats are generally mild and easy to control.

METABOLIC DISTURBANCES

DISORDERS ASSOCIATED WITH HYPERKALEMIA

- More than 95% of body potassium is intracellular, with only 2% to 5% extracellular. Therefore serum potassium values are not always reliable indicators of total body potassium stores.
- Clinical causes of hyperkalemia include the following:
  - Excessive administration of oral potassium supplements or potassium supplemented fluids.
  - Decreased renal elimination associated with oliguric or anuric renal failure.
  - Hypoadrenocorticism (Addison disease).
  - Drugs (potassium-sparing diuretics, ACE inhibitors, beta-blockers).
  - Ruptured urinary tract and urethral obstruction.
  - Translocation from intracellular to extracellular space (metabolic acidosis, rapid release from tissue during severe injury such as aortic thromboembolism).
  - Pleural effusion (possibly due to aldosterone antagonism).

Diagnosis, Therapy, and Prognosis

- ECG changes caused by hyperkalemia are dependent on magnitude and rate of development of hyperkalemia.

Key Point

Most patients with clinically significant hyperkalemia will have bradycardia, but, uncommonly, tachycardia can also develop (Fig. 13.2).

- Identification and treatment of the underlying cause of hyperkalemia is essential (see the section on Hypoadrenocorticism [Addison disease]).

DISORDERS ASSOCIATED WITH HYPOKALEMIA

- When severe potassium loss has occurred, severe muscle weakness or paralysis may develop.
- Clinical causes of hypokalemia include the following:
  - Decreased intake (anorexia, especially when combined with potassium-deficient fluid administration)
  - Excessive gastrointestinal loss
    - Chronic vomiting
    - Severe diarrhea
  - Overuse of enemas, laxatives, or exchange resins
  - Excessive urinary loss
    - Renal (renal tubular acidosis, postobstructive diuresis, chronic pyelonephritis)
    - Drugs (diuretics, amphotericin B, beta-agonists, theophylline)
  - Secondary hyperaldosteronism (liver failure, congestive heart failure, nephrotic syndrome)
  - Extracellular to intracellular transfer
    - Metabolic alkalosis
    - Insulin and glucose administration
    - Hyperinsulinism (insulinoma)
    - Ketoacidotic diabetes mellitus

CARDIAC PATHOPHYSIOLOGY

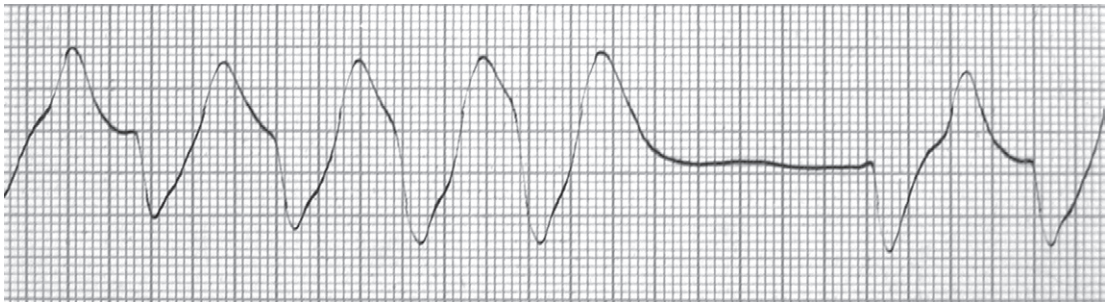
- Severe hypokalemia causes hyperpolarization of muscle fiber membranes and slows repolarization.
- ECG changes are manifested in delayed and abnormal repolarization, increased automaticity, and increased duration of the action potential.

DIAGNOSIS

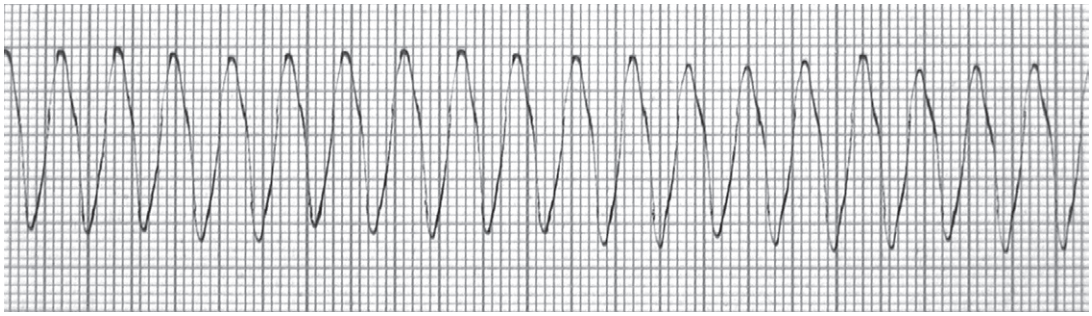
History and Physical Examination

- Muscle weakness may be mild or present as profound weakness and depression.

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A



B

Fig. 13.2 **(A)** Hyperkalemia in cat with urinary obstruction resulting in a wide-complex bradycardia. **(B)** Same cat had wide-complex tachycardia as treatment with insulin and dextrose was initiated. Normal sinus rhythm developed as treatment was continued (not shown).

- Ventroflexion of the head is frequently seen in cats with symptoms.
- Polyuria and polydipsia may occur.

Electrocardiography

- QT interval prolongation may occur.
- U waves may develop (usually a positive deflection immediately after the T wave).
- ST segment may become depressed (Fig. 13.3) with a gradual blending of T waves and what appears to be a tall U wave.
- Ventricular and supraventricular arrhythmias may occur.
- Clinical pathology is generally reflective of the underlying disease process contributing to hypokalemia. Urine concentrating ability may be impaired.

THERAPY

- Treat the primary disease process.
- Parenteral replacement with potassium chloride is recommended when potassium loss is severe, and the animal is symptomatic for hypokalemia (Table 13.1).

Key Point

When administering potassium chloride, do not exceed 0.5 mEq/kg/h.

- Parenteral administration may initially decrease the serum K^+ level. This effect is minimized by avoiding dextrose-containing fluids, administering K^+ at the correct rate and concentration, and starting oral potassium gluconate supplementation early.
- Return to alimentation and treatment of the primary disease responsible for hypokalemia will replace potassium deficits. Therefore prolonged oral maintenance therapy is rarely indicated, except when ongoing excessive loss of potassium owing to diuretic administration or polyuric renal disease is present.
- Hypokalemia markedly increases the likelihood of toxicity when digitalis is being administered. Serum potassium should be promptly checked and corrected when digitalis toxicity is suspected.
- Hypokalemia increases the risk of ventricular arrhythmias and decreases the responsiveness to class I antiarrhythmic agents (lidocaine, mexiletine, procainamide, quinidine). Serum potassium should be promptly checked and corrected when refractory ventricular arrhythmias are identified.

PROGNOSIS

- The prognosis is good if the underlying condition can be corrected or managed.

DISORDERS ASSOCIATED WITH HYPERCALCEMIA

- Hypercalcemia occurs when serum calcium concentration consistently exceeds 11.5 mg/dL in mature dogs and 10.5 mg/dL in cats or when ionized calcium exceeds 1.4 mmol/L. Young, actively growing

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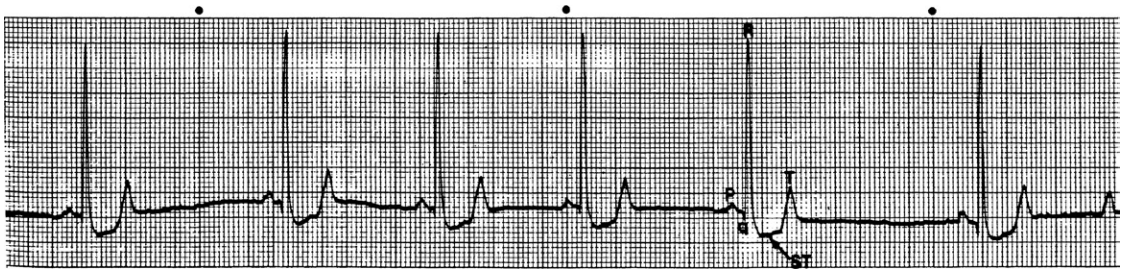


Fig. 13.3 ST-segment depression in a dog with hypokalemia (serum potassium 3.3 mEq/L) secondary to respiratory alkalosis. (From Tilley LP: *Essentials of canine and feline electrocardiography: interpretation and treatment*, ed 3, Malvern, 1992, Lea & Febiger.)

Table 13.1 Guidelines for Potassium Supplementation

SERUM POTASSIUM (MEQ/L)	MEQ KCL TO ADD TO 250 ML FLUID	MAXIMUM FLUID RATE (ML/KG/H)
<2.0	20	6
2.1–2.5	15	8
2.6–3.0	10	12
3.1–3.5	7	18
3.6–5.0	5	25

From Greene RW, Scott RC: Lower urinary tract disease. In Ettinger SJ, editor: *Textbook of veterinary internal medicine*, Philadelphia, 1975, WB Saunders.

dogs may normally have calcium values of greater than 12.0 mg/dL.

- Many different conditions can cause hypercalcemia. Hypercalcemia results from calcium bone resorption, increased gastrointestinal calcium absorption, increased serum protein binding of calcium, increased calcium binding to anions, and decreased renal and intestinal calcium removal from serum.
- The following conditions are associated with hypercalcemia:
  - Young growing puppy (normal)
  - Paraneoplastic syndromes (lymphoma, anal sac apocrine gland adenocarcinoma, occasionally other soft tissue tumors, primary and metastatic bony tumors)
  - Hypoadrenocorticism
  - Renal failure
  - Skeletal lesions (septic osteomyelitis, disuse osteoporosis, hypertrophic osteodystrophy)
  - Nutritional (hypervitaminosis D, hypervitaminosis A, calcium administration)
  - Primary hyperparathyroidism
  - Other (hemoconcentration, hyperproteinemia, severe hypothermia, laboratory error)

CARDIAC PATHOPHYSIOLOGY

- Cardiovascular effects of experimentally induced acute hypercalcemia include elevated systolic and diastolic blood pressure, especially with renal failure.
- Clinically, elevated calcium levels have little, if any, direct adverse effect on cardiac function. In severe hypercalcemia, cardiac arrhythmias or arrest may occur.
- Animals with hypercalcemia may be more predisposed to complications of digitalis intoxication.
- Long-standing hypercalcemia may predispose to calcification of the myocardium, blood vessels, and other soft tissues.

DIAGNOSIS

History and Physical Examination

- Regardless of its cause, hypercalcemia affects the kidney (renal dysfunction, polyuria, polydipsia, nocturia, nephrocalcinosis, renal failure, uremia), gastrointestinal system (anorexia, constipation, and vomiting from decreased gastrointestinal smooth muscle excitability), and nervous system (generalized skeletal muscle weakness from decreased neuromuscular activity).

Radiography

- Thoracic radiographs may indicate mediastinal mass(es), metastasis, abnormal bony densities, or osteolysis.
- Abdominal radiographs may show organomegaly, abnormal bone density, sublumbar masses, or sublumbar lymphadenopathy.
- Skeletal survey radiographs may exhibit isolated bony lesions that could account for hypercalcemia. Osteopenia, metastatic calcification, or focal bony lesions may be present.

Electrocardiography

- The characteristic ECG finding is a short QT interval. In extreme cases, the ST segment may fuse with the upstroke of the T wave.

Key Point

ECG changes do not correlate closely with serum calcium concentration.

- With severe hypercalcemia, bradycardia may occur.
- Hypercalcemia may predispose to arrhythmias of digitalis intoxication.
- ECG changes resolve after normalization of serum calcium.

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Clinical Pathology

- Hypercalcemia often causes diminished urinary concentrating ability. The urine is usually hyposthenuric or isosthenuric.
- Renal failure with azotemia may develop from progressive structural and functional alterations in the kidney.
- Hypophosphatemia or hyperphosphatemia may be recorded, depending on the presence or absence of renal failure and the underlying etiology of hypercalcemia.
- Serum alkaline phosphatase may be elevated if severe bone disease or concomitant hepatic disease is present.
- A hemogram may be normal, display hemoconcentration or anemia, or occasionally show evidence of leukemia (when this disease is associated with the underlying etiology).
- Bone marrow evaluation may be unremarkable or disclose evidence of lymphoma, leukemia, or multiple myeloma.

THERAPY

- Initial treatment is directed at reducing the systemic effects of hypercalcemia.
- Preexisting dehydration should be corrected and hydration maintained.
- Administration of 0.9% saline should be used to enhance the renal excretion of calcium.
- Furosemide (1–4 mg/kg IV, SC, PO q8–12 h) can be used if the patient is well hydrated.
- Sodium bicarbonate (1 mEq/kg IV every 10–15 minutes, up to 4 mEq/L maximum dose) may also help in cases of moderate-to-severe hypercalcemia.
- Glucocorticosteroids may limit bone resorption, decrease intestinal calcium absorption, increase renal calcium excretion, and be cytolytic in certain neoplasms. Administration may interfere with a diagnosis of neoplasia.
- Dietary therapy may be of benefit in some cats with idiopathic hypercalcemia.
- Definitive treatment is directed at the underlying etiology.

PROGNOSIS

- The prognosis depends on early detection, on the ability to establish a definitive diagnosis, and on the success of treatment. The prognosis of many causes of hypercalcemia is guarded to poor.

DISORDERS ASSOCIATED WITH HYPOCALCEMIA

- Hypocalcemia, although encountered infrequently, can cause profound clinical signs. It occurs when serum calcium concentration is less than 8 mg/dL in the dog and less than 7 mg/dL in the cat (ionized calcium less than 5 in dogs and less than 4.5 in cats).
- Serum calcium can be artificially lowered by hypoalbuminemia due to decreased protein binding.
- If hypoalbuminemia is present, then calcium should be corrected before further diagnostics.
- The following conditions may be associated with hypocalcemia:
  - Renal failure
  - Eclampsia (puerperal tetany)
  - Canine acute pancreatitis
  - Primary or secondary (nutritional) hypoparathyroidism
  - Idiopathic
  - Postoperative (during bilateral thyroid surgery)
  - Ethylene glycol toxicity
  - Hyperphosphatemia
  - Excessive diuretic therapy

CARDIAC PATHOPHYSIOLOGY

- Hypocalcemia causes an excitatory effect on nerve and muscle cells. Neuromuscular irritability, tetany, and seizures may result.
- Animals with coexisting congestive heart failure may experience decreased cardiac systolic function that improves with restoration of calcium levels (owing to the augmenting effect of calcium on myocardial contractility).
- Acutely induced hypocalcemia (i.e., treatment with chelating agents) can potentially cause sharp reductions in blood pressure resulting in cardiovascular shock.

DIAGNOSIS

History and Physical Examination

- Tetany, seizures, anorexia, vomiting, and abdominal discomfort are the predominant signs in affected animals.
- Synchronous diaphragmatic flutter has been reported in association with hypocalcemia.
- Synchronous diaphragmatic flutter is the contraction of the diaphragm in synchrony with the heartbeat due to irritability of the phrenic nerve.
- Aggressive rubbing of the ears, nose, face, and eyes can also be seen.
- Handling or stress may precipitate tetany.

Electrocardiography

- The typical ECG manifestation of hypocalcemia is QT prolongation, but QT interval duration is heart rate dependent (Fig. 13.4).

Clinical Pathology

- Clinical pathology will reflect abnormalities associated with the underlying disease process. Hypocalcemia is usually diagnosed when low serum calcium levels are detected during clinical investigation of tetany, seizures, renal failure, pancreatitis, or other disorders through analysis of a serum biochemical profile.

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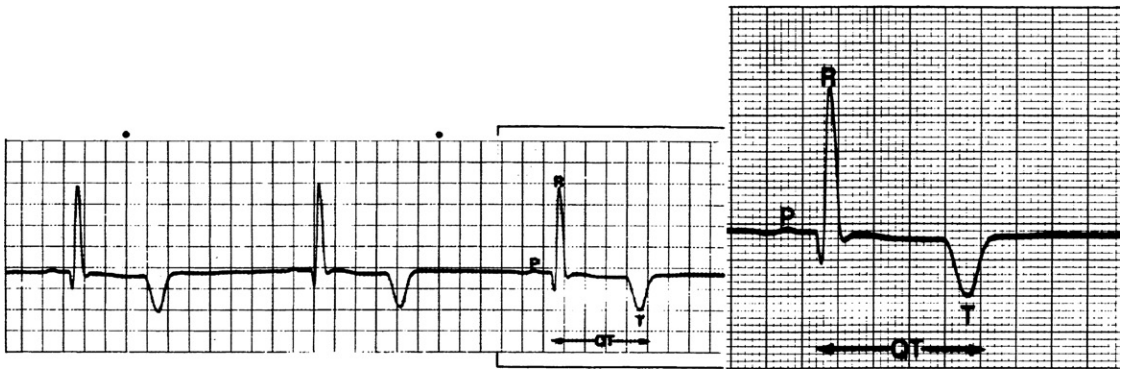


Fig. 13.4 Prolonged QT interval in a dog with severe hypocalcemia (2.2 mg/dL) secondary to ethylene glycol toxicity. (From Tilley LP: *Essentials of canine and feline electrocardiography: interpretation and treatment*, ed 3, Malvern, 1992, Lea & Febiger.)

THERAPY

- Severe hypocalcemia (<6 mg/dL) is a life-threatening emergency.
- Ten percent calcium gluconate is administered intravenously (0.5–1.5 mL/kg) slowly over a 15- to 30-minute interval.
- Calcium should be administered slowly because ECG changes do not closely conform to serum calcium levels, especially when other electrolyte and acid/base disturbances are present.
- ECG monitoring should accompany administration.
- Calcium infusion should be temporarily interrupted if QT interval shortening, ST-segment elevation, or bradycardia occurs, to be reinstated at a slower rate when these changes have abated.
- Gradual QT interval shortening from its prolonged state may accompany calcium infusion.

Key Point

Calcium is cardiotoxic if replaced too quickly; even a normal concentration of serum calcium may be toxic if replenished to that level too rapidly.

- Subsequent IV calcium administration may be repeated every 6 to 12 hours or as needed.
- Clinical signs of hypocalcemia may abate slowly despite calcium infusion. Resolution of clinical signs may not provide a reliable guide as an endpoint of adequate calcium administration. Some hypocalcemia signs (i.e., weakness, anorexia) may persist even if cardiotoxic doses of calcium are infused.
- Vitamin D therapy may be required in some disorders to maintain control of hypocalcemia. In treatment of postoperative parathyroid insufficiency, care must be taken to detect recovery of parathyroid function to avoid vitamin D–associated hypercalcemia.

PROGNOSIS

- For most causes of hypocalcemia that are carefully managed, the prognosis is good to excellent.

DISORDERS ASSOCIATED WITH HYPOMAGNESEMIA AND HYPERMAGNESEMIA

- Abnormalities in magnesium levels rarely affect cardiac function directly, but they may potentiate problems caused by other electrolyte derangements.
- Causes of hypomagnesemia are very similar to causes of hypokalemia.

CARDIAC PATHOPHYSIOLOGY

- Abnormalities of magnesium have minimal effects on the ECG with normal calcium and potassium levels.
- Hypomagnesemia will exacerbate problems due to hypocalcemia.
- Hypomagnesemia predisposes to digitalis-induced arrhythmias.
- Hypomagnesemia prevents correction of hypokalemia.

DIAGNOSIS

- Consider hypomagnesemia with conditions that lead to hypokalemia.
- Consider hypomagnesemia when there is difficulty correcting hypokalemia.
- Consider hypomagnesemia in patients with intractable cardiac arrhythmias.

THERAPY

- Treat the underlying disease process.
- Parenteral replacement if there is evidence of cardiac complications or if there is difficulty correcting hypokalemia. Magnesium sulfate at 0.75 to 1.0 mEq/kg/day in 5% dextrose in water (D_5W) as a constant rate infusion could be considered.

HYPOGLYCEMIA

- Hypoglycemia may be caused by the following:
  - Hyperinsulinism due to a functional islet cell pancreatic carcinoma
  - Insulin overdose for the treatment of diabetes mellitus

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- Nonpancreatic tumors
- Glycogen storage diseases
- Neonatal/juvenile hypoglycemia
- Septic shock
- Addison disease
- Hepatic disease

CARDIAC PATHOPHYSIOLOGY

- Irrespective of the etiologic factors, hypoglycemia triggers catecholamine release, which causes tachycardia, increases in myocardial oxygen demand, and potentially cardiac arrhythmias.

DIAGNOSIS

History and Physical Examination

- Clinical manifestations of hypoglycemia range from lethargy to episodic weakness and seizures.

Electrocardiography

- ECG changes are typically related to resultant elevated sympathetic tone, myocardial hypoxia, or concurrent electrolyte abnormalities such as hyperkalemia in Addison disease.
- ECG findings can include the following:
  - ST depression
  - T-wave flattening
  - QT prolongation
  - Supraventricular or ventricular arrhythmias
  - ECG changes are usually resolved with correction of hypoglycemia.

Laboratory Findings

- A low blood glucose level (<60 mg/dL) is usually present. A fasting blood glucose level may be needed to confirm hypoglycemia.
- Elevated insulin levels or abnormal insulin; the insulin/glucose ratio may be increased with insulinoma.

THERAPY

- A hypoglycemic crisis regardless of cause is managed by slow, IV administration of 50% dextrose to affect. A slow IV bolus of 0.5 to 1.0 mg/kg (over 1–3 minutes) could be considered.
- Treat the underlying disease or improve diabetic regulation.

PROGNOSIS

- Resolution of neurologic signs due to hypoglycemia is usually abrupt and virtually complete.
- Prolonged, untreated hypoglycemia may result in cerebrocortical hypoxic damage. Lack of symptom amelioration after therapy may indicate cerebral hypoxia and edema.

UREMIA

- Uremia is generally associated with renal failure.
- Uremia is common in geriatric populations.



Fig. 13.5 Right parasternal short-axis view at the base of the heart in a dog with protein-losing nephropathy. A well-circumscribed thrombus can be seen at the bifurcation of the pulmonary artery. *Ao*, aorta; *PA*, pulmonary artery.

- Renal failure can affect cardiac function and reduce elimination of many cardiac drugs (digoxin, most ACE inhibitors, and many beta-blockers).

CARDIAC PATHOPHYSIOLOGY

- Hypertensive cardiovascular disease, electrolyte imbalance, fluid overload, and anemia can contribute to the production of cardiac disturbances in uremic patients.
- Renal failure can cause elevations or reductions of potassium and calcium; associated changes may register on the ECG.
- Systemic hypertension is a common sequela of chronic renal failure in dogs and cats and is reported in 50% to 93% of dogs and 65% of cats.
- Pericardial effusion is reported as an uncommon sequela of uremia in cats and dogs. The effusion may occur secondary to uremic serositis.
- Pulmonary thromboembolism may occur associated with protein-losing glomerular nephropathy (Fig. 13.5).

Key Point

Uremic toxins have a cardiodepressant effect.

- Uremic pneumonitis can uncommonly cause noncardiogenic pulmonary edema.

DIAGNOSIS

History and Physical Examination

- Signs that may accompany uremia include polyuria, polydipsia, depression, anorexia, lethargy, vomiting, diarrhea, and weight loss.
- Uremic pneumonitis and metabolic acidosis cause dyspnea or tachypnea that may be confused with signs of congestive heart failure.

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Electrocardiography

- Nonspecific ECG abnormalities may be attributed to hypertension, anemia, electrolyte abnormalities, and pericarditis.
- Conduction defects or arrhythmias may occur.

Echocardiography

- Echocardiography may show changes consistent with hypertension or chronic anemia.

Clinical Pathology

- Clinical pathology may reveal abnormalities in the hemogram, urinalysis, and biochemistry profile consistent with uremia (increased blood urea nitrogen, creatinine, phosphorus, nonregenerative anemia, and isosthenuria).

THERAPY

- Treatment is directed at managing uremia and its underlying causes. Therapy generally includes parenteral fluids, protein and phosphorus restriction, phosphate binders, and sometimes calcitriol and erythropoietin.
- Management of severe azotemia in patients with concurrent heart disease is extremely challenging and often frustrating.
- Azotemia identified while treating congestive heart failure may be prerenal or renal in origin.
- Rehydrate cautiously.
  - Consider low-sodium fluids (half-strength saline).
  - Consider reduced administration rates (start at a maintenance rate).
  - It may take 48 hours to see improvement in azotemia.
- Monitor clinically and radiographically for development of congestive heart failure.
- Reduce doses or avoid cardiac drugs eliminated by the kidneys. If using ACE inhibitors or digoxin, monitor kidney function and electrolytes frequently. Monitor digoxin levels periodically, particularly if there is a decrease in appetite. Pimobendan, hydralazine, or nitroglycerin can be substituted for ACE inhibitors.
- Monitor and control systemic hypertension, electrolyte, and acid-base disturbances.

PROGNOSIS

- Resolution of the uremic state may improve ECG abnormalities, but arrhythmias may persist.
- The development of congestive heart failure in the face of severe azotemia carries a poor prognosis.

ANEMIA

- Anemia is a sign of a disease, not a disease entity.
- Evaluation should emphasize history (drug or toxin exposure), clinical signs related to other disorders complicated by anemia (fever, lethargy, bleeding, and weight loss), physical examination (petechiae, lymphadenopathy), and clinical pathology.
- Symptoms of reduced cardiac reserve associated with anemia depend on the severity of anemia, rate of anemia development, and on the presence and extent of underlying cardiac disease.

CARDIAC PATHOPHYSIOLOGY

- Tissue hypoxia results in peripheral vasodilation. This in conjunction with reduced blood viscosity leads to decreased systemic vascular resistance. The body responds to decreased systemic vascular resistance by increasing sodium and water retention, resulting in an increased stroke volume and cardiac output.
- Tissue hypoxia may also result in sinus tachycardia.
- When anemia is associated with increased viscosity (multiple myeloma or macroglobulinemia), cardiac output may fail to rise.
- Physiologic consequences depend on the developmental rate of anemia.
  - With acute blood loss (i.e., hemorrhage), hypovolemic shock may predominate.
    - Acute blood loss leads to volume depletion and a resultant decrease in cardiac output. In addition, acute anemia can lead to myocardial hypoxia and resultant depressed cardiac function.
  - With chronic anemia, blood volume is maintained, but systemic vascular resistance may fall, and cardiac output may rise as described previously.
    - The sodium and water retention may result in cardiac dilation and hypertrophy. Chronic anemia is usually well tolerated because of compensatory mechanisms that include increased cardiac output, redistribution of blood flow, and increased oxygen affinity of red blood cells.
    - Uncommonly the volume overload can result in congestive heart failure. In many of these cases, preexisting subclinical heart disease may be a factor.
    - Pulmonary thromboembolism may occur with autoimmune hemolytic anemia.

DIAGNOSIS

History and Physical Examination

- Chronic anemia produces variable symptoms that may include fatigue and mild exertional dyspnea. In rare situations, chronic anemia can lead to syncope.
- Acute anemia may present with collapse, weakness, lethargy, stupor, and dyspnea.
- Physical examination may reveal that the apex beat is hyperkinetic, bounding or pistol shot arterial

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pulses, pale or icteric mucous membrane color, and prolonged capillary refill time.
- A soft, systolic murmur at the left apex, generally grade 1 to 3 or 4, is common with severe anemia.
- Heart sounds may be accentuated.

Radiology
- Thoracic radiographs may display cardiac enlargement, but this will vary depending on the presence and extent of previously existing heart disease.
- Uncommonly, the cardiac effects of anemia can result in congestive heart failure (evidence of pulmonary venous congestion, pulmonary edema, or pleural effusion).

Electrocardiography
- Sinus tachycardia is often present. The heart rate varies with the rate of onset and degree of anemia.
- VPCs may be seen.
- Evidence of left ventricular enlargement (wide QRS complex and/or tall R waves) may be present.

Echocardiography
- It may show evidence of left ventricular, left atrial, right ventricular, or right atrial dilation.
- Hyperdynamic cardiac function (increased fractional shortening, decreased end-systolic left ventricular diameter) is usually seen in the presence of volume overload with an otherwise normal heart.
- Evidence of preexisting cardiac disease may be present.

Clinical Pathology
- The laboratory database varies considerably depending on the cause of anemia.

THERAPY
- Treatment of anemia depends on the underlying cause.
- Animals with congestive heart failure secondary to anemia should be given furosemide as needed. Once the anemia is controlled, chronic cardiac therapy may not be necessary.
- Animals with concurrent congestive heart failure and life-threatening anemia should be given furosemide and transfused simultaneously. Packed red blood cells may be more appropriate than whole blood owing to the smaller volume that can be administered to correct the anemia.
- Caution with fluid administration should always be exercised when cardiomegaly is present in the face of significant anemia.
- A decision as to when to transfuse a patient is based on the clinical assessment, which includes the rate of anemia development, the patient's state of compensation, and the presence of continued red blood cell loss.

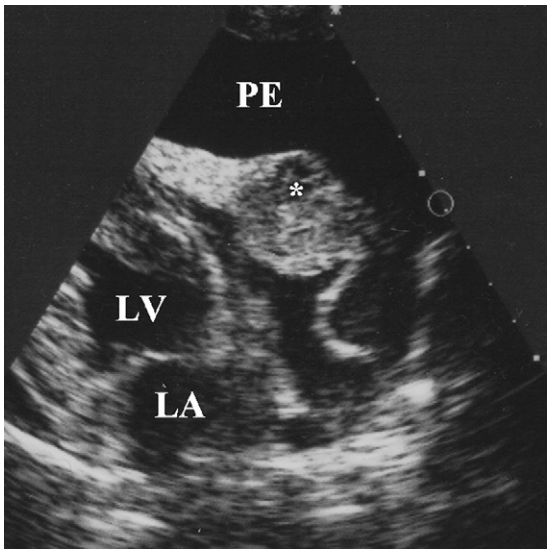


Fig. 13.6 Slightly oblique right parasternal long-axis view. A large soft tissue mass effect (*asterisk*) can be seen in the wall of the right atrium. Moderate pericardial effusion (*PE*) is present resulting in diastolic collapse of the right atrium. *LA*, Left atrium; *LV*, left ventricle.

PROGNOSIS
- Successful management of anemia and underlying causes confers a good short-term prognosis. Long-term prognosis depends on the cause of the anemia.

NEOPLASTIC AND INFILTRATIVE HEART DISEASES

HEMANGIOSARCOMA
- This tumor originates from endothelial cells.
- It can be seen in any portion of the heart but appears to be more common in the wall of the right atrium (Fig. 13.6).
- It is rare in cats.
- Average age of affected dogs is 10 years, no gender predilection, and the incidence is increased in German Shepherds.
- Metastasis is common.
- Other common sites of hemangiosarcoma include the spleen, liver, and lungs.

CHEMODECTOMA
- This tumor originates from neuroepithelial cells most commonly found in the aortic bodies.
- Masses are typically seen at the heart base. Because of the complexity of the heart base, small masses can be difficult to identify (Fig. 13.7).
- It is rare in cats.
- It is more common in dogs older than 6 years, there is no gender predilection, and there is an increased incidence in brachycephalic breeds.
- Metastasis is uncommon but can appear in lungs, liver, lymph nodes, and bone.

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OTHER CAUSES

- Lymphoma is the most common tumor that metastasizes to the heart in cats, but it also can be seen in dogs (Figs. 13.8 and 13.9).
- Ectopic thyroid carcinomas occur at the heart base and rarely in the right ventricular outflow tract.
- Cardiac myxomas are rare but are more common in the right heart (right atrial lumen) (Fig. 13.10).
- Rarely, cardiac fibroma, fibrosarcoma, and rhabdomyosarcoma are diagnosed in young dogs.
- Infiltrative myocardial disease (e.g., amyloidosis, hypereosinophilic syndrome) involving nonneoplastic cells is rare in dogs and uncommon in cats.

CARDIAC PATHOPHYSIOLOGY

- Tumors from other tissues gain access to the heart by invasive growth from adjacent structures or spread by hematogenous or lymphatic vessels.

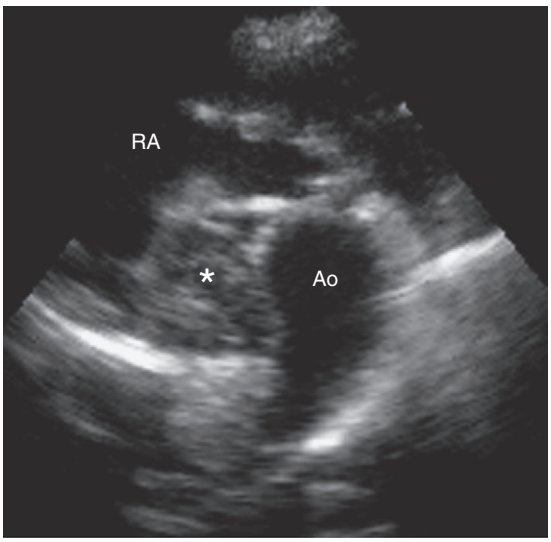


Fig. 13.7 Slightly oblique right parasternal short-axis view at the heart base above the left atrium. A small, soft tissue mass effect (*asterisk*) can be seen associated with the aorta (*Ao*) and above the left atrium. Mild-to-moderate pericardial effusion is also present. *RA*, Right atrium.

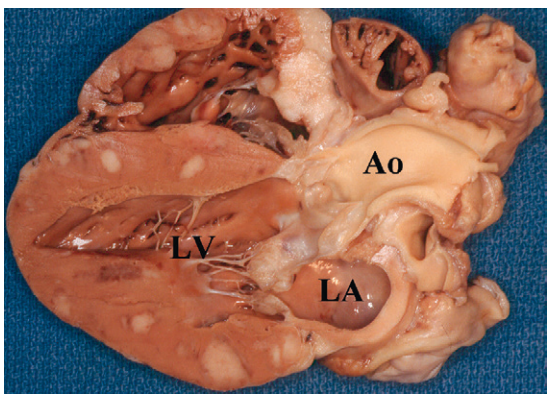


Fig. 13.8 Transverse section through the heart of a dog with cardiac lymphoma. Multiple pale nodules can be seen in the left and right ventricular myocardium. *Ao*, Aorta; *LA*, left atrium; *LV*, left ventricle.

- The relative infrequency of cardiac metastasis has been attributed to metabolic peculiarities of striated muscle, rapid coronary blood flow, efferent lymphatic drainage from the heart, and the vigorous kneading action of the myocardium.
- Myocardial neoplasia may cause congestive heart failure because of diastolic dysfunction, obstruction to ventricular inflow or outflow, pericardial tamponade, or arrhythmias.

DIAGNOSIS

History and Physical Examination

- Cardiac involvement may not always lead to clinical signs. Signs depend on the site and extent of metastasis. Resultant cardiac dysfunction may cause signs of congestive heart failure, weakness, or syncope.
- Pericardial effusion and tamponade may lead to similar clinical signs.
- Possible physical findings include muffled heart sounds on auscultation, jugular venous distention, ascites, weakness (often acute), hypokinetic pulses, tachypnea, weight loss, and pale mucous membranes.

Radiography

- Radiographic evidence of a cardiac mass is uncommonly seen (Fig. 13.11).
- Cardiac enlargement and rounding of the cardiac silhouette may be observed if pericardial effusion is present.
- Dilation of the caudal vena cava may be present secondary to cardiac tamponade or obstruction to blood flow within the lumen of the right heart. In general, the diameter of the caudal vena cava should be less than the length of the thoracic vertebra above the carina.
- Pneumopericardiography or angiocardiography may assess the thickness and any irregularities of the pericardium, epicardium, and endocardium.
- The use of MRI or CT angiography may allow visualization of a cardiac mass, but cardiac gating is necessary.

Electrocardiography

- Reduced QRS voltage on the ECG and electrical alternans occur in many but not all cases with pericardial effusion.
- Atrial or ventricular arrhythmias, conduction disturbances, and complete heart block can occur.

Echocardiography

- Echocardiographic evidence of pericardial effusion may be demonstrated. Diastolic right atrial or right ventricular collapse signifies pericardial tamponade. Dilation of the abdominal vena cava and/or hepatic veins is also typically seen with tamponade.

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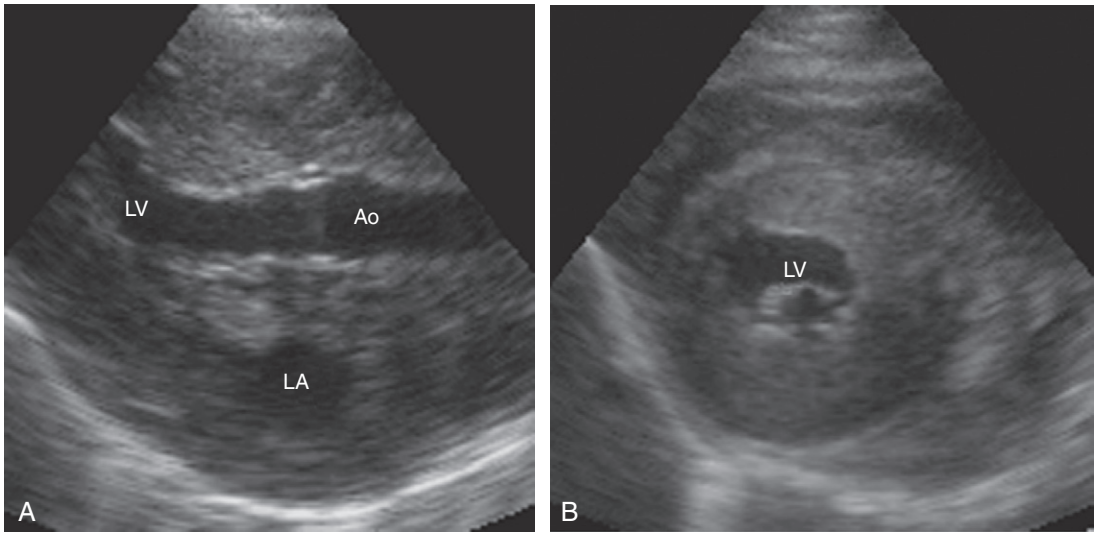


Fig. 13.9 Right parasternal long-axis **(A)** and short-axis **(B)** views in a cat with cardiac lymphoma confirmed on histopathologic examination. Note the marked thickening of the left ventricular and left atrial myocardium. *Ao*, Aorta; *LA*, left atrium; *LV*, left ventricle.

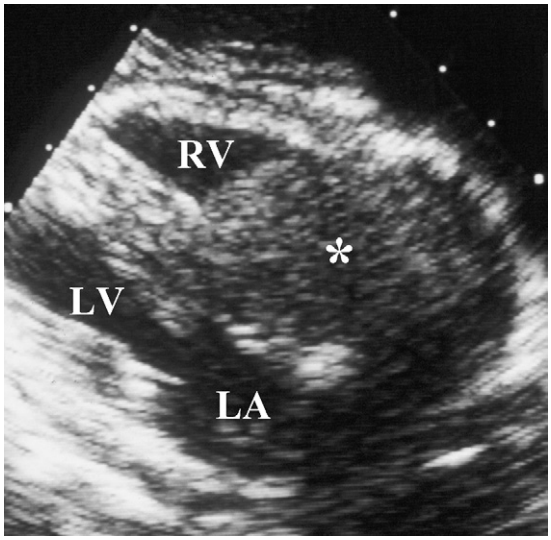


Fig. 13.10 Right parasternal long-axis view in a dog. A large, well-circumscribed mass effect (*asterisk*) is seen filling most of the right atrial lumen. The mass was confirmed as a myxoma on histopathologic examination. *LA*, Left atrium; *LV*, left ventricle; *RV*, right ventricle.

- Two-dimensional echocardiography may facilitate identification of cardiac neoplasia. Unfortunately, because of the elusive nature of some cardiac masses, the lack of a mass on echocardiogram does not rule out neoplasia.
- Whenever safely possible, an echocardiogram should be performed while pericardial fluid is present. The fluid can outline the borders of a mass, improving identification.
- Pericardiocentesis may uncommonly detect cytologic evidence of neoplasia. Most of the time, fluid analysis will come back as nonspecific hemorrhagic effusion.
- Biopsy of the tumor is possible without thoracotomy with techniques such as myocardial biopsy or thoracoscopy.

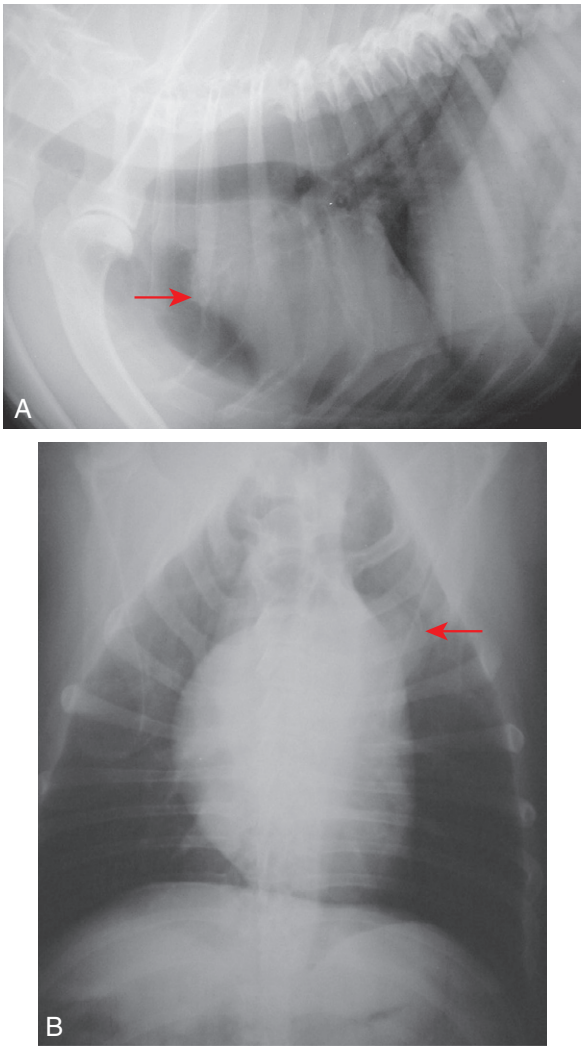


Fig. 13.11 Lateral **(A)** and ventrodorsal **(B)** radiographs from a Boxer with a large heart base mass. A bulge in the area of the cranial waist (*arrow* in A) of the cardiac silhouette can be seen on the lateral. A similar bulge can be seen on the ventrodorsal projection at the 2 o'clock position (*arrow* in B) on the cardiac silhouette.

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THERAPY

- Pericardiocentesis may offer temporary relief from cardiac tamponade.
- If recurrent pericardiocentesis is necessary, then partial pericardectomy via thoracotomy or a pericardial window via thoracoscopy could be considered.
- Surgery would likely improve the patient's quality of life but may not prolong life.
- Other than standard thoracotomy concerns and risks, the main complication with surgery is ongoing hemorrhage from the tumor. This can result in hemothorax.
- Animals with myocardial infiltration of tumors sensitive to chemotherapy or radiotherapy may respond favorably, but this is uncommon.
- Congestive heart failure and arrhythmias resulting from cardiac neoplasia can be difficult to treat.
- Surgical resection or debulking of cardiac masses is not commonly possible.
- Masses confined to the right atrial appendage can periodically be removed with resection of the appendage.
- Some intraluminal cardiac masses can be removed with cardiac bypass or inflow occlusion. Studies with long-term follow-up with such patients are not available.

PROGNOSIS

Hemangiosarcoma

- Surgical resection may be possible, but distant microscopic metastasis is almost always present.
- Poor response to chemotherapy in general, although a combination of surgical resection and chemotherapy may improve the prognosis to some degree.
- Pericardectomy may improve quality of life.

Chemodectoma

- In many cases, heart base tumors can be an incidental finding. Because they are often slow growing, they can be present for extended periods before clinical signs develop.
- Debulking surgery is an option, but complete resection of the tumor is rarely possible.
- Pericardectomy may improve quality of life.
- Radiation therapy may be beneficial in some cases, but general response to chemotherapy is poor.

Key Point

In general, long-term prognosis with cardiac cancer is poor because of difficulty in surgical resection, minimal response to chemotherapy and radiation therapy, and detrimental side effects of chemotherapy and radiation therapy on the heart.

PHYSICAL AND CHEMICAL AGENTS THAT AFFECT THE CARDIOVASCULAR SYSTEM

- Many noninfectious stimuli can cause myocardial injury. Damage can be acute and transient or chronic and lead to permanent myocardial changes. Effects are usually related to the severity, dose, and rate of exposure.

HYPERPYREXIA (HEAT STROKE)

- Hyperpyrexia is usually associated with heat stroke when the rectal temperature rises to 41°C to 44°C (106°F–111°F). Experimentally signs of heat stroke develop consistently at temperatures above 43°C (109°F) in dogs.
- Heat stroke develops in animals exposed to high environmental temperatures or during heavy activity in warm environmental temperatures. It commonly occurs in animals left in unventilated automobiles during the summer months.
- High humidity reduces evaporation of water from the oral and nasal cavities and reduces the ability of small animals to regulate their temperatures.
- Animals that are obese, have brachycephaly, are very young, are very old, or have cardiopulmonary disease are predisposed to heat stroke.
- Malignant hyperthermia is a rare but a potentially fatal abnormal response to anesthetic agents. It is characterized by an acute, rapid rise in body temperature and severe biochemical changes accompanied by muscle rigidity.

CARDIAC PATHOPHYSIOLOGY

- Heat-induced vasodilation results in hypotension and decreased organ perfusion.
- Respiratory alkalosis may result from panting.
- Dehydration with hemoconcentration may occur, in addition to mild hyperkalemia and hypophosphatemia.
- Tachyarrhythmias and cardiogenic shock can occur owing to myocardial hemorrhage, ischemia, and necrosis.
- Disseminated intravascular coagulopathy (DIC) may develop owing to destruction of clotting factors.
- Right-heart dilation may occur.
- Mechanisms suggested for myocardial injury include direct thermal effects, circulatory collapse with secondary myocardial hypoxia, decreased coronary blood flow, and secondary metabolic injury.
- Animals that recover from heat stroke may be prone to subsequent occurrences as a result of heat-induced damage to the thermoregulatory zone of the hypothalamus.

DIAGNOSIS

History and Physical Examination

- The diagnosis is usually made on the reported history of an animal being exposed to high environmental temperatures. However, some cases have a more subtle history.

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- Depending on the duration and severity of the heat stroke, clinical signs may include panting, sinus tachycardia, bright red oral mucous membranes, and hyperthermia. Extremities become hot to the touch.
- Red mucous membranes may turn pale because of diminished circulation or vasoconstriction.
- Watery diarrhea may occur, which can progress to bloody diarrhea.
- Stupor and coma with respiratory arrest can follow.
- With malignant hyperthermia, an acute, profound temperature elevation may be the first noticeable sign to the surgeon.
- If the heart rate is monitored, tachycardia may be the earliest indication.
- Skeletal muscle hypertonus may occur.
- ECG may show tachycardia, extrasystoles (especially if DIC develops), and ST-segment and T-wave abnormalities.
- Clinical pathologic alterations relate to the stage and severity of the hyperpyrexia.

THERAPY

- Therapy should begin with lowering the body temperature. Submergence in cool water and wetting the extremities with rubbing alcohol are useful techniques. Rectal temperature should be recorded every 5 to 10 minutes. Discontinue cooling when the body temperature drops below 39.5°C (103°F), to avoid hypothermia.
- If laryngeal paralysis, laryngeal edema, or tracheal stenosis is present, it may impede heat loss through the respiratory system. If these conditions are present, then a temporary tracheotomy or endotracheal intubation may have to be performed.
- Supportive care should include isotonic fluid therapy (0.45% saline or half-strength lactated Ringer solution with 2.5% dextrose), correction of acid-base and electrolyte abnormalities, and treatment for DIC.
- Nonsteroidal antiinflammatory drugs or antipyretics (e.g., aspirin, flunixin meglumine) are contraindicated. The use of glucocorticoids remains controversial, but they are often used when cerebral edema is suspected.
- When malignant hyperthermia is anesthetic related, all inhalation agents and relaxants should be discontinued. Hyperventilation with 100% oxygen should be started.
- For malignant hyperthermia, dantrolene 2 mg/kg IV, up to 10 mg/kg IV, has been used in the dog. No proved efficacy is known. Its use is based on experience from humans, horses, and pigs. Dantrolene exhibits muscle relaxation activity by direct action on muscle.
- Administer mannitol (0.25–2 g/kg of a 20% solution over 15–20 minutes) if cerebral edema is suspected or if stupor or coma develops.
- The patient should be monitored for several days for the development of complications, such as renal failure and DIC.

PROGNOSIS

- The prognosis for heat stroke is guarded to good if detected and treated early.
- Malignant hyperthermia is rare but seems to offer a worse prognosis.
- Coagulopathy and renal failure confer a worse prognosis.

HYPOTHERMIA

- Lowering of body temperature can occur accidentally, as a result of deep cooling from external cold, drugs, or interference with thermoregulatory centers during anesthesia. Physiologic consequences of hypothermia depend on the extent and duration of exposure.

CARDIAC PATHOPHYSIOLOGY

- Cardiac dilation with epicardial and subendocardial hemorrhage can result from severe hypothermia. Microinfarcts can occur in myocardial tissue.
- Lesions may result from circulatory collapse, hemoconcentration, sludging of blood in capillaries, or decreased cellular metabolism.

DIAGNOSIS

History and Physical Examination

- Signs vary with the degree of hypothermia (reduction in core temperature), mild (32°C–35°C or 89.6°F–95°F), moderate (28°C–32°C or 82.4°F–89.6°F), and severe (<28°C or 82.4°F).
- Signs include lethargy, ataxia, stupor, shivering, and bradycardia. Cardiopulmonary arrest may occur with severe hypothermia.

Electrocardiography

- Mild hypothermia results in prolongation of the PR interval, QRS duration and QT interval, APCs, and inverted T waves.
- Moderate hypothermia produces atropine-resistant bradycardia and ventricular arrhythmias.
- Severe hypothermia may cause cardiac arrest secondary to ventricular fibrillation or asystole.

THERAPY

- Treatment initially involves warming the patient.
- Active external warming with warm-water blankets is recommended for mild to moderate hypothermia. External warming causes vasodilation of skin vessels, which may initially transfer cooled blood to the body core, further lowering internal temperature. This is rarely a significant clinical complication with mild to moderate hypothermia.

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- Internal (core) warming is recommended if the rectal temperature is less than 30°C (86°F) or the cardiovascular status is unstable. This is accomplished by peritoneal dialysis with dialysate fluids warmed to 45°C (113°F).
- Appropriate supportive care (e.g., IV fluids, ventilation) must be administered. Severe hypothermia can cause cardiopulmonary collapse. Keep these patients well oxygenated and administer warmed IV fluids.
- Cardiac output and ECG abnormalities improve after warming.

- Evaluation of the patient for underlying systemic disorders that could have predisposed it to hypothermia should be performed once normothermia has been achieved.

CARBON MONOXIDE TOXICITY

- Carbon monoxide is a colorless, odorless, tasteless, toxic gas that has the molecular formula CO. CO is produced by the incomplete combustion of the fossil fuels: gas, oil, coal, and wood used in boilers, engines, oil burners, gas fires, water heaters, solid fuel appliances, and open fires. CO is a commercially important chemical. It is also formed in many chemical reactions and in the thermal or incomplete decomposition of many organic materials.
- Dangerous amounts of CO can accumulate when, as a result of poor installation, poor maintenance or failure, or damage to an appliance in service, fuel is not burned properly or when rooms are poorly ventilated and produced CO is unable to escape.

CARDIAC PATHOPHYSIOLOGY

- CO competes with oxygen for binding sights on hemoglobin, diminishing oxygen transport capacity. This results in myocardial hypoxia. Myocardial hemorrhage and necrosis may occur.

DIAGNOSIS

History and Physical Examination

- The history often supports exposure to fumes from a gasoline-burning engine.
- Clinical signs are often associated with cardiac hypoxia (sudden death) or hypoxia to the brain (convulsions, disorientation, and coma).
- The physical examination is often unremarkable.
- A blood sample will be cherry red.

Electrocardiography

- ECG abnormalities can include ST-segment changes, conduction abnormalities, or arrhythmias.

Specialized Diagnostic Test

- Carboxyhemoglobin blood levels can be measured.

TREATMENT

- One hundred percent oxygen should be administered.
- Any arrhythmias should be treated as needed.
- Supportive care should include correction of electrolyte and acid-base imbalances.

PROGNOSIS

- Clinical recovery usually occurs if the situation is detected and treated early.

TOAD POISONING

- The Colorado River toad (*Bufo alvarius*) and the marine toad (*Rhinella marina*, formerly *Bufo marinus*) secrete toxins from the parotid glands that can cause profound cardiotoxicity. The parotid gland secretions contain epinephrine, norepinephrine, dopamine, serotonin, bufotenine, bufagenins, and bufotoxins. The animal does not have to ingest the toad to become poisoned. Toxic parotid secretions can be absorbed through the oral mucous membranes just by holding the toad in its mouth.

CARDIAC PATHOPHYSIOLOGY

- Bufagenins and bufotoxins can have digitalis-like effects on the heart.

DIAGNOSIS

History and Physical Examination

- The pet is often observed playing with the toad.
- Clinical signs occur within minutes and may include hypersalivation, vomiting, diarrhea, weakness, pulmonary edema, and seizures. Coma and death can occur within 30 minutes.

Electrocardiography

- In experimental studies, the digitalis-like effect of the toxin may result in any type of arrhythmia. In natural exposure, arrhythmias are rare. The most common rhythms noted are sinus arrhythmia and sinus tachycardia.
- Severely intoxicated dogs with bradycardia, tachycardia, neurologic disability, or signs of shock should get an initial ECG. Monitoring the ECG is recommended if significant arrhythmias are noted.

THERAPY

- The patient's mouth should be rinsed immediately. Atropine may help decrease salivation but is reserved for patients with heart rates less than 50 beats/min. The inclusion of atropine to treat excess salivation in dogs with normal heart rates or tachycardia can lead to more severe arrhythmias.

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- Vomiting should be induced if ingestion was recent. The venom may enter enterohepatic circulation; therefore multiple doses of activated charcoal should be administered. Sorbitol cathartic is advised.
- Provide supportive care, including anticonvulsive and antiarrhythmic medication, as needed.
- Pentobarbital anesthesia will control seizures.
- Propranolol is quite effective for tachyarrhythmias. In patients without asthma or preexisting heart disease, doses of 0.02 to 0.06 mg/kg can be given slowly IV, to effect.
- Ventricular and supraventricular arrhythmias can be present. Treat arrhythmia as any other arrhythmia. In humans, the use of digoxin-specific antigen-binding fragments (digoxin Fab) has been reported in Bufo toad toxicosis, but there are no reports of its use in canine or other small animal patients. The dose is uncertain in dogs. In digoxin toxicity, one vial of digoxin Fab is needed to counter 1 mg of digoxin, then it is speculated you may need multiple vials to treat bufo toxicosis, which can be cost prohibitive.

OLEANDER TOXICITY

- Oleander (*Nerium oleander*) is an ornamental shrub found mostly in the southeast United States.

CARDIAC PATHOPHYSIOLOGY

- The toxin of oleander is a digitalis-like glycoside.

DIAGNOSIS

History and Physical Examination

- Signs of vomiting and diarrhea occur within 2 to 3 hours.
- ECG may reveal any type of arrhythmia due to the digitalis-like compounds.

THERAPY

- Supportive care that includes fluid therapy, electrolyte replacement, and control of vomiting and diarrhea is recommended.
- Arrhythmias should be treated as needed.

CHOCOLATE TOXICITY

- Toxic effects of chocolate are due to methylxanthines, in particular theobromine and caffeine.
- The median lethal dose of theobromine and caffeine is 100 to 200 mg/kg (0.2–0.5 oz baking chocolate/kg). Mild signs can occur with 20 mg/kg, severe signs at 40 to 50 mg/kg, and seizures at 60 mg/kg.

CARDIAC PATHOPHYSIOLOGY

- Theobromine increases cyclic adenosine monophosphate, causes release of catecholamines, blocks adenosine receptors, and increases calcium uptake by the sarcoplasmic reticulum.
- The net effect is to increase heart rate and the force of contractions.

DIAGNOSIS

History and Physical Examination

- Clinical signs associated with chocolate toxicity include vomiting and diarrhea, hyperactivity, ataxia, hyperthermia, muscle tremors, coma, and death.
- Tachycardia and bradycardia are frequently noted.
- ECG findings may include sinus tachycardia, sinus bradycardia, and ventricular arrhythmias.

THERAPY

- Symptomatic therapy should include prevention of absorption, increasing elimination, maintenance of life support, and control of cardiac arrhythmias and seizures.
- Vomiting should be induced if ingestion was recent. Activated charcoal (1–4 g/kg orally) should be administered. Because methylxanthines undergo enterohepatic recirculation, giving repeated doses of activated charcoal is usually beneficial in animals with symptoms.
- Arrhythmia management
  - Lidocaine is used for control of ventricular arrhythmias, if needed. Beta-blockers can be added if additional control and maintenance therapy is required. Because propranolol hydrochloride reportedly delays renal excretion of methylxanthines, metoprolol succinate or metoprolol tartrate is the beta-blocker of choice, if available.
  - Treat supraventricular tachycardia with beta-blockers (i.e., propranolol, metoprolol, atenolol).
  - Seizure control can be done with diazepam or barbiturate.

DOXORUBICIN CARDIOTOXICITY

- Doxorubicin is an anthracycline antibiotic used for its antineoplastic properties. Its mechanism of action as a chemotherapeutic agent is inhibition of nucleic acid synthesis. Cardiotoxicity manifested as arrhythmias or DCM usually develops after cumulative doses of 240 mg/m^2 in dogs, but toxicity can occur at dosages as low as 130 mg/m^2 (dog range 135–265 mg/m^2; cat range 130–320 mg/m^2). Clinical studies in humans have shown a reduced incidence of toxic cardiomyopathy when people are treated with prolonged infusions (24 hours) of doxorubicin. Prolonged infusion therapy in dogs is still under investigation.
- Acute cardiotoxicity is manifested by arrhythmias during or shortly after administration. DCM may develop with chronic treatment. Although histologic lesions and echocardiographic changes may develop, clinical signs of congestive heart failure are uncommon in cats treated with doxorubicin.

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CARDIAC PATHOPHYSIOLOGY

- The mechanism by which doxorubicin causes cardiotoxicity is unknown, but free radical generation and lipid membrane peroxidation may be involved in the pathogenesis. The heart rate at the time of drug administration may also determine the degree of cardiac toxicity, with slower heart rates associated with less toxicity.
- Histologic lesions consist of myocyte vacuolization, myocytolysis, and occasionally, interstitial fibrosis.

DIAGNOSIS

History and Physical Examination

- Clinical signs and physical findings are associated with DCM and include exercise intolerance, dyspnea and coughing, hepatomegaly, and ascites. Congestive heart failure usually develops acutely.

Electrocardiography

- In dogs, ECG abnormalities include ST-segment and T-wave changes, decreased QRS voltages, intraventricular conduction abnormalities, and atrial and ventricular arrhythmias. ECG changes are rare in cats treated with doxorubicin.
- ECG changes can occur acutely during drug administration. In this situation, stopping the drip and then reinstituting it at a slower rate may resolve the problem.

Radiography

- With DCM, radiography may show cardiomegaly, pulmonary edema, pleural effusion, hepatomegaly, and ascites.

Echocardiography

- The most consistent abnormalities with doxorubicin cardiotoxicity are increased left ventricular end-systolic internal dimensions and decreased fractional shortening. As cardiac function deteriorates, progressive left atrial enlargement can be seen.

THERAPY

- If congestive heart failure develops, the patient should be treated accordingly. Unfortunately, the therapeutic response is poor. An ECG is recommended before each doxorubicin treatment, especially in dogs. If arrhythmias develop, doxorubicin treatment should be discontinued. An echocardiogram is recommended after the third cycle in dogs or fifth cycle in cats, and every one to three cycles thereafter. Doxorubicin treatment should be discontinued if a reduction in contractility from baseline is recognized.
- Protection from cardiomyopathy has been demonstrated with dexrazoxane.
- The mechanism for cardio protection is not understood. One suggested mechanism is dexrazoxane, which prevents the formation of free radicals and subsequent lipid peroxidation that occurs from free iron and iron associated with the doxorubicin-iron complex.
- It is approved by the Food and Drug Administration (FDA) for use in females with doxorubicin administration against metastatic breast cancer.
- It has been shown in studies to protect mice, rats, hamsters, pigs, and dogs from doxorubicin cardiac toxicity.
- Although it appears to prevent cardiotoxicity, it does not protect against other toxic effects caused by anthracyclines.
- Dexrazoxane ameliorates anthracycline extravasation lesions.
- The recommended dose ratio of dexrazoxane to doxorubicin is 10:1. It is given as an infusion over 15 minutes, 30 minutes before doxorubicin is used.
- Expense of the drug limits its routine use.

INFECTIOUS/INFLAMMATORY DISEASES

- The heart can be involved in an inflammatory process because of many infectious agents. Myocardial cells, myocardial interstitium, or cardiac vessels may be affected.

CARDIAC PATHOPHYSIOLOGY

- Myocardial injury can occur from direct myocardial invasion of an infectious agent, from a myocardial toxin produced by the agent, or from a secondary immune-mediated reaction.
- Important causes of myocardial injury in small animals include the following:
  - Bacterial myocarditis
    - Pyogenic bacteria originate in septicemic states or from other septic foci.
    - Clinical signs range from subclinical and nonspecific (weight loss, lethargy) to apparent and specific (arrhythmias, congestive heart failure).
  - Viral myocarditis has been associated with canine parvovirus and distemper virus in dogs and with suspected but not yet identified viral pathogens in cats. Clinical abnormalities include cardiomegaly, arrhythmias, and nonspecific ECG abnormalities. The severity of the cardiac changes ranges from congestive heart failure with canine parvovirus to subclinical myocarditis with canine distemper virus.
  - Fungi and algae can infect the heart secondary to a disseminated disease process, often in conjunction with reduced host defense.
  - Protozoal myocarditis can occur in dogs with canine trypanosomiasis (*Trypanosoma cruzi*) or in dogs and cats with toxoplasmosis (*Toxoplasma gondii*).
    - Trypanosomiasis is discussed later.

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- Toxoplasmosis can produce signs characterized by the extent and severity of the multiorgan system involvement.

Therapy

- Treatment is often supportive and usually focused on the most prominent systemic manifestation of the disease process.

BORRELIOSIS (LYME DISEASE)

- Borreliosis is caused by the spirochete *Borrelia burgdorferi*. Transmission is by ticks of the *Ixodes* species. The incidence of cardiac involvement is unknown in animals. In humans, 10% of patients with borreliosis have cardiac involvement. Borreliosis in animals is usually acute and cured by antibiotics. However, the prognosis may be poor if second-stage disease involving the heart, kidneys, or central nervous system (CNS) develops.

CARDIAC PATHOPHYSIOLOGY

- Spirochetes have been isolated from myocardial biopsies in humans.
- DCM has been reported in a few humans with Lyme disease. Cardiac damage may involve an immune-mediated mechanism.

DIAGNOSIS

History and Physical Examination

- Ticks may be found on dogs, or owners may report recent exposure to ticks in endemic areas.
- Although nonspecific, clinical signs include anorexia, depression, and lameness.
- Physical examination reveals fever, joint pain, and lymphadenopathy.

Electrocardiography

- The most common ECG finding in humans is AV block.
- Ectopic beats and ST-segment abnormalities have also been reported in humans.
- AV block secondary to *B. burgdorferi* in the dog has been reported in one case.

Specialized Diagnostic Tests

- Commercial test kits are available, but titers should be interpreted in view of the history, physical examination, and clinical signs. False-positive and false-negative titers are not uncommon.

THERAPY

- Antibiotics are the treatment of choice for borreliosis. Tetracycline, doxycycline, ampicillin, or amoxicillin appear to be the most effective first line agents.
- Humans with cardiac involvement are often treated with IV ceftriaxone or cefotaxime.
- A 5- to 7-day course of antiinflammatory doses of corticosteroids may be helpful for heart block that does not rapidly resolve with appropriate antibiotic therapy.

TRYPANOSOMIASIS (CHAGAS DISEASE)

- Trypanosomiasis is a rare disease caused by the protozoan *Trypanosoma cruzi*. It usually occurs in young dogs in the southeastern United States. Transmission is through the bite of a reduviid bug.

CARDIAC PATHOPHYSIOLOGY

- During the acute stage (2–4 weeks after infection), cardiac damage occurs when trypomastigotes rupture from myocardial cells. This causes myocardial failure, conduction disturbances, and arrhythmias.
- If animals survive the acute stage, they may remain symptom free for months. During this time, myocardial degeneration occurs, and DCM develops. Myocardial damage during this stage may be related to immune mechanisms or to the release of toxic parasitic products.

DIAGNOSIS

History and Physical Examination

- Clinical signs during the acute stage are related to left-sided and (mainly) right-sided heart failure. Collapse and sudden death may be noted in a previously healthy dog. Gastrointestinal signs of anorexia and diarrhea are also common. The lymph nodes will be enlarged during the acute stage.
- Clinical manifestations during the chronic stage are associated with DCM, with signs of right-sided heart failure often predominating. Various cardiac arrhythmias also occur. The cardiomyopathy is not curable and responds poorly to treatment.
- Physical findings associated with heart failure—pale mucous membranes, weak femoral pulses, and ascites—can be seen during acute and chronic stages.

Electrocardiography

- Conduction disturbances, ectopic beats, and sustained arrhythmias can be seen during the acute and chronic stages.

Radiography

- Radiography may show cardiomegaly, pulmonary edema, pleural effusion, hepatomegaly, or ascites.

Echocardiography

- Echocardiography is normal during the acute phase. As the disease becomes chronic, ventricular contractility and wall thickness decrease, and cardiac chambers dilate.

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Specialized Diagnostic Tests

- Trypomastigotes may be seen on a blood smear during the acute stage. The patient becomes aparasitemic 2 to 4 weeks after infection.
- Indirect fluorescent antibody, direct hemagglutination, and complement fixation tests confirm antibodies to *T. cruzi*.
- Blood cultures can be performed but are time-consuming.

THERAPY

- Treatment of this disease is very unrewarding. By the time the diagnosis is made, the disease is often no longer responsive to antiprotozoal medication.
- Various therapeutic recommendations include oral benzimidazole (5 mg/kg PO q24h for 2 months), allopurinol (30 mg/kg PO q12h for 100 days), or nifurtimox (2–7 mg/kg PO q6h for 3–5 months).
- Alert owners and veterinary staff to potential zoonotic risk.

PARVOVIRUS

- Parvovirus has caused cardiac disease when it infected puppies less than 2 weeks of age. Current cases of parvoviral myocarditis are very rare.

CARDIAC PATHOPHYSIOLOGY

- Viral multiplication occurs in rapidly dividing myocardial cells, resulting in cell death and scarring.

DIAGNOSIS

History and Physical Examination

- The cardiac form of parvovirus often results in sudden death.
- Some puppies that survived the acute phase developed heart failure weeks to months later and died from cardiac arrhythmias or DCM 6 to 12 months later.
- ECG reveals atrial or ventricular arrhythmias.

Echocardiography

- In some infected dogs, echocardiography revealed abnormalities consistent with DCM, including decreased fractional shortening, left atrial and ventricular enlargement, and increased E-point septal separation.

THERAPY

- Heart failure is managed with diuretics, pimobendan, and ACE inhibitors.
- Arrhythmias should be treated accordingly.

MISCELLANEOUS DISEASES

SYSTEMIC LUPUS ERYTHEMATOSUS

- Cardiac disease due to systemic lupus erythematosus (SLE) is a common finding in humans, rare in dogs, and not reported in cats. Pericarditis is the most common cardiac abnormality associated with SLE in humans. Other abnormalities in humans include myocarditis, congestive heart failure, and valvular heart disease. Ventricular arrhythmias were reported in two dogs and heart failure in one dog diagnosed with SLE.

Cardiac Pathophysiology

- Pericarditis occurs secondary to vasculitis.
- Myocarditis and endocarditis can also occur.

DIAGNOSIS

HISTORY AND PHYSICAL EXAMINATION

- Clinical signs associated with heart disease are usually overshadowed by systemic signs of SLE.
- Clinical findings associated with arrhythmias or heart failure may be present.
- ECG may reveal ventricular arrhythmias.
- Echocardiographic abnormalities have not been reported in dogs or cats with SLE.

SPECIALIZED DIAGNOSTIC TESTS

- A diagnosis of SLE can be made with a positive antinuclear antibody titer in conjunction with clinical signs.

Therapy

- Immunosuppressive doses of prednisone (2 mg/kg PO q12h) are advised. If there is no improvement after 7 days, more aggressive immunosuppressive therapy should be considered.
- Pericardiocentesis is recommended if pericardial effusion is present.

NEUROGENIC CARDIOMYOPATHY

- In client-owned animals, CNS disease has been associated with myocardial lesions in all domestic species except the cat. Experimentally, stimulation of specific areas of feline brains can cause myocardial necrosis. Most cases involve trauma to the brain or spinal cord. CNS neoplasia, infection, encephalomalacia, and ruptured intervertebral discs can also result in myocardial damage.

CARDIAC PATHOPHYSIOLOGY

- Histologic lesions include degeneration or disintegration of myocardial cells with necrosis and mineralization. Scar formation may develop. The endocardium is most frequently involved, with occasional involvement of the subepicardium.
- Myocardial necrosis may be evident as early as 3 days after the CNS damage and generally is present within 5 to 10 days in affected animals. Cardiac arrhythmias are the most frequent clinical sequelae.

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- The myocardial lesions probably occur secondary to increased sympathetic tone and release of catecholamines.

DIAGNOSIS

History and Physical Examination

- Clinical signs are usually referable to CNS disease and sometimes arrhythmias.

Electrocardiography

- Atrial and ventricular arrhythmias, ST-segment depression, prolonged QT interval, and T-wave abnormalities have been reported.
- The diagnosis is often made on necropsy.

THERAPY

- Appropriate therapy for CNS disease is advised. Treat arrhythmias as necessary.

GASTRIC DILATION-VOLVULUS COMPLEX

- Gastric dilation-volvulus complex (GDV) is a life-threatening emergency in the dog. It is most common in large, deep-chested dogs. No specific etiology has been determined, but GDV is often associated with exercise following a large meal. Cardiac arrhythmias occur in up to 40% of patients with GDV. Most arrhythmias are ventricular in origin. Atrial arrhythmias have also been reported. Arrhythmias usually occur within 36 hours of admission. The presence of arrhythmias does not worsen the prognosis.

CARDIAC PATHOPHYSIOLOGY

- The exact mechanism for the arrhythmias is unknown. Theories include acid-base imbalances, autonomic imbalances, myocardial hypoxia, electrolyte imbalances, or a myocardial depressant factor.

DIAGNOSIS

History and Physical Examination

- Clinical signs are restlessness, pacing, lethargy, weakness, attempts to vomit, and abdominal distention.
- The physical examination findings often include pale mucous membranes; rapid heart rate; weak femoral pulses; pain on abdominal palpation; distended, tympanic stomach; and signs associated with shock.

Electrocardiography

- The ECG may reveal ventricular and occasionally atrial arrhythmias. The most common appears to be an accelerated idioventricular rhythm (AIR). The terminology for this arrhythmia is still in dispute. Other terms for this ventricular rhythm are slow ventricular tachycardia, fast idioventricular rhythm, or idioventricular tachycardia.
- The features of AIR are a wide, bizarre complex rhythm, typically with a rate similar to the underlying sinus rate. As the sinus rate slows, the ventricular rate may "capture" the heart rhythm. Commonly, fusion beats are seen as the rhythm waxes between the sinus and the ventricular rhythm. As the underlying sinus rate slows, the ventricular rate may also slow (Fig. 13.12).
- ST-segment and T-wave changes may be evident.

Radiography

- The stomach is gas-filled, distended, and often rotated.
- The cardiac silhouette may be small owing to decreased venous return.

TREATMENT

- Treatment should be directed toward gastric decompression and shock therapy.
- Any arrhythmias should be treated as needed; however suppression of AIR is generally not necessary as long as blood pressure and perfusion are not affected by its presence.

PANCREATITIS

- Pancreatitis is a disease most frequently diagnosed in middle-aged, obese dogs and cats. Although many factors have been associated with pancreatitis, the cause is often unknown.

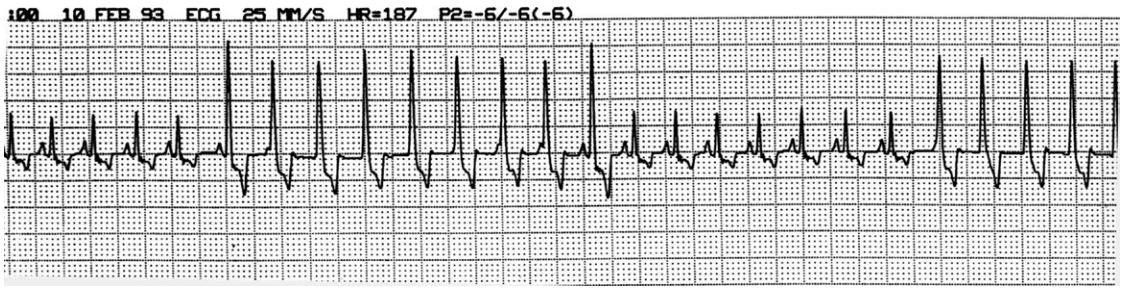


Fig. 13.12 Accelerated idioventricular rhythm. Beats 7 through 13 and the last four beats on the right are ventricular in origin. These beats represent an accelerated ventricular rhythm. Beats 6 and 14 are fusion beats, a hybrid of the sinus beat and the ventricular focus.

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CARDIAC PATHOPHYSIOLOGY

- A myocardial depressant factor is released from the pancreas, which can result in decreased myocardial contractility and cardiac arrhythmias.
- Activated pancreatic enzymes may also damage the myocardium directly or play a role in thrombus formation, resulting in myocardial ischemia.
- Acid-base and electrolyte imbalances may also contribute to arrhythmias.

DIAGNOSIS

History and Physical Examination

- Clinical signs are nonspecific and include vomiting, diarrhea, anorexia, and depression.
- Physical examination may reveal abdominal pain, fever, dehydration, and shock.
- Sudden death associated with cardiac complications can occur.

Electrocardiography

- Atrial and ventricular arrhythmias and ST-segment changes have been reported.

Radiography

- Abdominal radiographs may reveal an increased density, calcification, or gas in the area of the pancreas.

Clinical Pathology

- Leukocytosis, hyperglycemia, high liver enzymes, with high amylase and lipase in serum and peritoneal fluid are common findings in animals with pancreatitis.

THERAPY

- It is important to decrease pancreatic secretions by withholding food and water.
- Supportive care should include fluid and electrolyte replacement along with antibiotics.
- Cardiac arrhythmias should be treated as needed, although most arrhythmias will resolve spontaneously with resolution of the underlying disease.

TRAUMATIC MYOCARDITIS

- The term *traumatic myocarditis* is a catchall phrase for arrhythmias that may occur following blunt trauma. The arrhythmias seen after blunt trauma include supraventricular tachycardia, ventricular arrhythmias, or bradyarrhythmias. The trauma does not have to occur directly to the chest.

CARDIAC PATHOPHYSIOLOGY

- Blunt trauma mechanisms include the following: (1) unidirectional force, (2) bidirectional force (compressive), (3) indirect force, (4) force associated with deceleration, and (5) concussive force.
- The actual mechanism for the cause of the arrhythmias is not known. Direct damage to the heart from the trauma does not have to occur to result in the arrhythmias. Autonomic imbalance or reperfusion of ischemic tissue may have a role.

DIAGNOSIS

- Various arrhythmias, both atrial and ventricular, may be seen following trauma. The most common appears to be an AIR. The terminology for this arrhythmia is still in dispute. Other terms for this ventricular rhythm are slow ventricular tachycardia, fast idioventricular rhythm, or idioventricular tachycardia.
- The features of AIR are a wide, bizarre complex rhythm, typically with a rate similar to the underlying sinus rate. As the sinus rate slows, the ventricular rate may "capture" the heart rhythm. Commonly fusion beats are seen as the rhythm waxes between the sinus and the ventricular rhythm. As the underlying sinus rate slows, the ventricular rate may also slow (see Fig. 13.12).

THERAPY

- The main goal of therapy is supportive care for the underlying disease: fluid therapy, control of hemorrhage, oxygen therapy, analgesia, and correction of electrolyte imbalance.
- Therapy for the ventricular arrhythmias is necessary only if the tachycardia is persistent and the clinician feels it has a role in the patient's hypotension. Because the ventricular rate is often relatively normal, AIR does not necessitate antiarrhythmic treatment. If therapy is thought to be necessary, then lidocaine by IV route is recommended at 2 to 4 mg/kg as a bolus. The maximum dose to be given over a 10-minute period is 10 mg/kg. Lidocaine can cause nausea, vomiting, hypotension, and seizures. If this therapy suppresses the arrhythmias, then a constant-rate infusion of lidocaine can be given at 40 to 100 µg/kg/min. A repeat bolus of lidocaine may be needed once after starting the lidocaine infusion if more than 10 minutes elapse after injection of the original test bolus. It is not unusual for the constant-rate infusion of lidocaine to lose its effect after several hours.
- If lidocaine is not effective, then procainamide may be tried. The IV dose of procainamide is 2 mg/kg over 2 to 3 minutes. The total dose given over a 20-minute period should not exceed 15 mg/kg. The constant-rate infusion for procainamide is 25 to 50 µg/kg/min. Alternatively, oral, or intramuscular dosing could be initiated following the IV dose.
- Other therapies, such as other ventricular antiarrhythmics, beta-blockers, or magnesium (IV), have been suggested.

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PROGNOSIS

- Fortunately, the arrhythmias are generally not lethal. They tend to resolve over 3 to 5 days as the patient improves clinically. In general, the progression of underlying disorders, and not the arrhythmia, causes the patient's demise.
- If the patient requires antiarrhythmic therapy, it is usually discontinued before discharge or within several days of discharge.

Frequently Asked Questions

HOW USEFUL IS THE ECG IN DIAGNOSING ELECTROLYTE DISTURBANCES, AND WHAT CHANGES WOULD YOU EXPECT TO SEE?

The ECG can be helpful in raising suspicion regarding the presence of some electrolyte abnormalities, most notably hyperkalemia, but it is not a very sensitive test for detecting electrolyte abnormalities. One of the reasons that the ECG is not very sensitive is that the shape of the ECG complexes is influenced by several electrolytes (e.g., sodium, potassium, calcium) and further influenced by the patient's acid-base status. As potassium levels rise above normal, T waves become peaked with a narrow base, P-wave amplitude decreases, and the PR interval prolongs. If potassium is very high, QRS complexes widen, and P waves disappear (atrial standstill), and with further elevation in potassium, conduction delays lead to ventricular fibrillation and ventricular asystole. Hypokalemia produces prolonged QT intervals, U waves, and ST-segment depression, with possible ventricular and supraventricular arrhythmias. Hypocalcemia may cause QT interval prolongation, whereas hypercalcemia may shorten the QT segment, and with severe hypercalcemia, bradycardia may develop.

A PATIENT HAS A VENTRICULAR ARRHYTHMIA. WHAT SYSTEMIC DISTURBANCES SHOULD BE CONSIDERED?

VPCs may be seen with a variety of noncardiac conditions, including pancreatitis, GDV, traumatic myocarditis, splenic disease, neoplasia, cardiac masses, hypokalemia, trypanosomiasis, shock, heat stroke, and pyrexia. Drugs and plant toxins such as doxorubicin, methylxanthines (chocolate, theophylline), digoxin, sympathomimetics, and oleander can all cause VPCs.

A DOG HAS ECHOCARDIOGRAPHIC EVIDENCE OF MYOCARDIAL FAILURE BASED ON DEPRESSED INDICES OF SYSTOLIC FUNCTION (STROKE VOLUME, FRACTIONAL SHORTENING). WHAT NEEDS TO BE CONSIDERED IN ADDITION TO IDIOPATHIC DCM?

Systolic function can be depressed secondary to several systemic diseases, nutritional deficiencies, and toxic insults. Systemic abnormalities associated with depressed myocardial function include hypothyroidism, pancreatitis, sepsis and shock, and trypanosomiasis. Taurine and L-carnitine deficiencies can lead to DCM. Adriamycin toxicity leads to myocardial failure. Note that in some animals, especially large dog breeds, fractional shortening may be slightly below normal values established for smaller breeds and represent a normal variant.

A CAT HAS LEFT VENTRICULAR HYPERTROPHY ON THE ECHOCARDIOGRAM. WHAT SYSTEMIC DISTURBANCES NEED TO BE CONSIDERED ALONG WITH IDIOPATHIC HYPERTROPHIC CARDIOMYOPATHY?

Consider the possibility of systemic hypertension, acromegaly, hyperthyroidism, or infiltrative processes such as lymphoma. Dehydration causes a reduction in the size of the left ventricular lumen and as a result can give the impression of left ventricular hypertrophy.

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Chapter

14

Systemic Hypertension

Rebecca L. Stepien*

DEFINITIONS

Systemic hypertension (HT) is a sustained increase in blood pressure (BP). Systemic HT may be transient and occur when an animal is fearful or excited (situational hypertension), or sustained and pathologic (secondary hypertension and idiopathic hypertension).

- **Situational hypertension**: Transient increases in blood pressure associated with fear or excitement. Situational HT is not associated with any target organ damage and is diagnosed by repeat measurements in which values are within the normal range.
- **Secondary hypertension**: Sustained (repeatable) HT in association with an underlying disease triggering increased BP. Most sustained HT in dogs and cats is secondary HT, and target organ damage may be present.
- **Idiopathic hypertension**: Sustained (repeatable) HT that occurs in patients without detectable underlying disease present. The prevalence of idiopathic HT in dogs and cats is unknown. These patients may represent patients in the preclinical phases of causative diseases.

Although there is no widely accepted threshold for the diagnosis of systemic HT in dogs and cats, systemic HT is typically diagnosed whenever reliable BP measurements demonstrate a sustained elevation of systolic blood pressure (SBP) ≥160 mm Hg. Diastolic pressure measurement is not as reliably measured in dogs and cats, and current recommendations for diagnosis and therapy of systemic hypertension in these species are typically based on SBP values.

POPULATION AT RISK

CATS

- Chronic kidney disease (CKD), with or without proteinuria, and hyperthyroidism are the two most common causes of HT in cats. Although effective resolution of hyperthyroidism may reduce BP, occult CKD is suspected to be an underlying problem in some hyperthyroid cats and some of these cats will become overtly hypertensive after successful therapy for hyperthyroidism reveals underlying renal disease.
- Occasionally, hyperaldosteronism, pheochromocytoma, diabetes mellitus, or other endocrine diseases may also be associated with hypertension. In the case of diabetes mellitus, concurrent renal disease (commonly occurring in such patients) may contribute to hypertension.
- Many cats that are not hypertensive initially may become hypertensive when fluid therapy, steroids, erythropoietin, or vasoconstrictive drugs are given. In these cases, discontinuation of the causative therapy usually resolves the hypertension. Excessive fluid administration to a feline patient with undiagnosed HT may result in signs of congestive heart failure. Such patients may benefit from short-term therapy with a diuretic (furosemide 1–2 mg/kg q12h) until antihypertensive medications take effect to decrease SBP.
- Some clinicians are incorporating the routine measurement of BP into geriatric wellness examinations. This practice may occasionally identify a hypertensive cat with subclinical kidney disease; however, it should be realized that the risk of a false-positive result (i.e., high SBP measurement in a nonhypertensive patient) increases when the prevalence of a disease is low (e.g., in a clinically healthy population). Current American College of Veterinary Internal Medicine Hypertension Consensus Panel recommendations suggest adding screening BP assessment to the routine annual assessment of cats ≥9 years of age.
- BP measurement is advised in all cats with the following:
  - CKD
  - Endocrinopathies (e.g., hyperthyroidism, diabetes mellitus, hyperaldosteronemia)
  - Signs suggesting target organ damage (TOD) (tissue injury from persistently elevated BP)
    - Ocular signs (e.g., hemorrhage, hyphema, retinal detachment, retinal vessel tortuosity)
    - Neurologic signs (e.g., changes in mentation, nystagmus, head tilt, other cranial nerve signs, or seizures [focal facial or generalized])
    - New heart murmur or gallop heart sound
    - Radiographic cardiomegaly
    - Echocardiographically determined left ventricular hypertrophy (LVH)
      - LVH may not occur in all hypertensive cats.

*This chapter is a revision of the previous chapter by Scott A. Brown and Rosemary A. Henik.

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- Patients with LVH should have BP assessment to rule out HT before a diagnosis of hypertrophic cardiomyopathy is made.

DOGS

- CKD (i.e., chronic glomerular or tubulointerstitial disease associated with proteinuria) is most commonly associated with HT in dogs.
- Endocrine diseases, especially those involving pathologies of the adrenal glands (hyperadrenocorticism, pheochromocytoma, and hyperaldosteronism) and diabetes mellitus may be associated with HT.
- Increases in SBP have been linked to obesity and hypercholesterolemia, but it is unclear if obesity alone can cause HT.
- Resolution of the endocrinopathy may *not* restore normal BP in dogs.
- BP measurement is advised in any dog with the following:
  - CKD or proteinuria
  - Endocrinopathy
  - Ocular signs or neurologic signs of TOD as described previously
  - Echocardiographically determined concentric left ventricular hypertrophy

Key Point

CKD and endocrinopathies are commonly associated with systemic hypertension in cats and dogs.

CONSEQUENCES AND CLINICAL SIGNS OF HIGH BLOOD PRESSURE

OCULAR SIGNS

- Severe systemic hypertension can lead to ocular TOD. Findings associated with hypertensive injury include:
  - hemorrhage within the retina, vitreous, or anterior chamber
  - retinal detachment and atrophy
  - retinal edema; perivasculitis; retinal vessel tortuosity
  - glaucoma secondary to intraocular inflammation or hemorrhage
- Patients may exhibit photophobia.
- A sudden onset of blindness due to retinal hemorrhage and detachment is a common presenting complaint (Fig. 14.1).

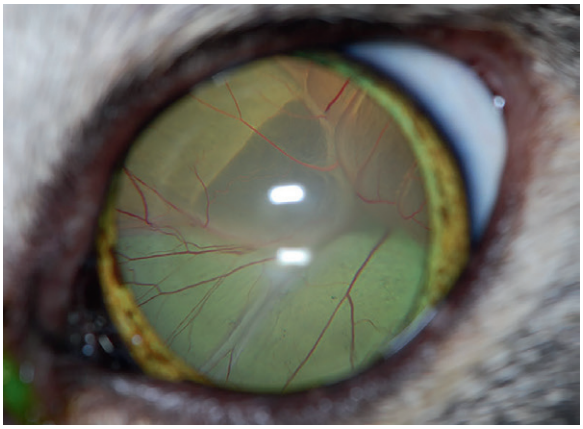


Fig. 14.1 Left eye of a 14-year-old cat with systolic blood pressure of 220 mm Hg. The detached retina and retinal vessels are visible behind the lens. Courtesy Dr. Ellison Bentley.

NEUROLOGIC SIGNS

- Signs consistent with cerebrovascular hemorrhage (head tilt, depression, ataxia, seizures) may be seen in cats and dogs with uncontrolled hypertension and are often associated with a poor prognosis.
- Cats with marked or sudden systemic HT (rapid rise in BP or SBP > 280 mm Hg) may have a syndrome of progressive stupor, head pressing, or seizures that rapidly resolves with effective antihypertensive therapy. This syndrome of hypertensive encephalopathy is due to cerebral edema caused by high intracapillary hydrostatic pressure that develops once the BP exceeds the autoregulatory range.
- Cats with chronic systemic hypertension may show signs of depression or decreases in activity that the owner may attribute to aging changes or be otherwise misinterpreted. When HT is diagnosed and effectively treated in these cats, rapid improvement in attitude and activity is often seen.

RENAL SIGNS

- Persistent elevation of SBP to values >160 mm Hg is associated with high risk of progressive renal injury in dogs and cats.
- Severity of renal injury in dogs has been correlated to the degree of BP elevation. Potentially pathologic renal changes induced by systemic HT include glomerular and tubulointerstitial changes. These changes can result in ischemia, necrosis, atrophy, and exacerbation of proteinuria. Gradual and additive changes may be difficult to quantify in living animals with preexisting kidney disease, but systolic HT at the time of presentation (>160 mm Hg) increases the odds of uremic crisis and death in dogs with CKD.
- Proteinuria is perhaps the best marker that high BP is injurious to the kidney. The presence of microalbuminuria or an elevated urine protein-to-creatinine ratio (>0.5 in dogs, >0.4 in cats) in a hypertensive animal (SBP ≥160 mm Hg) should be considered an indication of ongoing renal damage.

CARDIAC SIGNS

- Increased arterial pressure (i.e., afterload) increases left ventricular workload and may lead to systolic and diastolic dysfunction, left ventricular concentric hypertrophy, and secondary valvular insufficiency.

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Echocardiographic changes are highly variable and may be absent in hypertensive dogs and cats, but concentric hypertrophy may regress with antihypertensive treatment in affected animals.
- Cardiac murmurs or gallop heart sounds are common in patients with HT.
- Congestive heart failure secondary to systemic HT is rare but may occur in response to fluid therapy in a hypertensive patient.

MEASUREMENT OF BLOOD PRESSURE

PATIENT SELECTION

- Currently there is no evidence to suggest that BP should be measured in all animals.
- BP should be assessed in any patient with systemic disease known to be associated with hypertension, or in patients showing findings of target organ damage.
  - Animals with CKD, proteinuria, hyperadrenocorticism, hyperthyroidism, pheochromocytoma, hyperaldosteronism, or diabetes mellitus.
  - Animals with clinical signs attributable to high systemic arterial BP (TOD):
    - Blindness
    - Retinal hemorrhage, hyphema, or retinal detachment
    - Seizures, ataxia, mentation changes, or sudden collapse (signs compatible with cerebral vascular hemorrhage, intracranial edema, or stroke).
    - Decreased or altered mentation in cats, especially cats with CKD or hyperthyroidism
    - Proteinuria
    - Cardiac hypertrophy
    - Unexpected development of congestive heart failure signs after fluid therapy
  - Patients with marked obesity should be screened for hypertension as part of a metabolic diagnostic evaluation.

OVERVIEW OF MEASUREMENT METHODS

- BP may be measured by either direct or indirect methods. Direct BP measurement is the "gold standard," but it is technically difficult in unsedated dogs and cats and may be painful and associated with hematoma formation and other complications.
- Indirect techniques are more applicable to a veterinary clinical setting because they require less restraint and are technically easier to perform. They include the ultrasonic Doppler method (Doppler sphygmomanometry), oscillometric methods, and photoplethysmographic methods. High-definition oscillometry is a type of oscillometry that has recently been developed for veterinary use.
- All of the indirect techniques use an inflatable cuff wrapped around an extremity. The pressure in the cuff is measured with a manometer or pressure transducer. A squeeze bulb or automated device is used to inflate the cuff to a pressure in excess of SBP, thereby occluding the underlying artery. As the cuff is gradually deflated, changes in arterial flow are detected by one of several methods; the value for cuff pressure at various levels of deflation is then correlated with systolic, diastolic, or mean BP. The detection method varies among different indirect methods.
  - Doppler flow meters detect blood flow as a change in the frequency of reflected sound (Doppler shift) caused by the motion of underlying red blood cells. BP is read by the operator from an aneroid manometer connected to the occluding cuff placed proximal to the Doppler transducer.
  - Devices with the oscillometric technique detect pressure fluctuations produced in the occluding cuff resulting from the pressure pulse. Machines with the oscillometric technique generally determine systolic, diastolic, and mean BP as well as pulse rate.
  - Another device for measuring BP indirectly is the photoplethysmograph, which measures arterial volume by attenuation of infrared radiation and is designed for use on the human finger. It can be used in cats and small dogs that weigh less than 10 kg (22 lb).
- The ultrasonic Doppler and oscillometric methods have been well studied in conscious dogs and cats. In both species, the oscillometric devices tend to underestimate BP by increasing amounts as pressure increases. Another problem is the excessive time required to obtain readings in cats. The major limitation of the Doppler technique is the imprecise discrimination of the sounds designating the diastolic and therefore mean BP. The Doppler method may therefore be unreliable for the routine diagnosis and surveillance of patients with diastolic hypertension.
- In general, Doppler ultrasonic devices or high-definition oscillometric devices have been recommended for use in cats; either the oscillometric or the Doppler devices are recommended in dogs. For comparative purposes, the same device should be used each time in an individual animal.

Key Point

Doppler sphygmomanometry is the most commonly used method of BP measurement in unsedated cats; Doppler or oscillometric techniques can be used in dogs.

ENVIRONMENT AND PERSONNEL

- Provide an environment that is quiet and away from other animals.
- Owner presence may help calm the animal.
- Allow for a quiet acclimatization time for the animal of 5 to 15 minutes.
- The same individual (preferably a technician or nurse) should perform all BP measurements following a standard protocol.
- Diagnostic BP measurements can be obtained only in a patient that is unsedated, calm, minimally

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restrained, and motionless. A blanket on the floor or table may allow the patient to be more comfortable.
- Avoid measuring BP in panting dogs; panting can spuriously elevate BP readings.

CUFF SIZE AND PLACEMENT

- A complete range of pediatric cuff sizes should be available for an optimal patient limb circumference to cuff width ratio.
- For dogs, a cuff width that measures 40% of the circumference of the limb should be used; for cats, a width of 30% to 40% of the limb circumference should be used. The cuff width should be noted in the medical record and the same size cuff used for all future BP assessments.
- An oversized cuff may give erroneously low recordings; an undersized cuff may give erroneously high recordings. If the ideal cuff width is midway between two available sizes, the larger cuff should be used because it will theoretically produce the least error.
- The cuff may be placed around the appropriate limb at the level of the brachial, median, or dorsal metatarsal artery or around the tail at the site of the medial coccygeal artery. These cuff sites are relatively cylindrical, allowing the cuff to fit snugly. The "artery" arrow should be placed over the artery to position the inflatable bladder over the vessel, with the arrow pointing in either direction.
- In general, for the Doppler technique, the cuff is placed at the level of the median artery in the forelimb, and the transducer is placed between the carpal and the metacarpal pads (Fig. 14.2). Applying a small amount of alcohol and acoustic gel at the site of transducer placement may enhance the signal, but clipping the hair is usually not required.

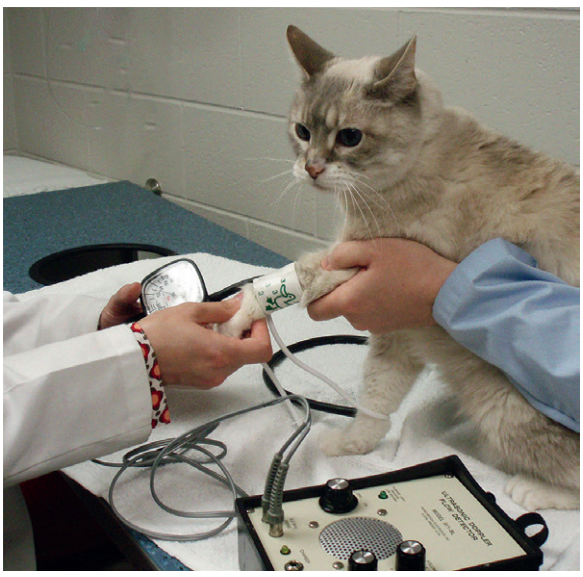


Fig. 14.2 Demonstration of the technique to obtain systemic blood pressure measurements with a Doppler transducer in a cat.

- In fractious cats, the oscillometric technique with the cuff at the tail head allows repeated readings without additional manipulation (Fig. 14.3).
- For the oscillometric technique, studies have demonstrated that the coccygeal (tail) artery, dorsal metatarsal artery, or median artery at the level of the radius in dogs all provide reliable values but values may not be consistent among sites within the patient. For comparative purposes, the same site for cuff placement should be used each time in an individual animal and recorded in the medical record (Fig. 14.4).
- During measurements, the extremity with the cuff should be positioned so that the cuff is at the level of the right atrium (heart base). Tail cuffs are at the correct level in lateral or sternal animals, but in a sitting animal, a cuffed forelimb may need to be raised to a "shake hands" position (see Fig. 14.2).

RECORD KEEPING

- A standard form for recording results of the BP measurement should be developed for the medical



Fig. 14.3 Use of the oscillometric technique to measure systemic blood pressure in a cat using a tail cuff. This technique allows minimal restraint to be used during measurements.



Fig. 14.4 Use of the oscillometric technique with cuff placement over the metatarsal artery in a dog. The oscillometric measurement method is most reliable in medium- and large-breed dogs.

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record. The following data should always be noted, along with each measurement obtained:
- Cuff size used (30%–40% of limb circumference in cats; 40% in dogs)
- Limb and cuff position used (including fore- or hind-, left, right, or tail)
- Time of day
- Time of administration of any antihypertensive medication(s)
- Technique used (i.e., Doppler or oscillometric)
- Degree of agitation, restlessness, panting, or limb movement
- Identity of technician performing measurement

- If a different room or unusual environmental conditions exist (e.g., a long wait before BP measurement with noisy dogs present), this should also be noted if higher-than-normal values are obtained.

TECHNIQUES

Doppler Sphygmomanometry

- For measurement using forelimb Doppler techniques in dogs or cats, cuff size is determined after the foreleg above the carpus is measured and the cuff is positioned so that the arrow on the cuff is over the artery, snugly wrapped around the leg, and secured by an adhesive strip or with a piece of tape.
- The patient should be comfortable, and the cuff position should be well tolerated with the cuff at or close to the level of the right atrium. An excellent audible blood flow signal can usually be detected by the Doppler flow meter from the median artery between the carpal and metacarpal pads by wetting down the hair with alcohol and applying coupling gel and the 10-MHz (i.e., pediatric) Doppler transducer.
- The Doppler transducer should be aligned parallel to blood flow so that the wire from the transducer is parallel with the limb (i.e., it emerges from under the paw). Unlike in the operative setting, the transducer is held in place, not taped.
- The Doppler cuff is inflated to a pressure 30 to 40 mm Hg higher than that required to obliterate the audible pulse, and then slowly deflated (about 2–5 mm Hg/s). A slower heart rate requires a slower deflation time to accurately determine systolic pressure. A too-slow deflation time, however, will result in discomfort to the animal.
- The first sound heard as blood begins to flow through the artery is read as the systolic pressure. The cuff should be completely deflated for several seconds before reinflation to allow for limb perfusion before the next measurement.
- Four to six measurements are advised over a 5- to 10-minute period because BP often falls with repeated measurements as the patient adjusts to the feel of the cuff inflating and deflating. The first measurement can be discarded as "range finding" and the average of three to five consecutive, consistent indirect measurements should be obtained. Any measurement obtained during limb movement should be discarded.
- If BP values are thought to be borderline or falsely high, the animal may be allowed to relax in a quiet room with or without the owner to acclimate to the environment and BP measurement repeated. In very anxious animals, a second measurement appointment can be made for reassessment.
- Sedation should be avoided in patients requiring blood pressure measurement to avoid altered results.

Oscillometry

- In dogs, the oscillometric method gives reliable systolic results, with the addition of diastolic and mean arterial pressures. In cats, BP measurements from high-definition oscillometry have been shown to be accurate, particularly for systolic BP. The rear limb is often preferred for the oscillometric technique in dogs, and the circumference of the metatarsus (i.e., below the hock) is measured for cuff width determination. In either species, use of a tail cuff may provide consistent results when limb manipulation is not tolerated.
- Medium- and large-breed dogs are positioned on a blanket on the floor with the technician or nurse, who snugly wraps the cuff with the cuff arrow over the artery, as described previously. Measurements should not be taken in standing animals.
- The oscillometric unit is activated, and the same number of readings is obtained as described above for Doppler measurements. If for any reason the oscillometric technique yields clearly spurious results (as in very small dogs), the Doppler method should be used. If cardiac arrhythmias are present, especially atrial fibrillation, oscillometric units may return an error message or not report BP accurately and Doppler techniques should be used.
- The average of all values obtained, or the average of all values obtained after the highest and lowest pressure readings are discarded, should be taken as the final value. Any clearly spurious BP value or any BP value for which the associated heart rate is incorrect is excluded. The results of each measurement should be recorded in addition to the session information mentioned previously in the section on record keeping.
- If results are borderline or inconsistent, repeat the measurement session later in the day or on another day.

LIMITING ANXIETY-INDUCED ARTIFACT (SITUATIONAL HYPERTENSION)

A clinic visit, hospitalization, a strange environment, restraint in the examination room, clipper noise and vibration, cuff placement, cuff inflation, and other unusual stimuli may induce anxiety in an animal

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during BP measurement. As a consequence, a falsely elevated value for BP may be obtained secondary to catecholamine release associated with this anxiety and is known as **situational hypertension** (previously called "the white coat effect"). The magnitude of this effect may be minimized by the following:

- Obtain BP measurements before a physical examination or other manipulations to which the animal may object (e.g., blood draws, imaging studies).
- Perform all measurements in a quiet room with a calm and reassuring manner.
- Allow the animal to acclimate to its surroundings for at least 5 minutes before obtaining BP measurements.

THERAPY OF SYSTEMIC HYPERTENSION

TREATMENT GUIDELINES

There is a clear association between marked systemic HT and TOD in dogs and cats, particularly for ocular and renal injury. However some of the mechanisms and thresholds of adverse effects of systemic HT are theorized on the basis of extrapolation from clinical studies in humans or from experimental studies in laboratory rodents.

- The American College of Veterinary Internal Medicine Hypertension Consensus Panel has recommended that treatment for systemic HT be based on reliable BP measurements and knowledge of TOD in an individual patient (Table 14.1).
- Antihypertensive treatment is indicated in any dog or cat with a sustained SBP >200 mm Hg, regardless of other clinical findings, due to the high risk of TOD.
- In both species, any animal with any level of HT (SBP > 160 mm Hg) and evidence of ongoing TOD should be considered a candidate for antihypertensive therapy.
- There is considerable uncertainty and difficulty associated with BP measurement in dogs and cats. Thus in animals in which the SBP is at the lower end of the hypertensive range (>160 mm Hg but less than 180 mm Hg) and no clinical abnormalities related to systemic hypertension are identified, the rationale for therapy is less clear. Currently, some clinicians recommend treatment for animals in this range and others do not. However, animals with this degree of HT that remained untreated should be monitored closely for increasing SBP and development of TOD that may indicate that HT is worsening and that severe HT has developed.
- Animals with no clinical signs and prehypertension should not be treated but should continue to be monitored over time (SBP and TOD screening).

Key Point

Animals with normal BP *or in which BP has not been measured* should not be treated with antihypertensive agents.

ANTIHYPERTENSIVE THERAPY

Overview

- Systemic arterial BP is the product of the cardiac output and the total peripheral vascular resistance; therefore antihypertensive therapy is generally aimed at reducing cardiac output, total peripheral vascular resistance, or both.
- Therapy may be loosely classified as dietary and pharmacologic.
- Treatment is generally conducted by sequential trials. In general, dosage adjustments or changes in treatment should be instituted no more frequently than every 2 weeks, unless extreme hypertension necessitating emergency treatment is present.
- When pharmacologic agents are used, a wide range of dosages should be considered, with initial dosages at the low end of the range.

Table 14.1 Staging of Systemic Hypertension in Dogs and Cats[a]

BP STAGE	NORMOTENSIVE	PREHYPERTENSIVE	HYPERTENSIVE	SEVERELY HYPERTENSIVE
RISK OF FUTURE TARGET ORGAN DAMAGE	**MINIMAL OR NO RISK**	**LOW RISK**	**MODERATE RISK**	**HIGH RISK**
Systolic BP (mm Hg)	<140	140–159	160–179	≥180
Treatment indicated?	No	No	+/–	Yes
Comment	• Monitor if high-risk disease is present	• Confirm with second measurement session • Monitor frequently if high-risk disease	• If TOD present: treat • If no TOD: – Confirm with second measurement session – Treat if HT confirmed	• If TOD present, treat immediately • SBP >200 mm Hg without TOD should be treated when confirmed with second measurement.

[a]Staging based on 2018 ACVIM Hypertension Consensus Panel guidelines.

BP, Blood pressure; *SBP*, systolic blood pressure; *TOD*, target organ damage. Evidence of TOD may include damage to the eyes (e.g., intraocular hemorrhage or retinal detachment), central nervous system (e.g., ataxia, mentation change, seizures, or otherwise unexplained neurologic signs), cardiovascular system (e.g., congestive heart failure), or kidneys (e.g., azotemia or proteinuria).

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- If an agent or combination of agents is incompletely effective, the dosage(s) may be increased, or additional agents may be added. Often, multiple agents are used concurrently, especially in dogs.
- In cats, apparent resistance to high doses of antihypertensive medications may indicate that situational HT is present, either alone or concurrently with true HT; careful attention to patient acclimation before confirmatory BP measurements may result in documentation of expected response to therapy.
- It may be difficult to restore BP to normal values when treating a hypertensive animal, but a reasonable clinical goal to minimize the risk of (additional) TOD is to lower the SBP to <140 mm Hg.

Duration of Treatment

- The diagnosis of HT associated with CKD necessitates lifelong antihypertensive treatment, with periodic dosage adjustments possible based on BP measurements.
- HT associated with hyperthyroidism or hyperadrenocorticism may resolve within 1 to 3 months after effective treatment of the underlying condition unless CKD is also present. Occasionally dogs with well-controlled hyperadrenocorticism remain hypertensive. Normotensive cats with hyperthyroidism may develop HT after hyperthyroidism is resolved; these cats likely have occult renal disease.
- In other patients, the duration of treatment cannot be predicted, but treatment may be lifelong to avoid or limit TOD. Periodic dosage adjustments based on BP measurements are indicated.

Dietary Therapy

- Though poorly studied, a low-sodium diet that provides <0.25% sodium on a dry-weight basis may be introduced. Dietary sodium restriction may be used as a first step if hypertension is mild (i.e., SBP < 170 mm Hg) and there is no TOD present.
- In some animals with HT, it may be more important to maintain adequate caloric intake rather than insist on a lower-sodium diet. Therefore drug therapy is instituted first, and when BP is stabilized, the animal may be switched to a lower-sodium diet.
- Obesity can elevate BP in humans and dogs and, perhaps, in cats. Consequently, weight loss (as well as metabolic screening) is desirable in obese, hypertensive animals.

Pharmacologic Agents

Overview of Antihypertensive Medications

- Recommendations for medical treatment of HT in dogs and cats have typically been extrapolated from human protocols, but recently, multicenter veterinary clinical trials have confirmed the efficacy of some antihypertensive medications.
- Vasodilating medications are considered first-line drugs for HT in veterinary patients.
- Current recommendations for medical therapy of HT in dogs and cats include the following:
  - Renin-angiotensin-aldosterone system (RAAS)–altering medications
    - Angiotensin-converting enzyme inhibitors (ACEI)
    - Angiotensin receptor blockers (ARBs), typically telmisartan
    - Aldosterone antagonists (e.g., spironolactone) may be used in conjunction with other antihypertensive medications to limit further RAAS activation.
  - Dihydropyridine calcium channel blockers (e.g., amlodipine)
  - Hydralazine
  - Phenoxybenzamine (α_1 antagonist) may be useful to treat HT associated with pheochromocytoma. In this situation, beta blockers are often added to therapy.
- Any of these agents may be given in concert with moderate dietary sodium restriction.
- In animals with systemic HT and CKD, ACEI or ARBs are the preferred initial choice in dogs, and amlodipine with ACEI or an ARB is the preferred initial choice in cats. Usually cats are treated with both amlodipine and an ACE inhibitor (or ARB) because amlodipine activates the renin-angiotensin-aldosterone system. In this setting, ACE inhibitors (or ARBs) are presumed to be renoprotective.

Vasodilators

- Angiotensin-converting enzyme inhibitors (ACEIs; e.g., enalapril or benazepril 0.5 mg/kg PO q12h in dogs and q24h in cats). In dogs, ACEI therapy usually results in an approximately 10% decrease in SBP. As a result, ACEI may be used as a single antihypertensive therapy for dogs with mild HT, especially when proteinuria is present. In cats, ACEI as a single agent does not typically provide sustained antihypertensive effects. In either species, ACEI may be used in conjunction with amlodipine therapy to limit RAAS activation that may occur with amlodipine use. Benazepril may be preferred in patients with CKD because it is eliminated by both urinary and biliary excretion (compared with only urinary excretion for enalapril). ACEI should not be given to dehydrated patients of either species.
- Angiotensin receptor blockers (ARBs; e.g., telmisartan)
  - Dogs: Telmisartan (1–2 mg/kg PO q24h) is an effective agent to lower BP and decrease proteinuria in dogs. Telmisartan may be used as a single agent in dogs with CKD or proteinuria or administered to patients receiving concurrent amlodipine, but close attention to follow-up BP and renal assessments are strongly recommended.

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Concurrent use of ACEI and telmisartan is not currently recommended due to lack of supportive data and unknown risk profile in dogs and cats.

- Cats: Telmisartan (2 mg/kg PO q24h) has been shown in two multicenter clinical trials to effectively lower SBP in cats by approximately 20 to 25 mm Hg on average and appears to have had a greater BP-lowering effect in cats with higher initial BP values. Notably, severely HT cats (initial BP > 200 mm Hg) were not included in either trial; the expected degree of response in these severely hypertensive cats remains unknown. Telmisartan is also effective for therapy of proteinuria in affected cats, and as is the case with ACEI, use of telmisartan should be avoided in dehydrated cats.

- Amlodipine besylate, a long-acting dihydropyridine calcium antagonist, reduces total peripheral resistance by its action on the smooth muscle of the vasculature.
  - Dogs: Amlodipine (0.2–0.5 mg/kg PO q24h) can be used to treat HT in dogs regardless of cause, but due to concerns over possible RAAS activation with amlodipine use, amlodipine use in dogs should be accompanied by ACEI or ARB administration. In either case, SBP and renal function should be monitored during therapy.
  - Cats: Amlodipine (0.625–1.25 mg PO q24h) has been used successfully as a single agent in hypertensive cats. Recent studies confirm clinical efficacy and suggest that the initial dose chosen for antihypertensive therapy in cats should be based on the initial SBP rather than body weight (initial SBP > 200 mm Hg: begin at 1.25 mg PO q24h, initial SBP <200 mm Hg: begin at 0.625 mg PO q24h). SBP reliably decreases significantly during amlodipine treatment, and significant adverse effects (i.e., hypotension, azotemia, hypokalemia, inappetence, and weight loss) are uncommon. Due to concern about RAAS activation when amlodipine is used as an antihypertensive, concurrent therapy with ACEI may be considered for cats receiving amlodipine.
- Hydralazine (0.5–2.0 mg/kg PO q12h), a direct-acting arterial dilator, can be added to an ACEI and amlodipine for refractory hypertension. Hydralazine acts quickly, resulting in a rapid decrease in pressure, and therefore may result in clinical signs of hypotension (e.g., tachycardia, ataxia, syncope, lethargy). Hydralazine should always be started at the lower end of the dosage range, especially in those already receiving other antihypertensive agents such as an ACE, ARB, or amlodipine. Because the RAAS is activated by hydralazine, coadministration of an ACEI (or ARB) and possibly spironolactone with hydralazine is appropriate.
- Phenoxybenzamine (0.25 mg/kg PO q12h in dogs or 2.5 mg/cat q12h in cats), an α_1 receptor antagonist, lowers BP by lowering peripheral vascular resistance. It is indicated for animals with pheochromocytoma and is given with a beta blocker to block the effects of catecholamines on the cardiovascular system.

Key Point

ACE inhibitors (or ARBs) are first-line drugs for hypertensive dogs, and amlodipine OR telmisartan are the first-line antihypertensive of choice in cats. Use of telmisartan as a single agent or addition of an ACE inhibitor to amlodipine is recommended for either species if CKD is present.

Other Medications

- **Spironolactone**, an aldosterone antagonist, is both a weak diuretic at higher doses (1–2 mg/kg PO q12h) and a neurohormone blocker at lower doses. It is a potassium-sparing diuretic and limits the effects of excess aldosterone (hypokalemia, HT) associated with hyperaldosteronism. Aldosterone levels are generally high with ACE inhibitor treatment (so-called aldosterone escape); therefore spironolactone may limit the renal and cardiac fibrosis as well as fluid retention associated with elevated aldosterone levels. Spironolactone does not have direct antihypertensive effects.
- Medications *no longer recommended* for chronic therapy of HT include diuretics or beta blockers.

Concurrent Therapies and Follow-Up Care

- In all animals treated for systemic hypertension, the routine examination should include a fundic examination, evaluation of any underlying diseases, and measurement of body weight, SBP, and serum concentrations of creatinine and electrolytes. The owner should be questioned for evidence of hypotension or drug toxicity, which may include lethargy, increased time spent sleeping, ataxia, or anorexia. Animals undergoing multiple drug regimens are more likely to exhibit adverse effects than those receiving a single antihypertensive agent.
- Therapy can be considered effective if follow-up SBP is ~120–140 mm Hg. If a medicated HT patient's SBP > 160 mm Hg, modifications of therapy should be considered.
- Once BP is controlled, the animal should be evaluated at 3-month intervals. A complete blood count, biochemical panel, and urinalysis should be evaluated at least once every 6 months.
- Many hypertensive animals have either overt or occult kidney disease. Other treatments for CKD should accompany antihypertensive therapy, as appropriate. Potassium supplementation is often needed in cats with CKD. Because animals with renal dysfunction generally have an impaired ability to adapt to sudden changes in sodium input, the

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administration of electrolyte solutions can lead to volume overload, worsened systemic HT, and pleural effusion (or peripheral edema) in animals with renal azotemia. Similarly, a sudden reduction in dietary sodium intake in an animal with renal azotemia can lead to extracellular fluid volume depletion. Some treatments, such as the administration of recombinant erythropoietin to elevate hematocrit, may exacerbate HT and should not be used until HT is controlled.

EMERGENCY MANAGEMENT OF SYSTEMIC HYPERTENSION

PATIENT SELECTION AND GOALS OF THERAPY

- Animals with neurologic signs or severe ocular manifestations of hypertension such as retinal detachment warrant aggressive treatment.
- The goal of emergency therapy is to incrementally reduce the SBP (~10% within the first hour, and an additional ~15% over the following few hours), rather than to immediately normalize the SBP.
- Any therapy for acute HT crisis should be accompanied by frequent reassessment of SBP patient condition (every 15 minutes for sodium nitroprusside and every 1–2 hours for oral medications) during therapy.

EMERGENCY THERAPY

- Sodium nitroprusside (1.0–10.0 μg/kg/min constant-rate intravenous infusion), an arterial (predominantly) and venous vasodilator acting as a donor of nitric oxide inside vascular smooth muscle cells, can be used for the initial treatment of hypertensive crisis. This drug must be given by constant-rate infusion, can be titrated very precisely according to BP response, and usually does not cause reflex tachycardia. Infusions of sodium nitroprusside should always be started at the low end of the dosing range and incrementally increased if decreases in SBP are not seen within 5 to 10 minutes. Sodium nitroprusside should *never* be administered as an intravenous bolus. Sodium nitroprusside solutions must be protected from prolonged exposure to light.
- If a constant-rate infusion is not possible, hydralazine can be given orally and usually shows initial effects within 2 hours. If the response is inadequate at 2 hours, the hydralazine dose can be repeated to uptitrate the effect.
- If intensive monitoring is not available in a veterinary hospital, oral amlodipine is an excellent alternative choice, especially in cats.
- Regardless of initial therapy chosen for the management of an acute hypertensive crisis, a drug of choice for long-term management of systemic hypertension should be instituted soon after presentation to facilitate the eventual transition to long-term maintenance therapy.

Frequently Asked Questions

1. BECAUSE DOGS ARE OFTEN MORE REFRACTORY TO THE EFFECTS OF ANTIHYPERTENSIVE TREATMENT THAN CATS, WHAT IS A REASONABLE STEPWISE APPROACH TO ANTIHYPERTENSIVE THERAPY IN DOGS?

- Whenever resistant (or refractory) HT is suspected, medication compliance and accurate SBP measurement should be rechecked and addressed.
- Hypertensive dogs (i.e., those with a sustained SBP > 160 mm Hg) are often proteinuric, given the underlying diseases associated with systemic hypertension in that species.
  - ACE inhibitors (e.g., enalapril, benazepril) have been shown to decrease proteinuria and cause balanced vasodilation; they are usually the starting drug in the treatment of canine hypertension. Enalapril or benazepril (0.5 mg/kg PO q12–24h) is initiated and SBP is measured after 2 weeks of treatment. Alternatively, telmisartan (an ARB) can used as a first agent; telmisartan may have more effective antihypertensive effects than ACEI as a sole agent (telmisartan 1–2 mg/kg PO q24h).
  - If hypertension is still present, amlodipine (0.2–0.4 mg/kg PO q24h) can be administered with the ACEI or ARB.
  - The combination of an ACEI or ARB and amlodipine is often effective, but if HT is sustained on these medications, maximizing the amlodipine dose or adding an additional medication having a different mechanism of action may be effective.
  - Hydralazine, a direct-acting arterial vasodilator, can be given at a starting dose of 0.5 mg/kg PO q12h, and slowly uptitrated in increments of 0.5 mg/kg to a maximum dose of 2.0 mg/kg. BP and serum creatinine should be monitored with each increase in dose.
  - Regardless of therapeutic approach, increased levels of aldosterone should be expected, and the addition of spironolactone (1–2 mg/kg PO q12h) is advised to limit the effects of aldosterone.

2. CATS AND DOGS WITH CKD OFTEN EXHIBIT SYSTEMIC HYPERTENSION. WHAT IS THE CAUSATIVE FACTOR IN THIS RELATIONSHIP, AND HOW SHOULD THESE ANIMALS BE MANAGED? HOW CAN YOU TELL IF HYPERTENSION IS DAMAGING THE KIDNEY?

This has often been referred to as a chicken-and-egg question, but it is more properly seen as an example of a complex positive feedback loop that complicates therapy in animals affected with both problems. High BP produces barotrauma within the microvasculature of the kidney, effectively destroying renal tissue over time (weeks to months). On the other hand, CKD produces abnormalities in body fluid volumes and can alter neurohumoral control of BP. These factors combine to make HT relatively common (approximately 25% prevalence) in dogs and cats with CKD. Furthermore, it is still generally accepted that something has to be wrong with the kidney for sustained systemic hypertension to be present.

- In the short term, high BP tends to improve glomerular filtration rate. This is why the level of azotemia should always be assessed shortly (3–14 days) after any changes in antihypertensive therapy to detect any increased azotemia secondary to the response to decreasing SBP.

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- Vasodilators with intrarenal effects (i.e., ACEI and ARBs) are often preferred for antihypertensive therapy in animals with kidney disease, largely because the vasodilatory effect may help to preserve renal function.
- Typical agents to select for initial therapy when both hypertension and renal azotemia are present in dogs are an ACEI or ARB (i.e., enalapril or benazepril, or telmisartan). In cats with CKD, telmisartan is preferred. Alternatively, amlodipine with RAAS blockade (ACEI or spironolactone) can be used.
- Perhaps the best index of hypertensive damage of the kidney is proteinuria. The presence of microalbuminuria or an elevated urine protein-to-creatinine ratio (>0.5 in dogs, >0.4 in cats) is generally an indication for the use of an ACEI (e.g., enalapril or benazepril) or an ARB (e.g., telmisartan).

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Chapter 15

Pathophysiology and Therapy of Heart Failure

Darcy Adin*

DEFINITIONS

- *Preload* is the degree of muscle fiber stretch just before contraction. This correlates to the volume of blood within the ventricle just before contraction.
- *Afterload* is the load against which a muscle exerts its contractile force. Cardiac afterload refers to the resistance that the ventricle must overcome to eject blood.
- *Heart disease* is any structural (microscopic or macroscopic) abnormality of the heart that may or may not result in heart failure.
- *Occult heart disease* refers to disease that is clinically silent but detectable with imaging (myocardial dysfunction, chamber dilation, arrhythmias). This description is usually used to describe patients with cardiomyopathy (e.g., Doberman Pinschers with dilated cardiomyopathy [DCM]).
- *Heart failure* is the pathophysiologic state that occurs when the heart is unable to function at a level commensurate with the requirements of the metabolizing tissues, or can only do so at elevated filling pressures.
- *Congestive heart failure (CHF)* is the accumulation of fluid in tissues associated with increased capillary hydrostatic pressures and elevated diastolic intracardiac pressures.
- *Low-output failure* can be defined as poor cardiac output, resulting in reduced tissue perfusion and inadequate tissue oxygenation. In general, the term low-output failure is used to describe clinical scenarios in which cardiac output is dramatically reduced by end-stage, primary, systolic dysfunction.
- *Right-sided heart failure* occurs when valve regurgitation, pericardial disease, outflow tract obstruction, or pulmonary hypertension cause elevated filling pressures in the right atrium or right ventricle. Volume and pressure overloading of the right ventricle can cause hepatic congestion accompanied by ascites, pleural effusion, and, rarely, peripheral subcutaneous edema. These diseases also reduce the forward flow of blood into the pulmonary circulation and the left heart, resulting in reduced stroke volume, cardiac output, and, possibly, signs of low-output heart failure. Pleural effusion in dogs is a manifestation of right-sided or biventricular heart failure, but cats can develop pleural effusion with pure left-sided heart failure apparently because the visceral pleural lymphatics drain into the pulmonary venous circulation.
- *Left-sided heart failure* occurs when valve regurgitation, impaired contractility, or diastolic dysfunction cause elevated filling pressures in the left atrium or left ventricle. These elevated filling pressures are transmitted to the pulmonary venous and capillary beds which can lead to fluid exudation into the interstitial and alveolar spaces (pulmonary edema). Pleural effusion in cats can be a manifestation of left-sided heart failure.
- *Heart disease staging* describes the characterization of patients with heart disease according to disease severity, so as to provide guidelines for stage-specific evidence-based treatments. This staging system is outlined in the 2019 American College of Veterinary Internal Medicine (ACVIM) consensus guidelines for diagnosis and treatment of myxomatous mitral valve disease in dogs.
  - **Stage A**: Predisposed to heart disease
  - **Stage B**: Heart disease is present without clinical signs
    - Stage B1: Heart disease is present with no evidence of cardiac remodeling
    - Stage B2: Heart disease is present with evidence of cardiac remodeling (chamber enlargement)
  - **Stage C**: Heart disease is present along with clinical signs of CHF. This stage can be divided into acute CHF and chronic CHF.
  - **Stage D**: Heart disease is present along with clinical signs of CHF which are refractory to standard CHF therapies. This stage can be divided into acute refractory CHF and chronic refractory CHF.

*For this edition, Darcy Adin updated this chapter which was previously written by Keith N. Strickland.

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Key Point

CHF occurs when cardiac filling pressures result in elevated venous and capillary hydrostatic pressures, with subsequent edema formation. Medical therapy for CHF is directed toward reducing filling pressures so that edema fluid can be mobilized through Starling's forces, suppressing activated neuroendocrine systems, and improving cardiac output.

HEART FAILURE PATHOPHYSIOLOGY

OVERVIEW

- The function of the cardiovascular system is to maintain normal arterial blood pressure and flow (cardiac output) while maintaining normal venous and capillary pressures during rest and exercise. A normally functioning cardiovascular system provides adequate blood flow to vital tissues (such as the brain, the heart, and the kidneys) for oxygen and nutrient delivery and removal of metabolic waste products.
- Heart failure as a result of advanced cardiac disease impairs the normal functions of the cardiovascular system, and if severe, can lead to life-threatening impairment of tissue perfusion and the inability to maintain normal venous and capillary pressures.
- The pathophysiology of heart failure is complex. Progressive heart disease is not simply explained by cardiac output reductions and it is becoming increasingly evident that neurohormonal, catecholamine, and inflammatory cascades participate in the decline of cardiac function. Additionally, the close relationship between renal dysfunction and cardiac dysfunction highlights the importance of these cascades to disease progression (cardiovascular renal disorders). The current model of heart failure pathophysiology embodies the idea that cardiac damage or dysfunction results in altered hemodynamics, leading to neurohumoral and sympathetic nervous system (SNS) activation to restore cardiac function and tissue perfusion through vasoconstriction and volume expansion. Chronic activation of these compensatory mechanisms, however, is maladaptive and contributes to progressive cardiovascular dysfunction, culminating in life-threatening CHF, low-output failure, or sudden death.
- Myocardial abnormalities that lead to the clinical syndrome of heart failure can be subdivided into systolic and diastolic dysfunction. Systolic function is determined by myocardial contractility, valvular competence, preload, afterload, and heart rate. Systolic dysfunction caused by abnormalities of any of these components impacts the ability of the ventricles to pump blood forward. Diastolic dysfunction occurs when either ventricular relaxation or compliance are impaired, resulting in higher filling pressures and low or normal filling volume. Although some diseases are predominantly characterized by one or the other, for many cardiac conditions both systolic and diastolic dysfunction contribute to clinical manifestations of heart failure.

ETIOLOGIES

Heart disease that is severe enough to lead to heart failure can be caused by a variety of abnormalities broadly categorized as myocardial failure (systolic dysfunction), diastolic dysfunction, preload abnormalities, and afterload abnormalities. Extracardiac diseases can also adversely affect the heart and contribute to or cause heart failure.

- **Myocardial failure (systolic dysfunction):**
  - Myocardial failure causes systolic dysfunction and activation of compensatory mechanisms, which increase sodium and water retention to increase venous return to the heart. This increased preload contributes to a larger end-diastolic dimension that helps to maintain a relatively normal total stroke volume (TSV) in the early stages. As the myocardial function deteriorates (as noted by a decrease in fractional shortening and ejection fraction), the ventricles continue to enlarge so that the TSV is maintained, even though the percentage of blood being ejected is decreasing. Severe left ventricular dilation causes distortion and dilation of the mitral valve annulus, resulting in functional mitral regurgitation and further volume overloading of the left ventricle. Once the compensatory limits are reached, further increases in blood volume (associated with sodium and water retention) and venous return cause an increase in cardiac filling pressures, with threat of CHF development.
  - Impaired myocardial contractility can occur with primary heart disease (e.g., idiopathic DCM) or secondary heart disease, which leads to DCM phenotype characterized by systolic dysfunction.
  - Primary DCM appears to be genetic in some breeds such as in Doberman Pinschers where mutations in the PDK4 and TTN genes are associated with disease. Other breed predispositions support familial and likely genetic causes in those breeds even though genetic mutations have not been discovered.
  - Some examples of conditions leading to secondary systolic dysfunction include:
    - Chronic volume overload (e.g., from mitral or tricuspid regurgitation, left-to-right shunts such as patent ductus arteriosus or ventricular septal defect, or severe, chronic anemia).
    - Nutritional deficiencies such as taurine deficiency in cats and some breeds of dogs (e.g., American Cocker Spaniels, Golden Retrievers, Dalmatians, Boxers, Welsh Corgis, Newfoundlands) and L-carnitine deficiency in Boxers. High-pulse diets appear to be

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associated with a DCM phenotype in some dogs.
- Metabolic diseases including feline hyperthyroidism, canine hypothyroidism, and chronic uremia.
- Some chemotherapy drugs (e.g., doxorubicin).
- Cardiac infiltration by neoplasia (e.g., lymphosarcoma) or amyloidosis.
- Chronic and sustained tachycardias.
- Myocarditis from a variety of infectious agents such as *Bartonella* spp, *Trypanosoma cruzi*, amongst others.

- **Volume overload** (excessive preload)
  - Chronic volume overload caused by valve regurgitation (especially mitral) is a common cause of heart failure in veterinary medicine. Incompetency of an atrioventricular valve resulting from myxomatous degeneration, endocarditis, or congenital malformation, allows retrograde ejection (regurgitation) of blood into the corresponding atrium during systole, concurrently reducing forward flow and decreasing cardiac output. Neurohormonal activation is an appropriate response to the reduction in cardiac output and leads to sodium and water retention and vasoconstriction. Volume retention and venoconstriction increase preload, which increases left ventricular diastolic size and total stroke volume to restore forward flow. The increased preload and severe regurgitation increase atrial and ventricular filling pressures, which can lead to CHF.
  - Valve regurgitation can be primary (degenerative, endocarditis, congenital) or secondary (functional) when it is associated with other conditions that alter valvular function such as ventricular hypertrophy, ventricular dilation (e.g., DCM), or ischemia.
  - Left-to-right shunts such as patent ductus arteriosus, ventricular septal defects, and arteriovenous fistulas cause volume overload of the cardiac chambers and blood vessels that are accommodating shunted blood flow. For example, dogs with patent ductus arteriosus develop enlarged left atrium and left ventricular chambers because shunted blood goes from the aorta into the pulmonary artery and then returns to the left side of the heart, bypassing the systemic circulation and the right side of the heart.
- **Excessive afterload** (pressure overload)
  - An abrupt increase in afterload causes a positive inotropic effect, a physiologic phenomenon known as the Anrep effect. However, when high afterload is severe or chronic, it depresses myocardial contractility and reduces the rate of ejection, reduces the amount of blood ejected for a given preload, and increases myocardial oxygen consumption which risks ischemic damage.
  - Pulmonary hypertension, systemic hypertension, and ventricular outflow obstructions (e.g., aortic or pulmonic stenosis) are examples of clinically significant causes of increased afterload.
  - Lesions that cause increased intraventricular systolic pressures usually do not cause CHF because the heart is generally able to compensate for the increased intraventricular systolic pressures with concentric hypertrophy (Laplace's law), and the ventricular systolic function remains relatively normal. When the obstruction to flow is severe (critical stenosis) or acute, however, pressure overload can result in myocardial failure and heart failure. More commonly, CHF occurs when there is a concurrent valvular insufficiency (e.g., pulmonic stenosis with tricuspid regurgitation, subvalvular aortic stenosis with mitral regurgitation or aortic regurgitation, pulmonary hypertension with tricuspid regurgitation).
  - Severe pressure overload can also predispose to sudden death because concentric ventricular hypertrophy impairs coronary perfusion, leading to regional hypoxia and a propensity towards arrhythmia formation.
- **Inadequate preload**
  - Adequate preload is necessary for adequate cardiac output. Preload reduction in some clinical situations (e.g., pericardial effusion or hemorrhage) can be a primary reason for decreased stroke volume and cardiac output.
  - In the case of pericardial effusion, the fluid in the pericardial sac mechanically limits ventricular filling and prevents compensatory mechanisms (such as volume expansion) that would normally restore ventricular end-diastolic volume. Elevation of intrapericardial pressure, progressive limitation of ventricular diastolic filling, and reduction of stroke volume and cardiac output characterize cardiac tamponade associated with clinically significant pericardial effusion and cause right-sided heart failure and low-output failure signs. The clinical course depends on the size and rate of accumulation of effusion and the compliance of the pericardial sac.
  - Acute tamponade is usually associated with hemorrhage from a neoplastic lesion, such as hemangiosarcoma, but it may also be associated with rupture of the left atrium secondary to severe mitral regurgitation. As the effusion accumulates, intrapericardial pressure increases and eventually exceeds the diastolic pressures in the right atrium and ventricle, thereby restricting venous inflow to the right heart. The restriction of venous return reduces right ventricular cardiac output, pulmonary blood flow, and left-sided venous return, leading to reduced left ventricular stroke volume and cardiac output with signs of low-output heart failure.

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- Chronic tamponade associated with the slow accumulation of pericardial effusion differs from acute tamponade in that the pericardium can stretch and there is time for sodium and water retention to compensate for the impediment of cardiac inflow, secondary reduction in stroke volume, and cardiac output. Although compensation may somewhat normalize cardiac output, signs of right-sided CHF are often present because of elevated filling pressures (jugular distention and ascites).

- **Diastolic dysfunction**
  - Diastolic dysfunction of the ventricles can lead to heart failure as a result of relaxation abnormalities, poor elasticity, or poor compliance and is characterized by elevated filling pressures in the absence of significant valve regurgitation, chamber enlargement, or systolic dysfunction.
  - Diastolic dysfunction increases ventricular end-diastolic pressure, which is transmitted to the corresponding atrium and the venous system. Elevations of venous and capillary pressures promote interstitial and alveolar pulmonary edema or ascites through hydrostatic factors.
  - Ventricular relaxation is abnormal in hypertrophic cardiomyopathy (HCM) because of altered myocardial architecture and energetics. The increase in ventricular or septal wall thickness is associated with decreased ventricular compliance (increased ventricular stiffness). In HCM, diastolic dysfunction typically progresses from abnormal relaxation to decreased ventricular compliance secondary to ventricular hypertrophy and fibrosis. Ischemia is also associated with ventricular relaxation abnormalities.
  - A reduction in ventricular compliance is characterized by increased stiffness of the ventricles. This change can be associated with:
    - Volume overloading and increased filling pressures
    - Increased muscle mass or wall thickness (e.g., primary or secondary concentric hypertrophy)
    - Decreased ventricular distensibility (usually associated with extrinsic compression of the heart)
    - Myocardial or endocardial fibrosis (e.g., restrictive cardiomyopathy, ischemic heart disease)
    - Noncardiac conditions such as systemic hypertension and anemia can adversely affect the heart and accelerate heart disease. For example, systemic hypertension can worsen mitral regurgitation caused by degenerative valve disease by increasing the resistance to blood exiting the left ventricle through the aorta. This results in more mitral valve regurgitation because the relatively lower left atrial pressure is the path of least resistance when the mitral valve does not normally coapt.

COMPENSATORY MECHANISMS

Most compensatory mechanisms require days to weeks to become fully activated (with the exception of the immediately activated SNS), which might be insufficient for acute and severe cardiac decompensation. Dogs with acquired heart disease, however, often show slow-but-progressive cardiac dysfunction that worsens over months to years. Compensatory mechanisms are appropriately activated during this subclinical period which can normalize cardiac output for a period of time. At some point, however, the compensatory mechanisms are overwhelmed, leading to acute decompensation. Several counterbalancing mechanisms are also activated, such as production of natriuretic peptides and alternative renin-angiotensin-aldosterone system (RAAS) metabolites; however, these effects are often overwhelmed by classical compensatory mechanisms promoting vasoconstriction and volume retention, especially when the underlying heart disease continues unchecked.

- **Frank-Starling mechanism**
  - Venous return and fluid status determine the preload of the ventricle. The Frank-Starling mechanism describes cardiac performance enhancement that occurs with increased preload. Physiologic increases in ventricular end-diastolic volume increase myocardial fiber length and allow the sarcomere to function near the upper limit of its maximal length (which is its optimal length), where it is able to generate the maximal amount of force during contraction.
  - To better understand the role of the Frank-Starling mechanism, consider the hemodynamic changes associated with exercise in the absence of heart failure. Cardiac output is increased during exercise through the following mechanisms: (1) increased heart rate and contractility through increased SNS activity, (2) increased venous return (preload) with a more vigorous contraction (Frank-Starling mechanism), and (3) reduced afterload associated with reduced peripheral vascular resistance.
  - In the failing heart, cardiac output and ventricular performance can be maintained within normal limits at rest only because the ventricular end-diastolic fiber length and the preload are already elevated; more preload cannot be recruited without pathologically elevating filling pressures. The factors that normally help increase cardiac output during exercise are already chronically activated, resulting in increased preload and ventricular end-diastolic pressures (especially in a noncompliant, dilated ventricle), with the threat of edema formation. The failing heart operates along the flat portion of the ventricular performance curve, where increases in ventricular volume and diastolic pressure do not increase ventricular performance.

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- **Renin-angiotensin-aldosterone system**
  - The RAAS is a complex neurohormonal compensatory system that functions to maintain blood pressure and tissue perfusion when cardiac output is reduced. Advanced heart disease and some therapies can stimulate renin release through reduced renal perfusion, decreased salt delivery to the macula densa, and SNS stimulation of beta-1 adrenoceptors in the juxtaglomerular apparatus of the kidney, thereby initiating the cascade of RAAS activation (Fig. 15.1). Angiotensin II is the major metabolite of this cascade, but other metabolites also contribute to classical effects of vasoconstriction and sodium and water retention. In addition to vasoconstriction, angiotensin II activates the SNS, increases synthesis and release of aldosterone from the zona glomerulosa of the adrenal cortex, causes the nonosmotic release of antidiuretic hormone, increases thirst, and promotes inflammation. It also stimulates the release of growth factors that promote remodeling of the vessels and myocardium. Vascular remodeling (smooth muscle cell growth and hyperplasia, hypertrophy, and apoptosis; cytokine activation; myocyte and vascular wall fibrosis) results in decreased vascular responsiveness to alterations in blood flow, decreased vascular compliance, and increased afterload. These changes lead to pathologic ventricular hypertrophy and exert cytotoxic effects, resulting in myocardial necrosis and loss of myocardial contractile mass, with subsequent cardiac dysfunction. Aldosterone causes sodium retention in the distal renal tubules to promote fluid retention. Aldosterone also promotes fibrosis of the myocardium, kidneys, and vascular smooth muscle contributing to cardiovascular and renal dysfunction. The RAAS cascade can be initiated within the circulation or within the tissues where it can have local effects.
  - RAAS activation is an effective and beneficial way to improve cardiac output in the short term, however, if the stimulus continues, RAAS activation becomes chronic and dysregulated and can contribute to disease progression. Like most physiologic systems, counterbalancing metabolites are also produced that effect natriuresis and vasodilation; however, with sustained cardiac dysfunction, these actions are outweighed by classical effects of sodium retention and vasoconstriction. Therapeutics that modulate the RAAS in such a way as to dampen the maladaptive, classical effects while enhancing the beneficial, alternative effects are an important part of the treatment regime for CHF.
  - Chronic RAAS activation is not a consistent feature of the early, preclinical stages of heart disease but it is clearly activated in CHF either due to the

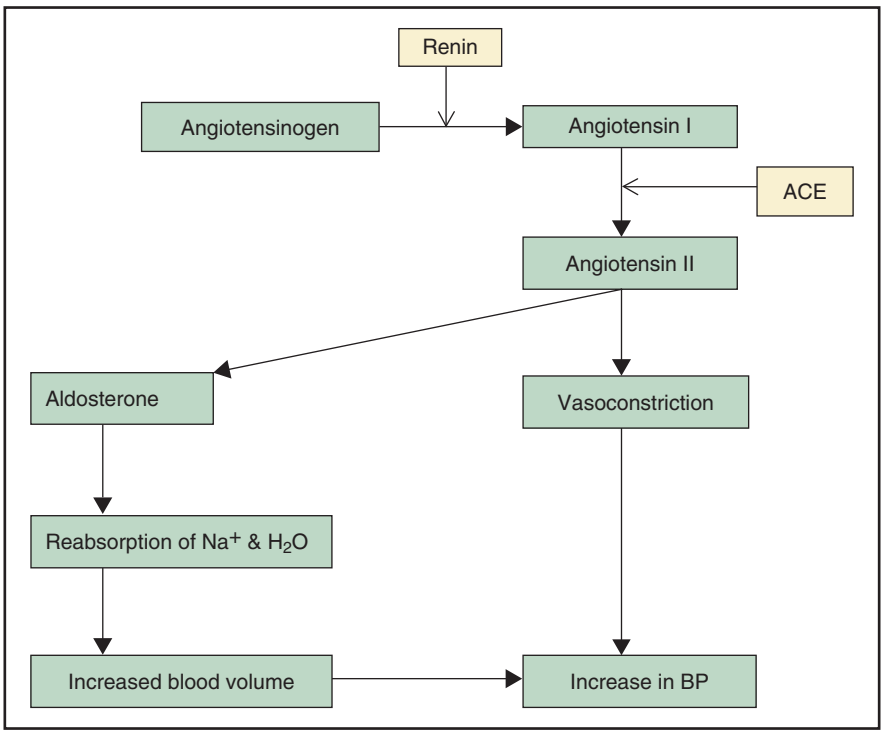


Fig. 15.1 Renin-angiotensin-aldosterone system (RAAS) cascade. Reduced renal perfusion, reduced salt delivery to the macula densa, and sympathetic nervous system stimulation are the primary stimuli for renin release, which leads to activation of the angiotensin-aldosterone system. RAAS activation results in sodium and water retention and peripheral vasoconstriction. *ACE*, Angiotensin-converting enzyme; *BP*, blood pressure; *RAAS*, renin-angiotensin-aldosterone system. (Used with permission of Dr. Walt Ingwersen, Ontario, Canada.)

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advanced disease, the use of RAAS-activating medications (e.g., diuretics) or both.

- **Sympathetic nervous system**
  - The autonomic nervous system plays a crucial role in the compensation of heart failure. The activity of the SNS is increased in part by baroreflex-mediated parasympathetic withdrawal, as well as by sympathetic activation by the RAAS. Early activation of the SNS helps to maintain cardiac output, blood pressure, and tissue perfusion by increasing venous return to the heart (through splanchnic vasoconstriction), RAAS activation, vasoconstriction of other vascular beds, and positive inotropic and chronotropic cardiac effects. Activation of the SNS early in heart failure is beneficial, but it then becomes maladaptive when chronically activated (Fig. 15.2).
  - Chronic activation of the SNS is associated with elevated levels of plasma norepinephrine (NE), cardiac NE depletion, downregulation and desensitization of beta-1 adrenergic receptors, and abnormal baroreflex function. Plasma NE levels appear to be increased because of a combination of increased release of NE from adrenergic nerve endings and reduced uptake by adrenergic nerve endings. The depletion of myocardial NE (serum NE levels increase and myocardial levels decrease) probably represents the depletion of the neurotransmitter in adrenergic nerve endings.
  - The downregulation of beta-1 adrenergic receptors occurs relatively soon (24–72 hours) after the initial SNS activation, making it progressively more difficult for the SNS to counter impaired contractility.
  - Chronic activation of the SNS also overloads the heart by increasing venous return to a heart that is already volume overloaded, increasing myocardial oxygen consumption by increasing heart rate and volume overloading, and damaging the myocardium, creating a substrate for arrhythmogenesis.
- **Myocardial hypertrophy**
  - Myocardial hypertrophy is a compensatory mechanism directed toward normalizing cardiac output, wall tension, and filling pressures. The relationship between wall thickness, pressure, and volume is evident from LaPlace's law, which states that wall stress = (pressure × radius)/wall thickness. Either high intracardiac pressure or volume will increase wall stress, and increasing wall thickness can help to normalize it. However, the type of hemodynamic load imposed on the heart (either volume or pressure) determines the type of hypertrophy (eccentric or concentric, respectively).
  - Eccentric myocardial hypertrophy (or volume-overload hypertrophy) is characterized by chamber dilation with normal wall thicknesses, and is a response to the increase in blood volume and

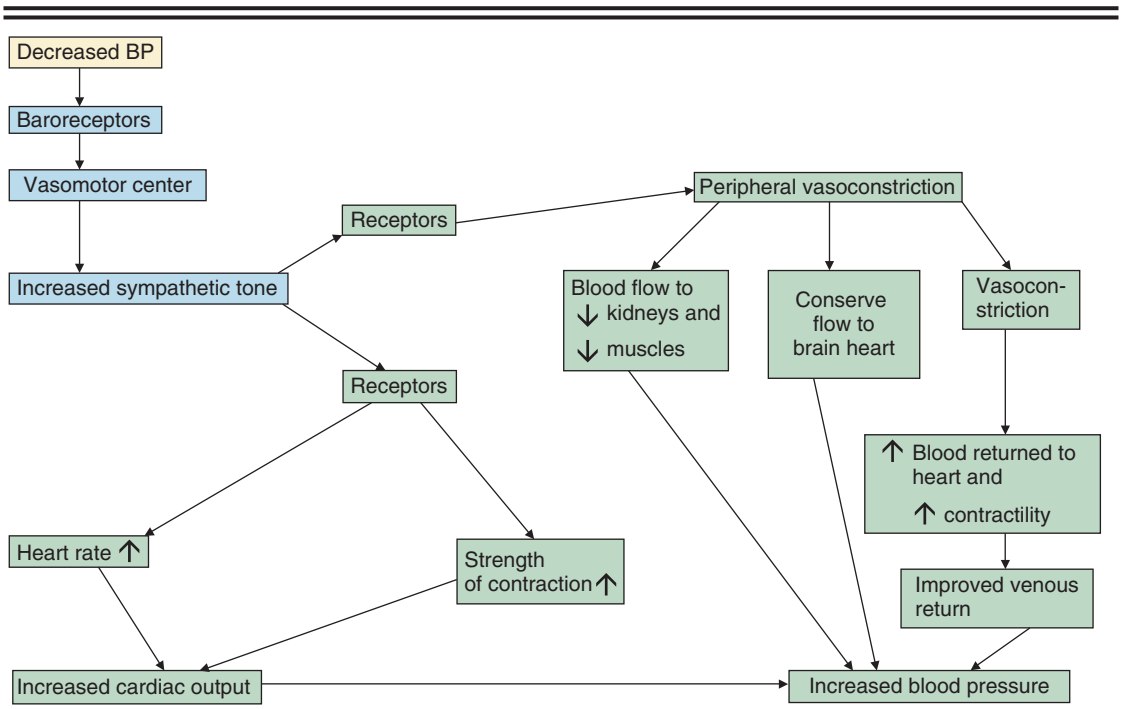


Fig. 15.2 Effect of compensatory sympathetic system in response to a reduction in blood pressure (*BP*). Decreased systemic BP results in activation of the sympathetic nervous system (SNS) and a cascade of consequences that are deleterious in the long term. (Used with permission of Dr. Walt Ingwersen, Ontario, Canada.)

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venous return associated with activation of the RAAS and SNS (increased diastolic wall stress). Chronic volume overload causes an increase in diastolic wall stress, and this leads to replication of sarcomeres in series, elongation of myocytes, and ventricular dilation. This compensatory mechanism allows the ventricle to pump a relatively normal amount of blood despite reductions in forward flow that occur with valve regurgitation or primary myocardial failure.

- Concentric myocardial hypertrophy (or pressure-overload hypertrophy) is characterized by thickening of the ventricular walls in response to increased systolic wall stress, and occurs as a compensatory mechanism to normalize systolic wall stress. Increased systolic wall stress stimulates the replication of sarcomeres in parallel, increasing myocardium thickness and thereby normalizing systolic wall stress. If the compensatory concentric hypertrophy is inadequate and the systolic wall stress remains increased, then afterload-mismatch and decompensation occur.

- **Additional compensatory mechanisms**
  - Endothelins are vasoactive peptides released from endothelial cells that play an important role in the regulation of vascular tone and blood pressure. Endothelin-1 is a strong, vasoconstrictive agent with inotropic and mitogenic actions and is also a strong stimulus for RAAS and SNS activation. Additionally endothelin-1 may play an important role in the pathogenesis of pulmonary hypertension.
  - Natriuretic peptides regulate salt and water homeostasis as well as blood pressure. They antagonize the RAAS and SNS, inhibit release of antidiuretic hormone, prevent myocardial fibrosis, and modulate cell growth and myocardial hypertrophy, thereby counterbalancing the volume expansion and vasoconstriction caused by other compensatory mechanisms. Atrial natriuretic peptide is primarily produced in the atria while B-type natriuretic peptide (BNP) is primarily produced in the ventricles. Both are released in response to chamber stretch, resulting in natriuresis, diuresis, and balanced vasodilation. The natriuretic peptides, especially BNP, are useful biomarkers of heart disease and CHF because blood concentrations are elevated in cardiac diseases characterized by volume expansion (such as DCM and mitral valve disease in dogs and cardiomyopathy in cats).
  - Inflammatory and immune mediators such as tumor necrosis factor alpha, interleukins (IL-1, IL-2, and IL-6), nuclear factor-kappa-B, and reactive oxygen species are thought to play a role in the progression of heart failure. The "cytokine hypothesis" suggests that heart failure progresses because cytokine pathways are activated in response to cardiac dysfunction and these mediators exert negative effects on the cardiovascular system.

Key Point

In the syndrome of heart failure, changes in ventricular mass and geometry are a culmination of compensatory hypertrophy (in response to pressure or volume overload) and pathologic hypertrophy (in response to activation of the RAAS and SNS).

Key Points

- The diagnosis of CHF is considered to be multimodal, on the basis of a careful history, physical examination, and ancillary diagnostics including electrocardiography, thoracic radiography, and echocardiography.
- Good-quality thoracic radiographs are invaluable in the diagnosis of left-sided CHF; however, cageside ultrasound is especially useful for the evaluation of unstable patients to determine if sufficient heart disease is present and determine if there is evidence for effusions and lung edema.

DIAGNOSIS OF CONGESTIVE HEART FAILURE

HISTORY

A complete medical history is important to establish the clinical course of the presenting complaint, as well as the presence of concurrent diseases that might complicate heart disease.

- Respiratory signs (including coughing, tachypnea, dyspnea), exercise intolerance, lethargy, and weakness are common complaints of clients with pets with symptomatic heart disease. Other signs of advanced heart disease and CHF include inappetence, weight loss, and syncope.
- Coughing is a common historical finding in dogs with heart disease. It can be due to either left atrial compression of the mainstem bronchus or concurrent airway disease (e.g., tracheal collapse and chronic bronchitis are common comorbidities in small-breed, geriatric dogs). Cough receptors are located in the large airways, but the initial accumulation of pulmonary edema is in the interstitium and alveolar space. Therefore pulmonary edema does not stimulate cough receptors until CHF fluid becomes severe enough to flood the large airways. Even early pulmonary edema, however, causes an increase in respiratory rate, and so tachypnea is a consistent clinical sign of CHF. It is important to recognize that although heart disease can cause a cough (from mainstream bronchus compression), this is distinct from and treated differently than CHF.
- Tachypnea (increased respiratory rate) is a common sign of CHF. Tachypnea can be caused by many nonpathologic mechanisms (e.g., stress) and

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is often overlooked by the client, especially in the early stages of CHF. Left-sided CHF with interstitial pulmonary edema causes stimulation of receptors within the pulmonary interstitium that reflexively increase the respiratory rate. The increase in respiratory rate can occur with or without the presence of hypoxia. Because of this concept, the resting respiratory rate may be used effectively to monitor the status of patients with left-sided CHF or who are at risk for CHF. The normal resting respiratory rate for most small animals is usually under 30 breaths per minute. Typically trends of increasing resting respiratory rate indicate progressive decompensation of left-sided CHF. Daily resting or sleeping respiratory rate monitoring by clients is a sensitive and practical way to detect CHF in its early stages. Several smartphone apps make it simple and convenient for owners to track breathing rates in their pets.

- Dyspnea (labored breathing) usually accompanies severe CHF. Dyspnea is exacerbated by exercise in patients capable of exercising. This is a term used in human medicine that describes how a person feels when trying to breathe. This term has been loosely applied to veterinary medicine to describe animals that have tachypnea with distress.
- Orthopnea (difficult breathing during recumbency) often occurs before dyspnea. Sometimes the client recognizes orthopnea because of reluctance of the pet to lie down or difficulty breathing when lying down.
- Nocturnal coughing or dyspnea typically occurs after the patient has been recumbent for some time.
- Exercise intolerance can be a very early sign of CHF in animals that are active but is difficult to identify in sedentary pets. Reduced tolerance for exercise results because cardiac function is abnormal, and the metabolic demands of tissues (especially the working muscles) are not met, leading to hypoxia, lactic acidemia, muscle weakness, and fatigue.
- Abdominal distention secondary to ascites occurs with right-sided or biventricular CHF and must be differentiated from effusions associated with other conditions, such as hypoproteinemia, liver disease, and abdominal neoplasia. Distended or pulsating jugular veins support a cardiac cause of ascites. Sometimes the abdomen can appear distended as a result of hepatomegaly from congestion but without ascites.

CARDIOVASCULAR EXAMINATION

- A cardiac murmur is present in many animals with CHF, but the presence of a murmur is not specific for CHF, and some animals (especially cats) might not have a murmur even when heart disease is advanced enough to cause CHF. A murmur is a series of auditory vibrations associated with turbulent (nonlaminar) blood flow and, occasionally, with vibrating valve leaflets. When blood flow velocities are higher-than-normal physiologic velocities or when blood viscosity is reduced (e.g., with anemia), blood flow tends to be nonlaminar and turbulent. Nonlaminar blood flow generates acoustic energy that vibrates the structures within the heart or the associated vessel(s). The intensity (loudness), timing in the cardiac cycle, frequency (pitch), configuration (shape), quality, duration, location, and direction of radiation of the sound created by the blood flow are important characteristics of cardiac murmurs. The murmur description can help to hone the differential diagnoses. For example, a left apical systolic murmur is most likely due to mitral regurgitation and recognizing this can prompt consideration for the various causes, including degenerative valve disease, infection, or dysplasia.
  - The intensity of the murmur refers to the loudness of the murmur and is graded on a scale from 1 to 6 (1 being the least audible murmur and 6 being the loudest).
  - The timing is defined as systolic, diastolic, or continuous.
  - The location is described as apical or basilar.
  - The configuration of the murmur refers to the shape of the sound created by the abnormal blood flow and is described as being either a plateau (regurgitant murmur) or a crescendo-decrescendo (ejection murmur) sound.
- Gallop sounds are abnormal heart sounds in dogs and cats that indicate advanced heart disease. A third heart sound (S3 gallop) occurs just after the second heart sound (S2) during the rapid diastolic phase of ventricular filling and is a very low-pitched sound heard best with the bell of the stethoscope. An S3 gallop occurs commonly in dogs with volume overload, particularly dogs with DCM. During early diastolic filling, blood rushes from the atrium into a noncompliant ventricle, which then vibrates, resulting in a low-frequency sound just after S2. A fourth heart sound (S4 gallop) occurs just before the first heart sound (S1) and is associated with atrial contraction. Ventricular filling in late diastole associated with atrial contraction vibrates the noncompliant ventricle, creating a low-frequency sound just before S1 and the onset of systole. S4 gallops are commonly auscultated in cats with hypertrophic or restrictive cardiomyopathy and are similar in intensity to the normal heart sounds. Summation gallops (S3 and S4) may occur during periods of tachycardia. An S4 gallop sound is more commonly found in cats, whereas an S3 gallop sound is more commonly found in dogs.
- Arrhythmias are common in animals with advanced heart disease and CHF. Electrocardiography is necessary for the specific rhythm diagnosis, but auscultation of an irregular rhythm and pulse deficits supports a rhythm abnormality that requires further investigation.

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- Atrial fibrillation is a common arrhythmia present in dogs with DCM and chronic mitral regurgitation secondary to degenerative valve disease, and also can occur in cats with cardiomyopathy. The onset of atrial fibrillation causes a decrease in cardiac output and a rise in atrial pressures due to the loss of coordinated atrial contraction. These changes often precede clinical decompensation and overt CHF.
- Ventricular tachyarrhythmias (ventricular premature beats and ventricular tachycardia) can occur in dogs with heart disease or with extracardiac disease (such as vehicular trauma, splenic disease, and gastric dilatation-volvulus). Ventricular arrhythmias are especially common in Boxers and Doberman Pinschers with cardiomyopathy. Ventricular ectopy might indicate an increased risk for sudden death.
- Bradyarrhythmias such as third-degree (complete) atrioventricular block often cause signs of reduced cardiac output (lethargy, weakness, syncope, and exercise intolerance) and occasionally, CHF.

- Abdominal palpation: Abdominal distension and/or ballotable abdominal fluid are consistent with right-sided CHF, although imaging and fluid analysis might be required to confirm clinical suspicion. The jugular veins are a window into right atrial pressure, such that jugular distension and pulsation above the thoracic inlet in a standing animal indicates elevated right heart pressures and supports right-sided CHF.
- Lung auscultation: The most common abnormal lung auscultatory abnormality in dogs with CHF is increased bronchovesicular sounds, but often soft inspiratory crackles can be heard with careful auscultation. Loud crackles usually indicate airway disease. However, lung sounds might not be overtly abnormal in dogs with CHF. Dull lung sounds ventrally indicate pleural effusion, a common manifestation of CHF in cats.
- Vital signs: It can be challenging to rapidly determine if a tachypneic patient is suffering from CHF or a noncardiac cause of respiratory distress; however, basic vitals such as heart rate, respiratory rate, and temperature can be helpful at directing the clinician one way or another. In addition to tachypnea, most patients with CHF are tachycardic and have either low or low-normal body temperature. Although there are always exceptions, findings of bradycardia, sinus arrhythmia, or elevated body temperature do *not* support CHF as a cause of respiratory distress.

BLOOD PRESSURE

While blood pressure abnormalities are not causative for CHF, it is important to assess blood pressure once a patient is stable enough for this procedure. Systemic hypertension should be treated because it can contribute to the progression of mitral valve disease. Blood pressure assessment is also critical when instituting and monitoring afterload reduction.

ROUTINE BLOODWORK

Baseline serum biochemistry parameters (blood urea nitrogen, creatinine, sodium, potassium, chloride, albumin, bicarbonate) provide information about renal function prior to starting cardiac medications that have the potential to affect the kidneys. While it is ideal to have this information before starting medications, a patient with breathing difficulty should not be restrained for venipuncture until stable. This might mean starting diuretic therapy without knowledge of renal parameters.

Key Points

Measurement of NT-proBNP is useful in both the general practice setting (to screen cats for occult heart disease that can then prompt follow-up with echocardiography) and in the emergency room setting (to determine if dyspnea is due to CHF or respiratory disease).

The feline NT-proBNP snap test is useful in the emergency setting to answer the question regarding the cause of dyspnea, but it should not be used in the asymptomatic patient because it does not provide quantitative results.

CARDIAC BIOMARKERS

- Natriuretic Peptides
  - Circulating natriuretic peptides are elevated in patients with significant cardiac remodeling. These concentrations can be used in combination with other diagnostic tests to differentiate between clinical signs associated with cardiac disease and those associated with primary respiratory disease. *N*-terminal pro-B-type natriuretic peptide (NT-proBNP) is the inactive product of BNP cleavage that has a long enough half-life to allow for detection using a commercially available test.
  - Normal NT-proBNP concentrations support the absence of CHF, but elevated concentrations can mean either heart disease or CHF in dogs. In general, dogs with degenerative mitral valve disease show progressive increase in NT-ProBNP concentrations with a significant increase during the 6-month period before symptomatic CHF. NT-proBNP concentrations might be useful for monitoring the response to CHF therapy because they are related to stretch of the ventricles.
  - NT-ProBNP concentrations can be used to identify cats with occult cardiomyopathy using a cutoff value of 100 pg/mL. NT-proBNP concentrations are also a useful discriminator of the cause of respiratory distress in cats, which is the basis for the SNAP test designed for use in the

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emergency setting. A positive SNAP test indicates a NT-proBNP concentration greater than 270 pg/mL which is specific for CHF as the cause of respiratory distress in cats. A negative SNAP test does not rule out underlying heart disease, but strongly supports that respiratory disease (not CHF) is the cause of distress. For this reason, the SNAP test should only be used in the emergency setting. Quantitative analysis of NT-proBNP provides more information for the asymptomatic cat because a lower cutoff value (100 pg/mL) is used for prediction of heart disease in cats.
    - It is important to note that NT-ProBNP levels can be elevated with renal insufficiency and pulmonary hypertension. To this end, NT-ProBNP levels may be helpful in monitoring the response to therapy for pulmonary hypertension.
- Cardiac troponin-I
    - This regulatory protein of the contractile apparatus is released into the serum as a result of myocardial cell damage, much like alanine transferase is released in the presence of hepatocellular damage. The most common causes of increased cardiac troponin-I are myocardial ischemia, myocarditis, neoplasia, gastric dilation-volvulus, and sepsis. The degree of cardiomyocyte damage is directly related to cardiac troponin-I concentrations and therefore has prognostic value.
    - Cardiac troponin-I concentration is not a good differentiator of respiratory distress in dogs or cats. NT-proBNP concentrations are better for this purpose.
    - Elevation in cardiac troponin-I is a predictor of the future development of DCM in Doberman Pinschers and other large-breed dogs (such as Great Danes) and therefore can be incorporated into screening for cardiomyopathy in this breed.

ELECTROCARDIOGRAPHY

- Electrocardiography is used to detect cardiac rhythm disturbances, chamber enlargement patterns, or conduction abnormalities that may be associated with cardiac disease. Electrocardiographic abnormalities are not specific for CHF and therefore should not be used for diagnosis of heart failure. Furthermore a normal electrocardiogram does not rule out the possibility of severe heart disease with secondary chamber enlargement.
- Patients with CHF are expected to have sinus tachycardia as a normal SNS response to dyspnea or poor cardiac output.
- Atrial fibrillation, atrial premature beats, and ventricular ectopy (single premature beats or ventricular tachycardia) are common findings in animals with CHF. The onset of atrial fibrillation can cause decompensation and onset of CHF, especially right-sided CHF.

THORACIC RADIOGRAPHY

- Thoracic radiography is important in the evaluation of patients with suspected heart disease or CHF. Three questions should be answered when evaluating a thoracic radiograph for heart disease:
    - Is cardiomegaly present? If so, which side of the heart is affected? The vertebral heart score (VHS) is a method of evaluating the size of the heart on a lateral thoracic radiographic view. The height or long axis of the heart (measured from the carina to the apex) and the width or short axis (measured at the widest part of the heart; perpendicular to the long axis) of the heart are measured and expressed as the number of vertebral bodies beginning with T4, measured caudally (normal <10.5 in dogs and <8.5 in cats). This score is reliable in dogs older than 3 months of age into adulthood but it can be impacted by skeletal abnormalities. There is variation between breeds (e.g., Pug, Pomeranian, Yorkshire Terrier, Dachshund, Bulldog, Shih Tzu, Lhasa Apso, Boston Terrier, Cavalier King Charles Spaniel, Boxer, and Labrador Retriever breeds can have increased VHS as a normal finding) so breed-specific reference ranges should be used when available. Vertebral-based assessments of radiographic size (e.g., vertebral left atrial size) have also been reported to capture left atrial enlargement more specifically than VHS. See Chapter 2 (Radiology of the Heart).
    - What is the pulmonary vascular pattern (e.g., normal, undercirculated, overcirculated, venous distention, or arterial distention)? Pulmonary venous distension is an expected, but not universal finding in left-sided CHF. Pulmonary arterial distension is consistent with pulmonary hypertension or heartworm disease. Cats with CHF (unlike dogs) often show both venous and arterial venous distension. Undercirculated pulmonary vasculature could be consistent with a right-to-left shunt while overcirculation is consistent with a left-to-right shunt.
    - Is there evidence of CHF (pulmonary edema, pleural effusion, or ascites)?
- The earliest evidence of pulmonary edema formation is pulmonary venous congestion (radiographically noted when the veins are larger than their corresponding arteries), which tends to occur when pulmonary capillary pressures approach approximately 15 to 20 mm Hg. Cats often show dilation of both pulmonary veins and arteries even when CHF is left-sided, unlike dogs who tend to just show pulmonary venous enlargement.
- As pulmonary venous and capillary pressures progressively increase with left-sided CHF, decompensation occurs, and fluid leaks into the interstitium (interstitial edema).

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- As pulmonary venous and capillary pressures continue to rise (e.g., 30–40 mm Hg), the alveoli become flooded with edema fluid.
- Cardiogenic pulmonary edema can be primarily perihilar (centrally located) in distribution, or it may be diffuse and generalized. As the edema progressively becomes more generalized, it is commonly noted first in the right caudal lung lobe, especially in dogs with mitral regurgitation. In cats, the manifestation of pulmonary edema can be variable, with some cats having a classic perihilar interstitial pattern and others having a patchy, mixed interstitial-alveolar pattern that is not primarily located in the perihilar region.
- Pleural effusion can occur in cases with biventricular or severe right-sided CHF in dogs. Pleural effusion in cats can be a manifestation of either left-sided or biventricular CHF.

ECHOCARDIOGRAPHY

- The three principal modalities of echocardiography are two-dimensional, M-mode, and Doppler echocardiographic evaluation of the heart. Echocardiography enables the trained specialist to identify diseases that cause or can be associated with heart disease or CHF, such as valvular leaks (e.g., mitral regurgitation), myocardial failure (e.g., DCM), intracardiac or extracardiac shunts (e.g., patent ductus arteriosus, ventricular septal defect), decreased ventricular compliance (e.g., HCM), and pericardial disease. Echocardiography is not useful to diagnosis CHF (although it can document elevated filling pressures consistent with CHF), however, and thoracic radiographs are the test of choice for this purpose. Echocardiographic findings can support or refute the presence of heart disease sufficient to cause CHF and can define the underlying diagnosis to refine the overall treatment plan. Therefore, echocardiography is typically performed once the signs of acute CHF are treated and the patient is stabilized.
- The combination of thoracic radiographs and echocardiography allows the clinician to define structural heart disease and identify CHF in most cases. Typically CHF is not present without atrial enlargement (with the exception of pericardial diseases and acute chordae tendineae rupture). The atria are usually compliant and will enlarge as filling pressures increase.

POINT-OF-CARE ULTRASOUND

- Cageside focused ultrasound (point-of-care ultrasound or POCUS) has become widely utilized in the urgent care setting to help rapidly determine the etiology of respiratory distress. This approach can be done in animals that are too dyspneic to safely undergo radiography. POCUS can be viewed as an extension of the physical examination in the emergency room setting. If heart disease is identified, however, this should be followed up by a full echocardiogram to evaluate all aspects of cardiac size and function, to accurate define the underlying disease, and to inform a treatment plan. Therefore, POCUS is complementary, but should not be viewed as a replacement for echocardiography.
- POCUS evaluates select images of the heart to determine if sufficient heart disease is present to cause CHF and if so, whether left-sided or right-sided cardiomegaly is present. Lung ultrasound is also part of POCUS and can be used in place of radiography to identify alveolar fluid which, along with other findings, can support the diagnosis of CHF.
  - Basic cardiac views, including the short-axis left-sided view at the heart base, allows indexing of the left atrial size to the aortic size to determine if left atrial enlargement is present. Moving more apically will produce an image showing the left and right ventricles in cross section. If the right ventricle is of similar size or larger than the left ventricle, right ventricular disease is likely present. Although pulmonic stenosis should be ruled out, finding right ventricular chamber enlargement and/or increased wall thickness relative to the left ventricle (particularly in a middle-aged or older dog without a history of a heart murmur), supports right ventricular hypertension and in most situations is consistent with pulmonary hypertension. Importantly the presence of right ventricular enlargement, especially when the left atrial size is normal, does *not* support left-sided CHF.
  - The thoracic cavity is also scanned for effusions (pleural and pericardial). This is an expedient way to come to a timely diagnosis of pericardial effusion that can be lifesaving. Masses associated with pericardial effusion might be readily evident depending on their size. If a mass is not seen on POCUS, a full echocardiogram should be performed once the dog is stable. POCUS can be used to identify a good location for centesis for either pleural or pericardial fluid removal.
  - The lung fields can be evaluated by assessing several quadrants on both sides of the chest. Increased numbers of B-lines (bright comet tails extending from the chest wall interface near the probe to the bottom of the ultrasound field) indicate fluid trapping in the alveoli. This is consistent with CHF (pulmonary edema), but it is not specific, because other causes of alveolar fluid are possible (e.g., pneumonia). However, finding numerous B-lines indicates CHF if other findings are supportive (e.g., left atrial enlargement is also present).
  - The caudal vena cava can be visualized from the subcostal probe position and provides an indication of right atrial pressure. For example,

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nondistensibility of the caudal vena cava indicates that right atrial pressure is elevated, which is supportive of right-sided CHF as the cause of third cavity spacing (e.g., pleural effusion or ascites).

Key Points

Dyspneic patients require a "hands-off" approach to minimize stress.

Point-of-care ultrasound can be considered an extension of the physical examination. In the emergency setting, it can be done with the patient remaining in the oxygen cage, providing a rapid assessment of left and right heart sizes as well as determination of the presence of effusions or lung parenchymal fluid that would be consistent with pulmonary edema.

THERAPEUTIC STRATEGIES FOR THE MANAGEMENT OF HEART FAILURE

OXYGEN

Provision of a higher inspired oxygen content (typically FiO_2 40%) helps inspired oxygen enter the bloodstream of dogs with the diffusion barrier caused by pulmonary edema.

POSITIVE INOTROPY

- **Pimobendan** is an inodilator, meaning that it causes peripheral vasodilation and positive inotropy; qualities that improve forward flow in dogs with heart disease caused by mitral regurgitation or DCM. Systemic vasodilation after pimobendan administration is a result of phosphodiesterase (PDE)-3 inhibition, which increases intracellular cyclic adenosine monophosphate (cAMP). While PDE-3 inhibition also brings some calcium into the cell, the major enhancement to contractility (positive inotropy) after pimobendan administration is through increased sensitization of actin and myosin to existing intracellular calcium.
  - Pimobendan is considered standard therapy along with RAAS inhibitors and diuretics for dogs with CHF due to either cause. It is also used in preclinical disease to delay progression to CHF. Current studies have demonstrated increased survival times and improved quality of life in patients with CHF (ACVIM stages C and D) secondary to DCM or mitral valve disease. Pimobendan delays the onset of clinical signs in Doberman Pinschers with occult DCM (preclinical) and dogs with heart enlargement secondary to degenerative mitral valve disease (ACVIM stage B2). Pimobendan is also beneficial for pulmonary hypertension secondary to chronic left-sided CHF and other disease scenarios resulting in myocardial failure, such as heartworm disease and congenital heart disease.
  - Although pimobendan is typically used for the chronic management of CHF, it should be administered in the acute setting as soon as is feasible. The tablet can be slurried in water and administered orally by syringe to reduce stress on the dyspneic patient. The author gives double the standard dose (0.5 mg/kg) for the first dose in the acute setting because this has been shown to more effectively reduce left atrial pressures than 0.25 mg/kg.
  - Pimobendan is effective in cats despite the differing pharmacokinetics compared to dogs (longer elimination half-life and higher plasma levels). It is indicated in cats with myocardial failure (e.g., DCM) because of its positive inotropic effect. Pimobendan is used by some clinicians to treat CHF in cats with other cardiomyopathies, although the evidence for efficacy is conflicting in HCM. It appears well tolerated in most cats, although it should be avoided in hypotensive patients with obstructive HCM.
  - The standard dosage of pimobendan in dogs and cats is 0.25 mg/kg PO q12h. Higher dosages and increased administration frequency are used by many cardiologists, especially when addressing recurrent CHF episodes.
- **Sympathomimetics**
  - Dobutamine and dopamine are drugs that mimic the actions of the SNS (i.e., alpha- and beta-adrenergic receptor agonists). They are used only in the therapy of acute severe CHF and are best suited to short-term IV use. Both dobutamine and dopamine exert a positive inotropic effect by stimulating myocardial beta-1 adrenergic receptors, which results in increased cAMP levels through adenylate cyclase stimulation. The increased levels of cAMP increase calcium movement through the slow inward calcium currents and increase calcium storage by the sarcoplasmic reticulum, making more calcium available to the contractile elements. Dobutamine stimulates both beta-1 and beta-2 adrenergic receptors in the peripheral vasculature, and this combination results in no major changes in blood pressure. The positive chronotropic effect (increased heart rate) of dobutamine at higher doses should be avoided in most cases.
  - Dopamine produces similar effects as dobutamine, but dobutamine is used more commonly because it favors blood flow to myocardial and skeletal muscle (whereas dopamine favors blood flow to the renal and mesenteric systems). Additionally dopamine infusion rates above 10 µg/kg/min can be associated with vasoconstriction and increased diastolic intraventricular pressures.
  - Adverse effects associated with sympathomimetic drug administration are usually dose-related and

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include tachycardia, arrhythmias, hypertension, gastrointestinal disturbances, and phlebitis, if extravasation occurs. These signs of catecholamine toxicity abate with discontinuation of the drug.
- The dose of dobutamine in dogs is 5 to 20 µg/kg/min IV continuous infusion diluted in 5% dextrose. The dose of dopamine is 2 to 8 µg/kg/min IV continuous infusion diluted in 5% dextrose.

RAAS INHIBITION

- **Angiotensin-converting enzyme inhibitors (ACEi)**
  - ACEi prevent the conversion of angiotensin I to angiotensin II. Angiotensin II is a potent vasoconstrictor, promotes sodium and water retention, and causes the release of aldosterone; therefore lowering its production dampens all of these maladaptive effects. ACEi also promote the formation of alternative RAAS metabolites, which have beneficial effects of vasodilation, natriuresis (sodium excretion), and diuresis.
  - Several large, multicenter clinical trials have demonstrated the safety and efficacy of ACEi in the treatment of CHF secondary to DCM and primary mitral regurgitation. ACEi decrease the clinical signs of CHF, improve quality of life, and decrease mortality when used in combination with conventional therapy.
  - Studies do not universally support benefit for ACEi in patients with asymptomatic mitral valve disease (ACVIM Stage B). However, some clinicians initiate ACEi therapy at this stage, especially for dogs with significant cardiac remodeling (e.g., severe cardiomegaly, pulmonary venous hypertension) or systemic hypertension (>160 mm Hg average systolic pressure).
  - The benefit of ACEi therapy is realized over weeks to months and therefore ACEi are typically started after hospital discharge (for acute CHF treatment) once the patient is eating and drinking well (i.e., they are not usually started in hospital). Clinical benefit in the acute setting is unlikely to be significant, and use at this time might predispose some patients to functional azotemia because of volume depletion and/or hyponatremia.
  - Side effects associated with ACEi therapy occur infrequently. Anorexia or inappetence, vomiting, hypotension, and azotemia are the most common side effects of ACEi therapy. Azotemia secondary to decreased glomerular perfusion represents the most important clinical consideration. Azotemia and renal dysfunction can occur from loss of protective mechanisms to maintain filtration pressures. Angiotensin II causes vasoconstriction of the renal efferent arterioles in an effort to maintain glomerular filtration pressures in the face of decreased renal perfusion. Therefore, the relative decrease in angiotensin II can significantly decrease glomerular perfusion and filtration, resulting in azotemia in patients who are strongly dependent on angiotensin II to maintain renal blood flow (e.g., those with preexisting renal disease, hyponatremia, or dehydration). As a result, ACEi should only be started in the hydrated patient with normal blood pressure and normo-natremia.
    - Renal function should be evaluated before starting an ACEi, within a week of starting an ACEi, and every 3 months thereafter, especially during concurrent diuretic therapy.
    - Because of the inherent decrease in glomerular filtration pressure associated with less angiotensin II, a small increase in serum creatinine is expected after ACEi initiation (typically < 0.3 mg/dL). Serum creatinine increases that are greater than 0.3 mg/dL should prompt downward dosage adjustments of diuretics and/or ACEi and if severe, at least temporary cessation of ACEi therapy.



Key Point

ACE inhibitors are recommended for patients with asymptomatic myocardial dysfunction (occult DCM); in patients with CHF secondary to degenerative mitral valve disease, DCM, and other etiologies (stages C and D); and in patients with systemic hypertension.

  - Several ACEi are used in veterinary medicine, and clinician preference often dictates which one is used. Enalapril and benazepril are the most commonly used, but others such as lisinopril and ramipril have been used in dogs and cats.
    - Enalapril is renally excreted, whereas benazepril undergoes renal and hepatic elimination, which potentially makes benazepril a better choice in patients with underlying renal disease. Benazepril also has superior tissue RAAS inhibiting qualities.
    - Most ACEi are dosed at 0.5 mg/kg PO q12–24h in dogs and 0.25 to 0.5 mg/kg PO q12–24h in cats.



Key Point

Comprehensive RAAS suppression is considered standard therapy for CHF. The phenomenon of aldosterone breakthrough in some dogs necessitates both an ACEi and spironolactone to protect against the deleterious effects of excessive aldosterone formation as a result of maladaptive RAAS activation in CHF.

- **Aldosterone antagonists (e.g., spironolactone)**
  - Spironolactone antagonizes the effect of aldosterone in tissues by binding to its receptor. Blocking

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the receptor in the distal renal tubule and collecting duct causes sodium excretion and potassium excretion, but despite inhibiting sodium excretion, spironolactone does not have an appreciable diuretic effect. Aldosterone antagonists (also called *mineralocorticoid receptor antagonists*) improve survival in people with heart disease, and several studies in dogs support similar delays of disease progression and improvement in survival. This cardioprotective effect likely occurs as a result of aldosterone blockade in the myocardium, vasculature, and kidneys which inhibits fibrosis in these organs.

- Aldosterone antagonism is an important part of comprehensive RAAS suppression because studies in people and in dogs show that 30% to 40% of those receiving appropriate ACEi therapy have aldosterone breakthrough. The cause of aldosterone breakthrough is not known but could be multifactorial. One study showed that an ACE polymorphism predisposed to aldosterone breakthrough in dogs. Aldosterone is profibrotic and contributes to disease progression, so if ACEi are insufficient for reducing aldosterone production in some patients, it is critical that an aldosterone receptor blocker be part of the treatment regime for myocardial protection. Spironolactone is currently a part of standard therapy for stage C or D CHF with ACEi, a loop diuretic (e.g., furosemide or torsemide), and pimobendan. Aldosterone angtagonism may also have a role in stage B2 heart disease but it has not conclusively been shown to delay disease progression in this stage.
- Although spironolactone does not have an appreciable diuretic effect, it can contribute to potassium retention which can be beneficial in patients who are also receiving potassium-wasting diuretics. Monitoring serum potassium concentrations is important with spironolactone, especially for patients also on an ACEi and not receiving diuretic therapy who might be at risk for hyperkalemia. Fortunately, hyperkalemia is uncommon but dose reduction or discontinuation would be indicated if it occurs. In one feline study, some cats experienced facial excoriations with spironolactone but this seems to be rare in clinical practice and might have been related to higher dosages used on that study.
- Spironolactone is administered at 2 mg/kg PO q24h. The dose can be increased to q12h to address persistent hypokalemia. Serum electrolytes should be measured before and 1 to 2 weeks after starting spironolactone, and every 3 months thereafter. This assessment can be combined with monitoring of renal function with ACEi therapy.

Key Points

Torsemide is a more potent and more bioavailable loop diuretic than furosemide.
Transition to torsemide can be an effective way to restore diuresis in patients with poor diuretic response.
Renal function and electrolytes should be closely monitored in patients receiving diuretics, but especially when torsemide is used.

PRELOAD REDUCTION (DIURETICS)

- **Loop diuretics** bind to the sodium/potassium/2-chloride ($Na^+/K^+/2Cl^-$) cotransporter in the thick ascending limb of the loop of Henle. Their use prevents the resorption of these electrolytes back into the bloodstream and promotes their loss into the urine with water following.
  - Adverse side effects of loop diuretics include intravascular volume loss and electrolyte abnormalities, such as hypokalemia, hyponatremia, and hypochloremia with metabolic alkalosis. Overzealous diuretic therapy can cause dehydration, pre-renal azotemia, and low cardiac output. Furthermore diuretics activate systemic compensatory mechanisms, such as the RAAS and SNS. Because of the potential for side effects, assessment of hydration status and serum renal values and electrolytes should be done after drug initiation and periodically thereafter.
  - Loop diuretics are lifesaving for dogs with CHF but they should not be used as monotherapy. Other CHF medications, including pimobendan, ACEi, and spironolactone are used to support myocardial function and counteract maladaptive neurohormonal activation, which can help to lower the need for diuresis.
  - Reduced diuretic responsiveness can occur over time with any loop diuretic, sometimes after a relatively short period of time. Various contributors to poor diuretic response are possible (e.g., noncompliance, poor bioavailability, rebound sodium and water retention between doses), but true diuretic resistance likely occurs secondary to neurohormonal activation and hypertrophy of distal nephron segments to resorb sodium (after the site of loop diuretic action). The loop diuretic dosage needed to control CHF signs is a marker of CHF severity and is a negative predictor of outcome. Therefore, strategies to prevent diuretic resistance and minimize diuretic dose might improve long-term outcome. Diuretic responsiveness might be restored by changing to a diuretic with high bioavailability (e.g., torsemide), especially when gastrointestinal edema is present, increasing the frequency of diuretic administration, or stacking diuretics (e.g., adding a diuretic with a different mechanism or site of action). Additionally ensuring that standard therapy is being administered

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(e.g., pimobendan, comprehensive RAAS suppression) is important. Adjunctive therapy (e.g., afterload reduction, dietary sodium restriction) can also help control CHF signs.

- Furosemide is the most commonly used loop diuretic in veterinary medicine.
  - Intravenous (IV) and intramuscular (IM) furosemide acutely increase venous capacitance secondary to release of vasodilatory prostaglandins. This vasodilatory effect occurs within the first 20 minutes after IV or IM administration, and the peak diuretic effect occurs after 30 to 45 minutes.
  - Oral furosemide produces diuresis for approximately 6 hours with a peak effect at 1 to 2 hours after administration. It has a wide range of bioavailability in dogs (10%–90%), which is further reduced by concurrent food.
  - The dose of furosemide for dogs is:
    (a) 1-3 mg/kg PO q8–12h for chronic CHF treatment. A mid-range dose of 2 mg/kg PO q12h is typically used for initial treatment of CHF, with dose escalations prompted by CHF recurrences. Total daily dosages exceeding 6 to 8 mg/kg/day for CHF control should prompt consideration of switching to torsemide or employing other strategies (e.g., diuretic stacking, sodium restriction, afterload reduction) because of the possibility of diuretic resistance.
    (b) Parenteral administration of furosemide for acute CHF is usually started at 2 mg/kg IV (or IM if IV access is not possible). This dose can be repeated every 1 to 2 hours until a clinical response is seen (i.e., respiratory rate reduction) after which the dose and frequency should be sharply curtailed. The clinician needs to be cognizant of the cumulative furosemide dose administered during acute CHF treatment to avoid adverse effects of volume and electrolyte depletion. Furosemide can also be administered as a constant rate infusion (0.6 mg/kg/h after a loading bolus with downward adjustment based on clinical response), which has been shown to cause more diuresis at equivalent dosages to intermittent bolus administration. This means that there is more possibility for azotemia and electrolyte depletion as well, so monitoring cumulative doses and curtailing the infusion after clinical response is important regardless of the method of administration. Most dogs in the authors practice require <10 mg/kg cumulative furosemide over 24 to 48 hours for the treatment of acute pulmonary edema.
    (c) Cats appear to be more sensitive to the side effects of furosemide than dogs and so most clinicians use lower doses to treat CHF in this species. The dose for treatment of chronic CHF in cats is 1 to 2 mg/kg PO q12–24h. Similarly, 1 to 2 mg/kg is administered IV for treatment of acute CHF. If constant rate infusion is used, a lower dose of 0.3 to 0.4 mg/kg/h is started with downward adjustment or discontinuation with clinical improvement.
- Torsemide is 10 to 20 times more potent than furosemide, has a longer duration of action (12–24 hours), and is more consistently bioavailable (90%). As a result, it provides longer and more reliable diuresis than furosemide and therefore can be used to address diuretic resistance.
  - Adverse effects of torsemide are the same as for furosemide; however, the greater potency means that dosage adjustments should be subtle to avoid volume depletion and electrolyte abnormalities.
  - Torsemide is most often used in veterinary medicine as a rescue diuretic to treat dogs that are poorly responsive to furosemide (often ACVIM stage D). Consideration to transition to torsemide is therefore reasonable for dogs that are experiencing continued CHF signs despite oral furosemide dosages of >6 mg/kg/day. Although it can be helpful in these situations, studies in dogs and people suggest it is also an appropriate first-intention treatment for CHF. In fact, the pharmacokinetic properties of torsemide might make it superior to furosemide. A specific example where torsemide can be justified for first-intention treatment is for right-sided CHF where gastrointestinal edema is expected to unpredictably impair furosemide absorption.
  - The starting dose of torsemide for first-intention treatment of CHF in dogs and cats is 0.15 mg/kg PO q12-24h. Dosage adjustment based on CHF signs, thoracic radiographs, and bloodwork is frequently necessary to avoid over or under-dosing, and therefore close monitoring of these diagnostics is very important. Torsemide is only available in oral formulations in the United States.
  - When torsemide is used for rescue therapy for refractory CHF (i.e., when diuretic resistance is suspected), the patient is transitioned from furosemide to torsemide by calculating the total daily dose of furosemide, dividing that dose by 15 (to account for potency differences) and dividing that into 2 daily dose administrations. For example, if a dog requires furosemide 3 mg/kg PO q8h to control CHF signs, the total daily furosemide dose (9 mg/kg/day) is divided by 15 to obtain the total daily torsemide dose (0.6 mg/kg/day) which is divided in half to obtain 0.3 mg/kg PO q12h. Because this dosage conversion is an estimation, it is

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important to monitor the patient for both dehydration as well as continued CHF.

- **Thiazide diuretics** (e.g., hydrochlorothiazide) act by inhibiting distal renal tubule electrolyte reabsorption. These diuretics are less potent natriuretic agents and are usually not successful in controlling signs of congestion when used as a monotherapy. In general, thiazide diuretics are administered when CHF is refractory and conventional therapy (pimobendan, ACE inhibitor, and loop diuretic) fails to control the clinical signs of congestion.
  - The addition of hydrochlorothiazide to a loop diuretic to address diuretic resistance is called "diuretic stacking." Diuretic stacking is successful because hydrochlorothiazide prevents the distal tubular sodium resorption that occurs with nephron hypertrophy in this region when loop diuretics are chronically administered. This approach, however, has the potential to cause significant azotemia and electrolyte derangement and therefore close monitoring of hydration status and bloodwork are very important.
  - It is prudent to start hydrochlorothiazide at a low dose when adding it into a treatment regime that already includes a loop diuretic. Dosages as low as 0.5 mg/kg PO every other day can be gradually increased to 2 to 4 mg/kg PO q12–24h if needed.
- **Proximal renal tubular diuretics** promote sodium loss in the proximal convoluted tubule with water following, however, because the kidney can resorb this tubular sodium more distally (e.g., loop of Henle, distal renal tubule), these drugs are not particularly effective for diuresis. However, when they are added onto loop diuretics (i.e., diuretic stacking), they can provide more sodium in the tubular fluid, which the loop diuretics then block the absorption of, boosting natriuresis and diuresis.
  - Acetazolamide is uncommonly used in dogs to treat refractory CHF. It promotes chloride retention and bicarbonate excretion which can help to correct the hypochloremic metabolic alkalosis that can occur with loop diuretics. Both hypochloremia and alkalosis might contribute to diuretic resistance and so acetazolamide might prove helpful because of added diuresis and correction of hypochloremic metabolic alkalosis. A dose of 4 to 5 mg/kg PO q12h has been used in a limited number of cases.
  - Sodium-glucose cotransporter 2 (SGLT2) inhibitors block the resorption of sodium and glucose in the proximal convoluted tubule. This class of drugs was developed to treat diabetes in people and was subsequently found to improve survival for patients with heart disease. These drugs are not currently used for the treatment of CHF in dogs but represent an emerging treatment which might be useful in veterinary medicine.
- **Venodilators** cause relaxation of systemic veins to cause pooling of blood in the veins thereby reducing preload. They reduce congestive symptoms by redistributing blood volume within the circulatory system (the capacitance vessels and the splanchnic veins) and reducing venous return, which results in lower filling pressures (preload). Venodilation can therefore reduce edema formation (especially pulmonary edema) and allow time for diuretics to remove excess intravascular fluid through the kidneys. Nitrate vasodilators are excellent preload reducers in humans, although their efficacy in dogs and cats is unproven. The development of nitrate tolerance limits the continuous use (i.e., more than 36 hours) of these agents. Tolerance may be avoided by intermittent use (24 hours on and 24 hours off) and possibly with concurrent use of ACEi.
  - Two percent nitroglycerin paste is often used in the acute CHF setting along with diuretics, pimobendan and oxygen. The dose in dogs is ¼ to 2 in. cutaneously q6–8h for 24 to 48 hours, whereas in cats a smaller amount is use (1/8–¼ in.). The paste should be applied to hairless areas such as the pinna or the axillary region. If perfusion to the ears is poor (e.g., ears are cold), the axilla or groin will provide better absorption. Because nitroglycerin is absorbed transcutaneously, gloves should be worn during administration. A piece of white tape to the ear is a good way to alert veterinary staff to the presence of nitroglycerin paste on the animal.
  - Isosorbide dinitrate is an oral nitrate that is dosed at 0.2 to 1.0 mg/kg PO q8h in dogs.

AFTERLOAD REDUCTION

- Off-loading the ventricles through afterload reduction is an important aspect of treating CHF. Arterial vasodilators reduce systemic vascular resistance, thereby improving cardiac function and forward flow. Myocardial systolic wall tension is an important determinant of myocardial oxygen consumption. By decreasing systemic vascular resistance (and, therefore, afterload and systolic wall tension), arterial vasodilators reduce myocardial oxygen consumption. In addition, the reduction in afterload results in an increase in cardiac output. Arterial vasodilators decrease the volume of regurgitation across an insufficient valve and, therefore, increase forward flow in patients with mitral or aortic regurgitation (Fig. 15.3). Arterial vasodilators might also lessen the magnitude of left-to-right shunting in patients with ventricular septal defects. Conversely, arterial vasodilators exacerbate right-to-left shunting in patients with tetralogy of Fallot or with nonrestrictive ventricular septal defects.
- Afterload reduction is especially helpful in dogs with recurrent or refractory CHF due to primary mitral valve disease in both the acute and chronic setting. Although treatment of systemic hypertension is

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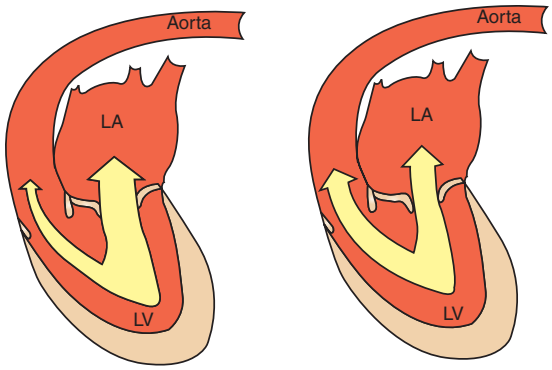


Fig. 15.3 Drawings of the left side of the heart from a dog with severe mitral regurgitation before and after administration of an afterload-reducing agent. Note that forward flow (out the aorta) increases with afterload reduction because of the reduction in systemic vascular resistance. *LA*, Left atrium; *LV*, left ventricle.

crucial in dogs with any degree of mitral regurgitation (because high afterload worsens mitral regurgitation), afterload reduction is useful to treat CHF even in normotensive patients. A minor reduction in blood pressure can result in significant increased forward flow because of the reduction in systemic vascular resistance that occurs. Systemic hypotension, however, is a contraindication to using arterial vasodilators and this approach is typically reserved for patients with systolic blood pressure above 110 to 120 mm Hg so that a reduction in systolic blood pressure (e.g., 15–20 mm Hg) can be safely targeted.

- Although ACEi lower blood pressure by reducing the formation of angiotensin II, they are generally considered weak afterload reducers.
- Pimobendan is an inodilator that reduces systemic vascular resistance through PDE-3 inhibition.
- **Amlodipine** is a calcium channel blocker with only peripheral vascular effects and no cardiac effects. It has a relatively long half-life (24 hours) and slow onset of action which means that signs of hypotension are uncommon. Amlodipine effectively lowers blood pressure in most patients and side effects are uncommon. Occasionally gingival hyperplasia necessitates replacement with another vasodilator (e.g., hydralazine). Amlodipine has been shown to activate the RAAS, so ACEi should be used concurrently.
  - The dose of amlodipine in dogs is 0.05 to 0.2 mg/kg PO q12–24h. The dose is titrated to achieve a blood pressure in the normal range (if hypertensive) or a 15 to 20 mm Hg drop in blood pressure.
  - The dose of amlodipine in cats is 0.625 to 1.25 mg/cat PO q12–24h.
  - In addition to monitoring blood pressure, renal values should also be monitored 1 week after initiation and periodically thereafter.
- **Hydralazine** is a direct-acting vasodilator with a relatively rapid onset of action (1 hour), which means that clinically evident hypotension can occur after dosing. The use of amlodipine has largely supplanted hydralazine when treating systemic hypertension or as adjunctive therapy for CHF management, however, it is still used when gingival hyperplasia occurs with amlodipine.
  - Hydralazine is started at 0.5 mg/kg PO; the dose is gradually increased until a clinical response is elicited, a drop in arterial blood pressure of 15 to 20 mm Hg occurs, or up to a maximum dose of 3.0 mg/kg PO q12h without systemic hypotension.
  - Side effects of hydralazine include hypotension, gastrointestinal upset, reflex tachycardia, and RAAS activation.
- **Sodium nitroprusside** is a parenteral, direct-acting vasodilator with mixed arterial and venous vasodilation. It is an extremely potent vasodilator that is primarily used to rescue dogs with severe, decompensated CHF in conjunction with standard therapy of oxygen, pimobendan, and parenteral furosemide.
  - Sodium nitroprusside is diluted in 5% dextrose for IV constant rate infusion. The dose is initially 1.0 µg/kg/min, but titrated to effect by monitoring blood pressure to obtain a drop in systolic blood pressure of 15 to 20 mm Hg. If the blood pressure cannot be obtained due to patient instability, the infusion is gradually increased (to a maximum dose of 10 µg/kg/min) until the respiratory rate decreases. Nitroprusside infusions are typically limited to 24 to 48 hours due to the potential for cyanide poisoning with longer infusions. Once a clinical response occurs and the patient stabilizes, an oral arterial vasodilator (e.g., amlodipine) is started and the patient is weaned off of nitroprusside.
  - Hypotension, tachycardia, nausea, and vomiting are the most significant adverse effects of nitroprusside administration but these are managed by reducing or discontinuing the infusion because of the very short half-life (minutes).

DIETARY MODIFICATION

- A reduction in dietary sodium intake can blunt the tendency to conserve sodium and to develop edema. Theoretically the use of low-sodium diets in combination with vasodilators and ACE inhibitors can reduce reliance on diuretics to control edema and signs of CHF. Early in the course of CHF, dietary sodium restriction should be in the form of elimination of high-salt-containing snacks, which can be unrecognized sources of salt intake in clinical patients. Mild to moderate dietary salt restriction can be helpful for the control of CHF, but severe salt restriction activates the RAAS, causing further progression of heart failure. There is little data to support sodium restriction in preclinical disease, which could be detrimental because of the

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RAAS-activating effects of salt restriction. Another disadvantage of markedly sodium reduced diets is unpalatability. Several reduced-sodium prescription diets are available commercially for dogs with CHF and these newer diets are also formulated to provide adequate protein and are fortified with supportive amino acids and fatty acids.

- General recommendations for dietary sodium intake levels in patients with heart disease are as follows:
  - Asymptomatic heart disease (ACVIM stage B): avoid high-sodium diets (recommend <100 mg/100 kcal)
  - Early or stable CHF (ACVIM stage C): mild sodium restriction (recommend <80 mg/100 kcal)
  - Severe CHF (ACVIM stage D): more aggressive sodium restriction may be beneficial

Key Point

An important consideration regarding sodium restriction in patients with CHF is to avoid treats or foods with excessive sodium content.

SUPPLEMENTS

- **L-carnitine** supplementation might be indicated in patients that have myocardial failure associated with definitive myocardial L-carnitine deficiency; however, this requires myocardial biopsy to diagnose, which is not practical. Some Boxers with cardiomyopathy have been noted to respond to L-carnitine supplementation and so it is reasonable to supplement Boxers with a DCM phenotype with L-carnitine. In most cases, however, myocardial L-carnitine deficiency is a result of the underlying disease and myocardial failure rather than the cause of the myocardial failure.
  - The dosage for L-carnitine supplementation in dogs is 50 to 100 mg/kg PO q8h.
- **Taurine** supplementation (sometimes in combination with L-carnitine) has been effective in reversing or improving disease in some dogs with DCM secondary to taurine deficiency (e.g., American Cocker Spaniels and other breeds) and in cats with taurine-deficient DCM. The addition of taurine to feline diets after 1987 dramatically reduced the incidence of taurine deficiency-induced DCM in cats. Although DCM secondary to taurine deficiency in cats is uncommonly encountered today, it is prudent to evaluate whole blood and plasma taurine levels or to supplement taurine on a trial basis in all cats with DCM. In patients with taurine deficiency-induced DCM, supplementation with taurine results in clinical and echocardiographic improvement typically within 3 months. While taurine supplementation has been part of the standard treatment for dogs with high-legume diet-associated DCM, taurine deficiency does not appear to be the cause of this condition. Taurine, however, might have benefits aside from deficit replacement, including positive inotropy and anti-oxidation and is safe to administer even in the absence of deficiency. The dosage for taurine supplementation:
  - Dogs: 30 mg/kg PO q12h.
  - Cats: 250 to 500 mg PO q12–24h.
- **Fish oil:** The n-3 polyunsaturated fatty acids levels are reduced in dogs with CHF and fish oil supplementation can normalize these plasma fatty acid abnormalities. A dose for omega-3 fatty acids has been proposed to be 40 mg/kg/day EPA (eicosapentaenoic acid) and 25 mg/kg/day DHA (docosahexaenoic acid) for patients with heart failure. In this 3:2 ratio of EPA:DHA, fish oil appears to have a positive effect on appetite and reduces cardiac cachexia, particularly if started before the development of cachexia. Fish oil supplementation at this dosage is also associated with a decrease in ventricular ectopy in Boxers with arrhythmogenic right ventricular cardiomyopathy.

ANTIARRHYTHMIC MEDICATIONS

- Atrial fibrillation is a common arrhythmia in dogs with advanced heart disease. The onset of atrial fibrillation can precipitate decompensation and clinically evident CHF. In addition to the loss of atrial contraction and its contribution to cardiac output, the rapid ventricular response rate reduces filling time and increases myocardial oxygen consumption. Conversion back to sinus rhythm is not usually pursued because the underlying atrial enlargement predisposes to a high risk of recurrence. The primary goal of treating atrial fibrillation in a CHF patient is rate control. Calcium channel blockers and digoxin are the two most commonly used medications for this purpose.
  - **Diltiazem** is a calcium channel blocker that has primarily cardiac effects. It slows impulse conduction through the atrioventricular node which reduces the ventricular response rate to atrial fibrillation. Although diltiazem has mild negative inotropic effects (reduced contractility), which are undesirable in DCM patients, the benefit of increased filling time associated with rate slowing results in clinical benefit. Diltiazem is also useful for treating other supraventricular tachyarrhythmias.
    - Diltiazem is available in immediate release and slow-release formulations. The dose in dogs of the immediate release formulation is 0.5 to 2.0 mg/kg PO q8h. Slow-release tablets allow for twice daily administration and are dosed at 2 to 6 mg/kg PO q12h. It is important to note that the slow-release formulations are often packaged as a capsule with 60-mg tablets inside and are labeled by the milligrams of the capsule (i.e., a 240-mg capsule contains four

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60-mg tablets inside). It is crucial to communicate the desired dose and instructions to the pharmacist and to provide clear instructions to the owner to avoid overdosing the patient.
- Diltiazem is sometimes administered to cats with HCM for its positive lusitropic (enhanced relaxation) effect; however, this has not been proven to be clinically beneficial. The dose in cats is 7.5 mg/cat PO q8–12h of the immediate release formulation or 45 to 60 mg/cat PO every day of sustained-release formulations.

- **Digoxin** is useful for the treatment of atrial fibrillation (and other supraventricular arrhythmias) because it slows conduction through the atrioventricular node by both direct and vagal effects. It does not usually provide sufficient rate control as monotherapy but is best combined with diltiazem for optimal rate control. Digoxin also partly restores baroreceptor reflexes that are desensitized by the chronically elevated sympathetic tone associated with CHF and it is a positive inotrope, which is beneficial for CHF patients, especially those with DCM. The positive inotropic effect of digoxin is through inhibition of sarcolemmal Na^+/K^+-ATPase, which causes an accumulation of sodium that is then available for exchange with extracellular calcium through the Na^+/Ca^{++} exchanger. The exchange results in increased intracellular calcium which interacts with actin and myosin to improve contractility.
  - Although the use of digoxin for positive inotropy has largely been replaced by pimobendan, it remains a valuable drug for rate control when treating supraventricular tachyarrhythmias.
  - Oral and IV routes of administration are available for digoxin but oral administration is most common.
    i. Dogs are dosed at either 0.22 mg/m^2 PO q12h or 0.002 to 0.005 mg/kg PO q12h
    ii. Cats are dosed at ¼ of a 0.125-mg tablet PO q48h (approximately 0.008 mg/kg/day or every other day).
  - Digoxin has a long half-life (24 hours) so steady state occurs 3 to 5 days after initiation. Serum digoxin levels should be determined 1 week after initiation of therapy to ensure that serum concentrations are not above or below the therapeutic range. The author aims for 6 to 8 hours post-pill digoxin concentrations that are in the lower half of the therapeutic range (under 1.0 ng/mL). Many factors affect the serum levels attained in a particular patient receiving digoxin, making it important to monitor serum concentrations to detect and address toxicity early. Some of these factors include:
    i. Drug form: the elixir is more bioavailable than tablets.
    ii. Route of elimination: patients with renal disease have impaired drug excretion.
    iii. Concurrent medications: drugs such as quinidine and drugs that inhibit hepatic microsomal enzymes (e.g., tetracyclines, chloramphenicol) can increase digoxin serum levels.
    iv. Thyroid status: hypothyroid and hyperthyroid animals often require less digoxin than euthyroid animals.
    v. Body condition: digoxin is not distributed in fat or third spaced fluids; therefore dosage should be based on lean body weight. Obesity, pregnancy, or the presence of ascites require downward dose adjustments.
    vi. Electrolyte abnormalities increase the susceptibility to myocardial digoxin toxicity. In particular, hypokalemia allows more digoxin to bind to the Na^+/K^+ATPase pump, increasing intracellular calcium and the potential for proarrhythmia. Electrolyte status should be monitored periodically as part of routine CHF monitoring, but for this reason, is particularly useful when patients are receiving digoxin.
  - The narrow therapeutic range for digoxin means that signs of toxicity can occur relatively commonly. Starting at the low end of the dosage range, being aware of patient aspects that might increase the risk for toxicity, and close monitoring of blood levels are important for the successful use of digoxin. The signs of toxicity are variable but in general fall into one or more of the three following categories:
    i. Gastrointestinal (inappetence, anorexia, diarrhea, nausea, vomiting). These are usually the first signs of toxicity.
    ii. Cardiac (arrhythmias such as first- and second-degree atrioventricular block, ventricular arrhythmias, and atrioventricular dissociation). A nonrespiratory sinus arrhythmia in a patient receiving digoxin may indicate high serum levels.
    iii. Neurologic (lethargy and depression)
    iv. The treatment of digoxin toxicosis is based on its elimination half-life, and the goal is to return digoxin serum levels to within target range (0.8–1.0 ng/mL). In healthy dogs, the serum half-life of digoxin is approximately 24 to 36 hours. Therefore, it takes about 1½ days to decrease the serum level to half the original level.

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Key Points

A combination of diltiazem and digoxin provides the best heart rate control in atrial fibrillation.

24-Hour Holter monitoring is the most comprehensive method to evaluate heart rate control in atrial fibrillation, with a goal of an average daily heart rate less than 125 bpm.

- Ventricular arrhythmias can occur in dogs with CHF. While clinically significant ventricular arrhythmias are uncommon in dogs with mitral valve disease, they are common in dogs with DCM and appear to increase risk for sudden death. When complex ventricular ectopy is present (e.g., couplets, triplets, ventricular tachycardia) in dogs with CHF, careful consideration to the risk versus benefit of adding antiarrhythmic medications should be made. Although treatment of severe arrhythmias can be lifesaving, all antiarrhythmics have the potential to be proarrhythmic, some antiarrhythmics have negative inotropic qualities (e.g., beta-blockers) which can precipitate CHF decompensation, and the ability of antiarrhythmics to prevent sudden death has not been proven.
  - Mexiletine is a class I antiarrhythmic drug that suppresses ventricular arrhythmias through inhibition of sarcolemmal sodium channels. It does not negatively affect contractility and therefore is a good choice for dogs with DCM.
    - The dose in dogs is 4 to 8 mg/kg PO q8h.
    - Side effects include gastrointestinal upset (giving with food can reduce this possibility) and neurologic signs
    - Cats might have increased susceptibility to the adverse effects of mexiletine because it is the same class as lidocaine and so mexiletine is not generally used in cats
  - Sotalol has mixed class III (beta-blocker) and class II (potassium channel blocker) antiarrhythmic effects. It is a very effective ventricular antiarrhythmic but the negative inotropic effects of beta blockade have the potential to worsen CHF. If it is used in dogs with advanced heart disease or CHF, it should be up-titrated under close observation and combined with pimobendan (possibly with higher doses during initiation of sotalol). Boxers with arrhythmogenic right ventricular cardiomyopathy are often very responsive to sotalol. Myocardial dysfunction is uncommon in this disease making sotalol at good choice for many boxers.
    - The dose in dogs and cats is 1 to 2 mg/kg PO q12h
    - Side effects include weakness, lethargy, hypotension, and worsening of CHF signs.
  - Amiodarone has properties of all four antiarrhythmic classes but is predominately a potassium channel blocker (class III). It is a very effective antiarrhythmic but has greater potential for side effects than the other medications, including thyroid and hepatic dysfunction. It is therefore typically reserved for refractory ventricular arrhythmias.
    i. Other medications are sometimes used as adjunctive therapy in patients with CHF.
  - **Beta-adrenergic receptor blockers** (selective beta-1 agents such as atenolol and nonselective agents such as propranolol or carvedilol) are used to treat specific cardiac conditions. Patients with pathologic concentric hypertrophy (e.g., HCM, aortic or pulmonic stenosis, and tetralogy of Fallot) theoretically benefit from administration of beta-blockers. These patients can have symptoms during exercise because of myocardial ischemia or dynamic outflow tract obstruction, both of which can be improved with beta-blockers. Additionally marked concentric hypertrophy predisposes the myocardium to hypoxia, and tachycardia exacerbates this scenario. Beta-blockers reduce myocardial oxygen consumption by reducing the heart rate. However, studies have not shown a survival benefit for dogs receiving atenolol for either subvalvular aortic stenosis or pulmonic stenosis. Despite some interest in the use of beta-blockers to treat CHF because of their ability to upregulate myocardial beta receptors and improve myocardial function in people and experimental models in dogs, they have not been shown to be beneficial to treat naturally occurring heart disease in the dog. When they are used for this purpose, slow and careful up-titration is crucial because the negative inotropic effects can precipitate CHF signs.
    - Carvedilol, a nonselective beta-blocker with alpha1-blocking and antioxidant effects, used for cardioprotection is started at a dosage of 1.56 mg PO q24h for 1 to 2 weeks, then 1.56 mg PO every 12 hours for 2 weeks, and then titrated upward to the maximal tolerated dosage (0.4 mg/kg PO q12h) over the following several weeks.
    - Atenolol is the most common choice for treating dogs with severe subaortic

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stenosis or pulmonic stenosis. The dose is 0.5 to 1.0 mg/kg PO q12h.
- Atenolol is sometimes used in cats with HCM, especially if they show dynamic left ventricular outflow obstructions. The dose is typically 6.25 mg/cat PO q12h, that is sometimes increased to 12.5 mg/cat PO q12h.
- Propranolol is the beta-blocker of choice for dogs with tetralogy of Fallot because of its nonselective effects. The beta-1 blocking effects reduce dynamic obstruction in the right ventricular outflow tract and the beta-2 blocking effects cause peripheral vasoconstriction which lessen right-to-left shunting. The dose in the dog is 0.3 to 1.0 mg/kg PO q8h.

- **Antithrombotics** are used to prevent thromboembolic complications of heart disease in cats. The risk of thromboembolism in cats increases with left atrial enlargement regardless of the underlying heart disease (most often HCM but similar risk for other cardiomyopathies). Antiplatelet and anti-factor Xa inhibitors are the most common oral medications used for this purpose. Dogs do not require antithrombotic medications because they do not appear to have increased risk for thromboembolic complications with heart disease.
  - Clopidogrel irreversibly inhibits the P2Y subtype of the ADP receptor which inhibits platelet activation and impairs cross-linking by the protein fibrin. Clopidogrel has been shown to prolong the time to a thromboembolic event by 8 months in predisposed cats. Clopidogrel is dosed at 18.75 mg/cat PO q24h. It is best given within a gel capsule to disguise the bitter taste. Because clopidogrel interferes with platelet function for the life of the platelet, it should be discontinued 5 days before elective surgery or dentistry. Additionally it is prudent to limit venipuncture to peripheral veins that can be bandaged after sampling.
  - Oral factor Xa inhibitors (e.g., rivaroxaban) are also used for their anticoagulation properties for cats at risk of thromboembolic disease. The dose of rivaroxaban is 2.5 mg/cat PO q24h. This is sometimes added to clopidogrel in high-risk cats where it has shown to have synergy with clopidogrel.
  - Low-molecular-weight heparins (e.g., dalteparin) are sometimes used to reduce the risk of thromboembolism for at-risk cats but their expense, need for subcutaneous administration, and lack of supportive evidence for dosing frequency make these drugs an uncommon choice. If used, they are typically added to clopidogrel in a very high-risk cat or to bridge the withdrawal time for clopidogrel prior to elective surgery. The dose of dalteparin is 100 U/kg subcutaneously q8-24h and enoxaparin is 1 to 1.5 mg/kg subcutaneously q12–24h.
  - Aspirin and warfarin are no longer recommended for thromboembolism prevention in cats.
- **Sildenafil** is a PDE-5 inhibitor that selectively reduces pulmonary vascular resistance. Because its effects are limited to the pulmonary vasculature, it avoids the side effect of systemic hypotension while targeting the pulmonary vasculature. By lowering pulmonary arterial resistance and pressure, sildenafil improves signs of exertional syncope and can resolve pulmonary hypertension-induced pulmonary infiltrates. It is effective for precapillary pulmonary hypertension as a result of congenital heart disease, chronic pulmonary disease, heartworm disease, and possibly congenital heart disease with Eisenmenger physiology (left-to-right shunt that results in pulmonary hypertension and subsequent right-to-left shunting). The use of sildenafil to treat postcapillary pulmonary hypertension (caused by left-sided heart disease such as mitral regurgitation or DCM) is controversial. Some clinicians avoid it when significant left-sided heart disease is present because reducing pulmonary vascular resistance can increase pulmonary blood flow to the left atrium which already has elevated pressures and therefore this approach the potential to worsen CHF.
  - The dose in dogs and cats is 2 to 3 mg/kg PO q8–12h.
- **Theophylline** is a nonselective PDE inhibitor that causes bronchodilation. Inhibiting PDE results in the accumulation of AMP, which causes increased calcium influx and smooth muscle relaxation and bronchodilation. Many dogs with mitral valve disease also have airway disease and so bronchodilation can be helpful to control the signs of cough. In addition, the change in calcium ion movement in other tissues, such as nodal and myocardial tissue, results in a positive chronotropic and inotropic effect. This agent may be helpful in dyspneic patients with fatigue of the muscles of respiration. Dogs and cats can experience

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transient gastrointestinal disturbance (usually self-limiting and resolving within the first 2 weeks of drug administration), tachycardia, and hyperexcitability and/or restlessness. Occasionally, the positive chronotropic effects (increased heart rate) of theophylline is useful for dogs with bradyarrhythmias (e.g., atrioventricular block or sinus node dysfunction) that are not candidates for permanent pacemaker implantation, especially if they exhibit some atropine responsiveness.
  - The dose of theophylline in dogs is 5 to 10 mg/kg PO q12h.
  - The dose of theophylline in cats is 25 to 50 mg/cat in the evening.
- **Cough suppressants** are often necessary to reduce the frequency of coughing in dogs with left mainstem bronchial compression secondary to left atrial enlargement. Many older dogs with mitral regurgitation also have chronic lower airway disease and so specific treatment for that (including theophylline and sometimes anti-inflammatory doses of prednisone) might be needed to control cough.
  - Hydrocodone is an opioid agonist that can be effective for cough suppression. The dose in dogs is 0.2 mg/kg PO q6–12h as needed, but it can be up-titrated to control cough as long as sedation does not occur.
  - Diphenoxylate is an opioid marketed for its antidiarrheal properties but that also is an effective cough suppressant. Unlike hydrocodone, it does not cross the blood-brain barrier and so does not have sedation as a side effect. Constipation can be a side effect. The dose in dogs is 0.2 to 0.5 mg/kg PO q12h.
- **Sedatives** are sometimes needed for dogs and cats with acute CHF. Anxiety associated with dyspnea can increase the work of breathing and so sedation can improve oxygenation while diuretics and vasodilators are instituted. Butorphanol is a mixed opioid agonist-antagonist and is a good choice for animals with advanced heart disease because it is minimally cardiovascularly depressant. The dose is 0.1 to 0.3 mg/kg IV or IM.
- **Electrolyte supplementation** is sometimes necessary in patients being treated for CHF. Electrolyte depletion can be a side effect of diuresis and might be negatively impacted by reduced appetite. Hypokalemia predisposes to weakness and arrhythmias. There are a variety of potassium gluconate formulations available (e.g., gel, tablets, powder). If both hypokalemia and hypochloremia are present, then oral potassium chloride supplementation (tablets) is a good choice. The potassium supplement dose is 0.5 to 1.0 mEq/kg/day PO or guided by serum concentrations.

SPECIFIC TREATMENT PROTOCOLS

Concise summaries of medications that are indicated for the treatment of specific diseases are described in the following section. Please see earlier sections for more information regarding details of the medications and their use.

a. The following treatment protocols for treatment of canine CHF are adapted from the 2019 consensus statement by ACVIM Specialty of Cardiology and are expanded here to include the treatment of DCM and other forms of heart disease. When CHF therapy for dogs with chronic valve disease or DCM is considered, it is helpful to think of disease as a continuum from stage A through stage C/D.
   - **Stage A:** These patients are at risk of having heart disease because of genetic predispositions, a family history of heart disease, breed predispositions, or concurrent systemic disease with cardiovascular implications. Examples include Cavalier King Charles Spaniels (at risk for chronic valve disease), Doberman Pinschers (at risk for DCM), and Boxers (at risk for arrhythmogenic right ventricular cardiomyopathy). No therapy is indicated for dogs in stage A; however, managing predisposing conditions such as systemic hypertension might be helpful.
   - **Stage B:** Heart disease is present in stage B dogs, but they do not show clinical signs of heart disease. Client education to increase awareness of signs of CHF (tachypnea, dyspnea, coughing) is important at this stage. Home resting or sleeping respiratory rate monitoring can aid in early detection of progression to CHF. Periodic reevaluation for signs of disease progression and complications are also prudent and might include annual thoracic radiographs for dogs with mitral regurgitation and echocardiograms and thoracic radiographs every 6 to 12 months for dogs with DCM.
     - **Stage B1:** This stage is characterized by the presence of heart disease as detected by echocardiography but the absence of cardiac remodeling (i.e., all cardiac chamber sizes are normal).
       - There currently is no evidence to support treatment with any class of medication at this stage of disease.

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- **Stage B2:** This stage is characterized by the presence of heart disease and cardiac enlargement (e.g., left atrial and left ventricular enlargement).
  - Pimobendan has been shown to delay progression to CHF (stage C) in dogs with degenerative mitral valve disease and DCM supporting its use at this stage. For patients with certain forms of DCM, therapy with pimobendan and benazepril can delay the onset of clinical signs (or progression to stage C).
  - Strong evidence does not exist to support using RAAS inhibitors (e.g., ACEi, spironolactone) in dogs with preclinical mitral valve disease. Some clinicians use RAAS suppression in "late"-stage B2 dogs that have severe left atrial and ventricular enlargement because dogs with advanced heart disease might be more likely to be RAAS activated than dogs with only mild cardiomegaly.
  - ACEi might be indicated for the treatment of preclinical (occult) DCM based on one study that showed delay in the time to CHF onset.
- **Stage C:** Signs of CHF are present in this stage. Most dogs with heart disease that progress to stage C are expected to require CHF medications for the remainder of their life. Reversion back to stage B is rare but can occur if the mitral valve is surgically repaired or if a reversible cause of DCM is diagnosed (e.g., taurine deficiency). Home respiratory rate monitoring is crucial at this stage to ensure CHF control and for early detection of recurrences. Blood pressure, serum biochemical evaluation, and thoracic radiography are routinely evaluated 1 to 2 weeks after treatment modifications and every 3 to 4 months. Stage C can be subdivided into acute or chronic CHF.
  - Acute CHF: Stabilization of patients with acute CHF requiring hospitalization includes:
    i. Oxygen
    ii. IV furosemide
    iii. Pimobendan
    iv. ± sedation (e.g., butorphanol)
    v. ± afterload reduction (i.e., nitroprusside)
    vi. ± additional inotropic support (e.g., dobutamine)
  - Stable CHF: Chronic oral administration of "quad" therapy (utilizing the four standard medications below) is recommended for dogs with stage C heart disease after control of acute CHF signs:
    vii. Loop diuretic (e.g., furosemide or torsemide)
    viii. Pimobendan
    ix. ACEi
    x. Spironolactone
    xi. Some dogs might need adjunctive therapy to address other aspects of their heart disease or concurrent airway disease (e.g., cough suppression, heart rate control, bronchodilators, afterload reduction with amlodipine, electrolyte supplementation)

Key Point

The dosage of furosemide should be adjusted (within a range specified by the clinician) on the basis of the resting/sleeping respiratory rate trend and other clinical signs as monitored by the owners.

- **Stage D:** Dogs with CHF signs that cannot be controlled with standard doses of medications (i.e., "quad" therapy as above and requiring >8 mg/kg/day of furosemide) meet the current criteria for stage D CHF. The definition of refractory CHF might be expanded in the future to include other markers such as urinary sodium concentration and hypochloremia, which have been shown to be associated with more advanced CHF and diuretic resistance. Consideration to the following medications are given for dogs with stage D CHF, in addition to standard CHF therapy as outlined for stage C. Close monitoring of renal values, blood pressure, and radiography are indicated, similar to stage C.
  - Replace furosemide with torsemide
  - Diuretic "stacking" (i.e., add hydrochlorothiazide or acetazolamide especially if hypochloremic metabolic alkalosis is present)
  - Escalate pimobendan dose by increasing the dosage and/or frequency
  - Add amlodipine for afterload reduction
  - Manual centesis if effusions are present
  - Moderate salt restriction
  - Rate control if atrial fibrillation is present
  - Appetite stimulation and electrolyte supplementation as needed

b. **Feline Cardiomyopathy** (see Chapter 8). A combination of echocardiography, thoracic radiography, thyroid evaluation, blood pressure assessment, and ECG are needed to accurately diagnose and define feline heart disease. Secondary cardiac changes are possible as a result of other diseases (e.g., hypertension, hyperthyroidism) but in the absence of these conditions, heart disease in cats is most commonly due to HCM and less commonly due to DCM, restrictive cardiomyopathy, and unclassified cardiomyopathy.

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- *Preclinical cardiomyopathy:* Aside from the benefit of antithrombotic therapy in cats with left atrial enlargement, no other medications have been shown to delay the onset of CHF in cats, regardless of the type of cardiomyopathy. Additionally once the disease has progressed to CHF, treatment is largely the same regardless of cardiomyopathy type. One notable exception is the rare diagnosis of DCM, which should prompt measurement of blood taurine concentrations, taurine supplementation, and pimobendan therapy. There might be specific situations to justify using other medications in preclinical cardiomyopathy in cats, but published literature does not necessarily support clear treatment choices. For example, atenolol is often used in cats with HCM that have a significant left ventricular outflow tract obstruction to reduce the obstruction. Although this can often be accomplished, long-term benefit with this medication has not been demonstrated. Some clinicians use diltiazem in cats with HCM for its lusitropic effect, but there is no evidence to support this as beneficial either. Therefore many cats with occult heart disease are treated only with antithrombotic medications with close monitoring for progression and the onset of CHF.
- *Symptomatic cardiomyopathy:*
  - CHF: Cats with symptomatic CHF typically have pulmonary edema or pleural effusion. It is crucial to avoid stressing cats in CHF with diagnostic testing and so a hands-off approach should be employed whenever possible. The use of cageside ultrasound (POCUS) can be very helpful in quickly coming to a diagnosis of CHF in a low-stress manner so that treatment can be instituted quickly.
    - Furosemide
    - Oxygen in the acute CHF situation
    - Clopidogrel (or other antithrombotic)
    - Some clinicians administer pimobendan to cats with CHF, but the evidence supporting this is mixed with a retrospective study of HCM cats with CHF showing benefit and a prospective study of HCM cats with CHF failing to find benefit. It is important to note that pimobendan is not FDA approved in this species and while some clinicians feel it is helpful in HCM cats with CHF, some consider it to be contraindicated. Clear communication with the owner is needed if pimobendan is used in this situation. On the other hand, pimobendan is indicated for myocardial dysfunction in cats (e.g., DCM).
    - Thoracocentesis should be performed for cats with pleural effusion as their manifestation of CHF.
    - Atenolol typically requires dose reduction or discontinuation once cats progress to CHF because the beta blockade seems to hinder adequate CHF control in some cats.
    - Once CHF has been stabilized and cats are eating and drinking in the home environment, an ACEi and spironolactone can be started.
  - Thromboembolism: Despite the use of, or before the use of antithrombotics, some cats experience arterial thromboembolism. Although definitive treatment can be pursued (e.g., clot dissolution or removal), the survival rates are similar to palliative care. The key aspects of supportive care for the cat with arterial thromboembolism are:
    - Treatment of concurrent CHF
    - Pain control (typically with opioids)
    - Rapid antithrombotic treatment: clopidogrel can be "loaded" by administering a full 75-mg tablet PO followed by ¼ tablet (18.75 mg) PO q24h
    - Concurrent heparin therapy while in hospital (either dalteparin, enoxaparin, or unfractionated)
    - Nursing care (warmth, physical therapy, nutrition)

c. **Pericardial Disease**. The management of chronic pericardial effusion and cardiac tamponade is relatively straightforward. An echocardiogram is performed to confirm the diagnosis of pericardial effusion and to attempt to ascertain if the underlying cause is neoplasia. Even if signs of right-sided CHF are present, diuretic therapy is contraindicated because it further reduces cardiac preload which can worsen hypotensive signs. Pericardiocentesis is the treatment of choice and is performed to relieve the compression on the heart from elevated intrapericardial pressure. If the effusion returns more than once, a pericardiectomy is recommended (see Chapter 12).

d. **Congestive Heart Failure Associated with Chronic Heartworm Disease**. The approach to dogs with CHF associated with chronic heartworm disease involves cage rest, diuretic therapy, pimobendan, and heartworm adulticide therapy. Many dogs with severe heartworm disease have pulmonary hypertension and so sildenafil is indicated to address this. Cage rest for a minimum of 1 week before and 4 to 6 weeks after adulticide therapy is crucial to reduce the potential for worm thromboembolism. Heart failure medications (ACEi, furosemide, spironolactone, pimobendan, and sildenafil) may be discontinued in some patients 4 to 8 weeks after adulticide therapy (see Chapter 10).

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Frequently Asked Questions

WHEN IS ACTIVATION OF THE SNS MALADAPTIVE TO THE CARDIAC HEALTH OF THE ANIMAL? SELECT ONE.

A. In a normal animal that is in a flight-or-fight stress response
B. In an animal with RAAS activation
C. In an animal with early heart failure
D. In an animal with chronic heart failure
(D is the correct answer.)

RATIONALES

A. The activation of the SNS is essential for the animal under stress. With an increase in the heart rate, the cardiac output, and selective vasoconstriction (e.g., to gut) and vasodilation (e.g., to muscles), the animal may engage the cardiovascular system effectively to deal with an acute threat.
B. The acute activation of the SNS results in stimulation of beta-1 adrenergic receptors in the juxtaglomerular apparatus of the kidney. Angiotensin II also causes activation of the SNS, so these two systems are closely intertwined, not maladaptive.
C. Early activation of the SNS in early stages of heart failure helps to maintain:
   - Blood pressure
   - Tissue perfusion
   - Cardiac output

   (How? By increasing venous return to the heart via vasoconstriction of the splanchnic vessels, vasoconstriction of other various vascular beds, and positive inotropic and chronotropic cardiac effects)
D. This is a maladaptive situation because chronic SNS overactivity produces the following:
   - Downregulation and desensitization of beta-1 adrenergic receptors
   - Abnormal baroreflex function
   - An overload of the heart by increasing venous return
   - An increase in myocardial oxygen consumption (by increasing heart rate)
   - Cardiac NE depletion and elevated plasma NE
   - Damage to the myocardium, resulting in potential arrhythmogenesis

HOW ARE NATRIURETIC PEPTIDE LEVELS USEFUL IN THE ASSESSMENT OF HEART DISEASE?

These assays can be an important addition to the diagnostic toolkit for selected heart diseases. Circulating natriuretic peptide levels can be used to differentiate symptoms associated with cardiac disease from those associated with primary pulmonary disease.

Natriuretic peptides are salt and water homeostasis regulators, and they are involved in blood pressure control so changes in these parameters may help us to understand the stage of the cardiovascular condition. Their potential value is as diagnostic and prognostic markers in patients with CHF particularly.

Natriuretic peptide levels are elevated in many disease conditions resulting in expanded fluid volume (e.g., DCM, chronic valvular insufficiency in dogs, and cardiomyopathy in cats).

SUGGESTED READINGS

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Chapter 16

Treatment of Cardiac Arrhythmias and Conduction Disturbances

Marc S. Kraus and Anna R.M. Gelzer

Cardiac arrhythmias are defined as variations of the cardiac rhythm from normal sinus rhythm. Some cardiac arrhythmias are benign and clinically insignificant and require no specific therapy; others may cause severe clinical signs such as syncope or degenerate into malignant arrhythmias (i.e., ventricular fibrillation [VF]), leading to cardiac arrest and sudden death. The goal of this chapter is to discuss treatment strategies for the management of clinically important arrhythmias.

GENERAL REMARKS

Antiarrhythmic drugs (AAD) target two general areas of the heart because of their specific electrophysiologic properties:

- Sinoatrial and atrioventricular (AV) nodal tissue: Depolarization of nodal cells is calcium channel driven. For the treatment of arrhythmias that originate or encompass the sinoatrial or AV nodal tissue, calcium channel blockers (CCBs) and beta-blockers (BBs) are primarily used. The most prescribed CCB for these indications is diltiazem (available orally [PO] and intravenously [IV]). The beta1-selective BBs atenolol and esmolol (IV only) are the most frequently used antiarrhythmic BBs. Sotalol, a BB combined with marked potassium channel-blocking characteristics, is also used for supraventricular arrhythmias involving nodal tissue. Digoxin may also be used as an antiarrhythmic agent for its vagomimetic effects, which can indirectly prolong AV node conduction time. Its antiarrhythmic application is limited to slowing the ventricular response in the treatment of atrial fibrillation (AF). For drug dosages, consult Tables 16.1 and 16.2.
- Atrial or ventricular myocardium: Depolarization is sodium channel driven and repolarization is potassium channel dominated. For treatment of arrhythmias that originate from the atrial and ventricular myocardium, Na channel blockers (NCBs), potassium-channel blockers (KCBs), or combinations thereof are used, sometimes in conjunction with BBs. The NCBs used for treatment of arrhythmias in dogs are lidocaine (IV only), mexiletine, and procainamide. The most important KCB is sotalol, which also has BB properties, as well as amiodarone, which is predominantly a KCB but also has potent NCB and some CCB and BB activity. For drug dosages, consult Table 16.1 and Table 16.2.

Even though a correct diagnosis of an arrhythmia may be obtained by a short in-hospital electrocardiogram (ECG), a 24-hour Holter recording is required in some patients with an intermittent arrhythmia to establish a definitive diagnosis. For optimal long-term management of most arrhythmias, a 24-hour Holter recording should be acquired in addition to the ECG before starting antiarrhythmic therapy.

The decision regarding how and when to treat an arrhythmia should be based on the clinical signs and urgency of intervention. Emergency management that includes IV drugs may be required before a 24-hour Holter recording can be obtained. Both diltiazem and esmolol are available in IV formulations, allowing emergency treatment of excessively rapid supraventricular arrhythmias (SVAs). Lidocaine is the most important IV drug used for life-threatening ventricular arrhythmias.

- The benefits of a 24-hour Holter recording include in-depth assessment of quantity and quality of the arrhythmia(s). The following parameters should be examined: number of abnormal beats (supraventricular or ventricular) relative to overall number of beats, duration and rate of runs of abnormal beats, average hourly and daily heart rate (HR), amount of time when the HR is >120 and <50 beats/min, and presence and length of pauses. A diary kept by the client or hospital staff with the sleep/wakefulness activity or observed events such as syncope or excessive anxiety or panting help correlate ECG changes on the Holter with clinical signs. These parameters are vital for a baseline evaluation of a patient's arrhythmias and needs for antiarrhythmic therapy.
- If AAD therapy is instituted, it is critical to obtain a repeat Holter recording after 1 to 2 weeks to determine if the drugs are efficacious in suppressing the abnormal rhythms or possibly harmful by being proarrhythmic (i.e., causing more arrhythmias or excessive pauses). This can only be established if a comparison to the pretherapy baseline is performed.
- If Holter monitoring shows that a drug is effective, it is recommended to monitor the progression of the arrhythmia by repeat Holter recordings every 6 to 12

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Table 16.1 Agents for Rate Control/Abolishing Arrhythmias in Canine Patients With Arrhythmias

DRUG	ORAL ADMINISTRATION	INTRAVENOUS ADMINISTRATION	INDICATION
Diltiazem XR (Dilacor)	3–5 mg/kg BID		AF, AFL, AT, OAVRT
Diltiazem	0.5 mg/kg TID titrated up (max 1.5–2.5 mg/kg TID)	0.1–0.2 mg/kg bolus, then CRI 2–6 μg/kg/min	Acute AF, AFL, OAVRT
Atenolol	1–2 mg/kg SID to BID		AFL, AT, OAVRT, VT
Esmolol		50–100 μg/kg bolus (repeat up to max 500 μg/kg); 50–200 μg/kg/min CRI	Acute AF, AFL, AT, OAVRT, VT
Sotalol	1–2.5 mg/kg BID		AF, AFL, AT, VT
Digoxin	0.003 mg/kg BIDLiquid suspension available for small dogsMax dose for Dobermans: 0.25 mg BID		AF
Procainamide		10–15 mg/kg IV bolus slowly over 2 minutes; if needed start a CRI: 25–50 μg/kg/min	AT, VT
Amiodarone	5–15 mg/kg BID for 1–2 weeks (loading dose)[a] 6–15 mg/kg SID (maintenance dose)[a]		AF, AT, VT
Lidocaine		2 mg/kg IV bolus, repeat 3 times, if needed start a CRI: 30–80 μg/kg/min	VT, AF if because of narcotics
Mexiletine	4–8 mg/kg TID		VA, VT, usually not effective as monotherapy

AF, Atrial fibrillation; *AFL*, atrial flutter; *AT*, atrial tachycardia; *BID*, twice daily; *CRI*, continuous-rate infusion; *OAVRT*, orthodromic atrioventricular reciprocating tachycardia; *SID*, once daily; *TID*, three times daily; *VA*, ventricular arrhythmias; *VT*, ventricular tachycardia.
[a]Recommended dose range in veterinary medicine is anecdotal and variable.

Table 16.2 Agents for Rate Control/Abolishing Arrhythmias in Feline Patients With Arrhythmias

DRUG	ORAL ADMINISTRATION	INTRAVENOUS ADMINISTRATION	INDICATION
Diltiazem XR	30–60 mg SID or BID (start with 30 mg SID)		AF, AT, accessory pathway
Diltiazem	10 mg/kg SID	0.1–0.2 mg/kg bolus, then CRI at 2–6 μg/kg/min	Acute AF, accessory pathway
Atenolol	6.25–12.5 mg SID or BID		AT, accessory pathway, VT
Esmolol	NA	50–100 μg/kg bolus (repeat up to max 500 μg/kg); 50–200 μg/kg/min CRI	Acute AF, AT, accessory pathway, life-threatening VT
Sotalol	1–2.5 mg/kg BID (1/8 of an 80-mg tablet BID[a])		VT
Procainamide	2–5 mg/kg BID or TID	10–15 mg/kg IV bolus slowly over 2 minutes or CRI at 25–50 μg/kg/min	AT, accessory pathway
Lidocaine	NA	0.25–1 mg/kg IV bolus (use with extreme caution in cats)	Life-threatening VT

AF, Atrial fibrillation; *AT*, atrial tachycardia; *BID*, twice daily; *CRI*, continuous-rate infusion; *NA*, not available; *SID*, once daily; *TID*, three times daily; *VT*, ventricular tachycardia.
[a]Recommended dosage range is anecdotal and variable.

months, depending on the severity of the arrhythmia. If an animal experiences recurrent syncope during that time, a repeat Holter is recommended.

- In animals with life-threatening ventricular arrhythmias that need to be stabilized immediately, it may not be advisable to first obtain a baseline 24-hour Holter recording, before initiating an AAD. However, it is still useful to evaluate the rhythm once the animal is receiving chronic oral antiarrhythmic therapy.

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TREATMENT OF SUPRAVENTRICULAR ARRHYTHMIAS

Supraventriular arrthmias (SVAs) include rhythms that originate in the sinus node, atrial tissue, and AV junction. Importantly, SVA must be differentiated from accelerated normal sinus rhythm. Physiologic sinus tachycardia can be caused by many conditions, including fever, hypotension, hypovolemia, anemia, heart failure, adrenergic medications, and anxiety. In these cases, management should be focused on correcting the underlying cause or disease resulting in increased sympathetic tone.

- Because of the mechanism of action of AADs primarily on either nodal tissue versus myocardium/Purkinje fibers, it is useful to assess SVA as either AV node *independent* (does not need the AV node to sustain the rhythm) or AV node *dependent* (requires the AV node to sustain the rhythm).
- An SVA is AV node independent if the ECG contains P waves not conducted to the ventricle, without termination of the SVA. Interventions such as vagal maneuvers or drugs that slow AV node conduction can help identify the underlying mechanism:
  - If the atrial activation rate (PP interval) is unchanged following the intervention but the ventricular rate slows because of AV block, the SVA is likely AV node independent.
  - If the intervention abolishes the SVA and restores normal sinus rhythm (even if only transiently), the arrhythmia is likely AV node dependent.
- Examples of AV node–independent rhythms include sinus node reentrant tachycardia, atrial fibrillation (AF), atrial flutter (AFL), and focal atrial tachycardia (FAT). These arrhythmias can be challenging to manage because the AADs (NCBs, KCBs, or combinations thereof) are often not very efficacious for termination of these arrhythmias. If the abnormal rhythm cannot be abolished with drugs, the secondary strategy of treatment aims at controlling the ventricular response rate by slowing the AV node conduction with CCBs, BBs, or digoxin, while accepting that the atrial arrhythmia continues.
- AV node–dependent SVAs include AV reentrant tachycardia (accessory pathway [AP] mediated) and AV nodal reentrant tachycardia (not documented in animals). AV node–dependent arrhythmias are usually treated with drugs that prolong the AV node conduction (CCBs and BBs) or, less commonly, that slow conduction in the AP (NCB, KCB), resulting in termination of the reentrant circuit.

Key Point

To help guide treatment of SVAs, they should be characterized as AV node dependent or AV node independent.

ATRIAL FIBRILLATION

- Atrial fibrillation (AF) is the most common SVA in veterinary practice. In dogs and cats, AF is usually a chronic arrhythmia associated with advanced stages of chronic AV valve insufficiency or cardiomyopathy. In these patients, the ventricular response rate is often markedly elevated, which contributes to the clinical signs of heart failure. AF can also occur in the absence of overt structural heart disease (lone or primary AF). In these cases, the ventricular response rate can be normal or only mildly elevated.

Key Point

The management of AF largely depends on the average HR. A baseline 24-hour Holter recording acquired in the home is ideal to determine the average HR of a patient.

Therapy of atrial fibrillation

- AF is an AV node–independent arrhythmia, caused by multiple simultaneous intraatrial reentrant circuits. Two strategies exist for management of AF: (1) rhythm control, which entails termination of AF and restoration of sinus rhythm, either medically or by electrical cardioversion. Dogs with normal cardiac function or only mild dysfunction and normal to mildly elevated ventricular response rates may be good candidates for rhythm control strategy, but rates of recurrence of AF are high. (2) Rate control, where it is accepted that AF persists, but the ventricular response rate is modulated by AAD that slow AV node conduction velocity. This strategy is most common in veterinary medicine, as the underlying heart disease is often advanced and rate control can be administered on an outpatient basis.

  Rate control: Slowing of AV node conduction is achieved with either a CCB (diltiazem) or a BB, often combined with digoxin (drug dosages are listed in Table 16.1). Due to the presence of systolic dysfunction in many patients with AF, we prefer to use diltiazem over a pure BB, as it is well tolerated, even in dogs with severe systolic dysfunction.

  It is recommended to attempt to decrease the 24-hour Holter-derived mean HR to <125 beats/min. This is based on the results from a prospective study (ORCA), which demonstrated that a mean HR of <125 beats/min conveyed a significantly longer median survival (608 days), compared to only 33 days in dogs with a mean HR >125 beats/min. In our experience, diltiazem XR (as the extended-release brand Dilacor)—at a starting dose of 3 mg/kg q12h—combined with digoxin 0.003 mg/kg PO q12h is a useful strategy to achieve a 24-hour mean HR of <125 beats/min. Digoxin monotherapy does not control the ventricular response rate adequately during times of excitement, stress, or exercise. Diltiazem

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monotherapy may be adequate in dogs with a HR that is only moderately elevated. The 240-mg extended-release capsule can be opened to access the four inner tablets at 60 mg each, for more accurate dosing. If the 24-hour mean HR remains >125 beats/min, diltiazem XR can be titrated upwards to 5 mg/kg q12h, while digoxin should not be increased.

- AF occurs rarely in cats: There is usually significant underlying heart disease present, resulting in markedly enlarged atria and very rapid AF. Rate control with a target HR of 130 to 150 beats/min may be achieved with either a CCB or BB (for drug dosages for antiarrhythmic drugs in cats, see Table 16.2).

Key Point

Heart rate during AF affects survival; a target mean HR <125 beats/min should be sought in dogs with AF.

RHYTHM CONTROL

Medical conversion of chronic AF to sinus rhythm with AAD is rarely achieved in dogs, but both amiodarone and sotalol have been reported to result in medical cardioversion.

- Occasionally, the administration of opioids has been associated with the induction of AF in large dogs. This is likely caused by the increased vagal tone that occurs with opioids. Treatment with 2 mg/kg **lidocaine** IV within 4 hours of AF onset has been demonstrated to restore sinus rhythm. Vagolytic drugs (atropine) should prevent onset or recurrence of AF in such cases.

Electric Cardioversion

- In dogs with AF, but normal hearts or only mild structural heart disease (primary or lone AF), electrical cardioversion of AF to sinus rhythm can be achieved. The goal of cardioversion is to avoid structural or functional remodeling from chronic AF, even if the HR during AF is slow. The rate of recurrence of AF after successful cardioversion is high, and the morbidity associated with repeat transthoracic cardioversions under general anesthesia makes this management less practical. Initial treatment with amiodarone is recommended to (1) attempt pharmacologic cardioversion, (2) reduce risk of AF recurrence immediately postcardioversion; however, no studies in veterinary medicine have proven these concepts.

Transthoracic electrical cardioversion procedure

- Procedure requires a brief general anesthesia: Propofol: 1 mg/kg IV; Etomidate 0.7 mg/kg (two times possible), Midazolam 0.3 mg/kg IV.
- "Fast Patch" electrodes or handheld paddles can be used for cardioversion.
- Dog is shaved before application of patch electrodes or paddles over the heart on both lateral sides of the thorax.
- Dog is positioned in lateral recumbency to optimally "position" the heart between the two patch electrodes or paddles.
- The defibrillator must have the ability to synchronize to the R waves. Defibrillator ECG cables need to record the patient's ECG to determine if synchronization to the R wave is accurate. Without synchronization or false synchronization to T waves (occurs if the T wave is taller than the R wave), the cardioversion shock can induce VF.
- Using a biphasic defibrillator (recommended): Start with 0.5 to 2 J/kg; if no cardioversion occurs, increase the dose by 50 J and repeat until a max of 360 J.
- Using a monophasic defibrillator: Start with 4 J/kg; if no cardioversion occurs, increase dose by 50 J and repeat until a maximum of 360 J.
- Short, transient runs of ventricular tachycardia, sinus pauses, or AV block are common following electric cardioversion.

ATRIAL FLUTTER

- Atrial flutter (AFL) is relatively uncommon in veterinary patients. Theoretically AFL could set the stage for development of AF because of the remodeling that occurs with continuous rapid activation of the atrial myocardium. In some patients, AFL coexists with AF on a 24-hour Holter, which might represent a transition phase to chronic AF. AFL is an AV node–independent intraatrial macroreentry rhythm. The atrial activation rate (PP interval) is 300 to 600 beats/min. AFL is paroxysmal or chronic and can be associated with excessively high ventricular response rates. AV conduction usually changes between 1:1 and 3:1 or 2:1 caused by variable degrees of AV block.
- A baseline 24-hour Holter is recommended to determine if the arrhythmia is chronic or paroxysmal. If it is chronic, therapy is indicated.
- If it is paroxysmal and infrequent, treatment may be postponed, but a reevaluation by Holter 6 months later should be performed to check for progression from paroxysmal to chronic AFL or presence of concurrent AF.

Therapy of Atrial Flutter

- Ideally treatment is aimed at abolishing the reentry circuit, which can be achieved with catheter ablation therapy. Catheter ablation is a procedure that uses radiofrequency energy (RF) to destroy a small area of heart tissue that is either in the pathway of the reentrant circuit or a focal area which is the origin of an arrhythmia. Catheter ablation destroys the abnormal tissue without damaging the rest of the heart. This procedure can be performed only at

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specialized electrophysiology laboratories, which are available at only few veterinary referral centers.
- Therefore medical management is usually pursued. Pharmacological slowing of the atrial reentry circuit using sotalol, amiodarone, or procainamide may also terminate the arrhythmia; however, abolishing the AFL with drugs is often unsuccessful. Propafenone and flecainide are used in humans with AFL, but we have had little success with these drugs as the optimal dose for dogs has not been established. Therefore often it is more effective to treat this arrhythmia with a rate control strategy, similar to treatment of AF, with CCBs or BBs (for drug dosages, see Table 16.1). Digoxin monotherapy is ineffective for management of AFL.
- A posttreatment 24-hour Holter should be obtained to determine if the therapy was effective at suppressing the AFL or producing the desired degree of AV block, thus slowing the ventricular response adequately. It also allows assessment of possible drug toxicity, which may manifest as bradycardia or pauses secondary to excessive AV block. Pauses, if they occur only during sleep or rest, are usually of no concern.

FOCAL (ECTOPIC) ATRIAL TACHYCARDIA

- Focal atrial tachycardia (FAT) occurs when ectopic foci in the atria develop the ability to fire rapidly and spontaneously. It is an AV node–independent arrhythmia. FATs arise from a single site within the left or right atrium, in contrast to macroreentrant atrial arrhythmias (e.g., AFL) and AF, which involve multiple sites or larger circuits. FAT is often paroxysmal and may gradually speed up and slow down (warm-up and cool-down period). This finding suggests that the underlying mechanism involves enhanced automaticity. However, FAT may have automatic, triggered, and microreentrant mechanisms that cannot be distinguished easily on the surface electrocardiogram. P-wave morphology may be similar to that in sinus tachycardia, or vary, depending on the site of origin. The RP interval is typically longer than the PR interval. The PR interval can vary in FAT, and the HR can vary from 150 to 300 beats/min, which can cause anxiety or panting, and restlessness in affected dogs. Syncope is rarely reported. If the arrhythmia burden is high, FATs can result in tachycardiomypathy and congestive heart failure. In cats, FAT is rare.
- A baseline 24-hour Holter should be obtained to determine what percentage of time a dog is in FAT (arrhythmia burden), and how fast the HR is during the tachycardia.

Therapy of Focal Atrial Tachycardia

- Ideally treatment should be aimed at abolishing the arrhythmia to preserve myocardial function using RF ablation therapy. Because this type of interventional therapy is not readily available to veterinary patients, medical management is usually the treatment of choice. Suppression of the FAT is attempted by using sotalol, amiodarone, or procainamide. In people, propafenone is used for treatment of FAT, but no studies in dogs have established efficacy of this agent. In fact, targeting FAT directly with these drugs is often unsuccessful. Thus rate control is also commonly used to treat FAT. Digoxin is ineffective for management of FAT.
- A posttreatment 24-hour Holter should be obtained to determine if the therapy was effective at suppressing the FAT or slowing the ventricular response adequately if rate control was pursued. It also allows assessment of bradycardia or pauses secondary to excessive AV block. Pauses, if they occur only during sleep or rest, are usually of no concern.



Key Point

Although amiodarone is a potent antiarrhythmic drug, its benefits must be balanced against its slow onset to action and adverse effects, which include hepatic toxicity, gastrointestinal disturbances, and blood dyscrasias in canine patients.

ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIA

- AV nodal reentry tachycardia (AVNRT) is a form of microreentry within the AV node that gives rise to rapid, simultaneous activation of the ventricles and atria. AVNRT is AV node dependent; thus AV node–slowing medication may terminate the arrhythmia. Thus far, AVNRT has not been reported in dogs.
- For treatment of AVNRT, see the following discussion of treatment of atrioventricular reentrant tachycardia (AVRT).

ATRIOVENTRICULAR REENTRANT TACHYCARDIA

- Atrioventricular reentrant tachycardia (AVRT) is a macroreentry arrhythmia whose circuit comprises the AV node as well as an accessory (AP) that can conduct impulses from the atria to the ventricles directly, thereby bypassing the AV node and His-Purkinje system. Response to a vagal maneuver or IV drug challenge (diltiazem IV) may be used to confirm its AV node–dependent properties. One-to-one AV association is a requisite of AVRT because the atria and ventricles are both integral parts of the arrhythmia circuit. If AV dissociation occurs spontaneously without termination of the SVA, AVRT can be excluded.
- AVRT may be a paroxysmal, intermittent SVA that can result in very regular, rapid HR up to 300 beats/min. In some animals, it can be sustained over several hours and thus result in cardiac dysfunction (tachycardiomyopathy) and heart failure. Typically,

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a 24-hour Holter may be required to make a definitive diagnosis of this SVA, as well as determine the arrhythmia burden. Animals with AVRT may be symptom free or may show signs of syncope, episodic weakness, or lethargy.

- In dogs, the accessory pathway is usually able to conduct in only one direction. Most APs in dogs allow conduction in the retrograde direction (from the ventricles to the atria). An arrhythmia occurs when an impulse from the atria passes through the AV node to the ventricles and then travels retrogradely over the AP back up to the atria. This form is prevalent in Labrador Retrievers and is termed orthodromic AV reciprocating tachycardia (OAVRT).
- Most APs in dogs do not allow conduction in the antegrade direction (from atria to the ventricles) (concealed conduction) and are therefore not identifiable during normal sinus rhythm. In cats with AVRT, however, the AP may conduct in both directions so that ventricular preexcitation is present during normal sinus rhythm. Preexcitation occurs when the atrial depolarization travels through both the AV node as well as the AP, which has a faster conduction velocity, thus allowing earlier activation of the ventricles. The ECG morphology of a P-wave slurring directly into the upstroke of the QRS complex (delta wave) is a distinct feature of antegradely conducting AP. The presence of delta waves may create the appearance of wide-complex tachycardia and thus resemble VT.

Therapy of Atrioventricular Reentrant Tachycardia

- Ideally, treatment should be aimed at abolishing the arrhythmia to preserve myocardial function using RF ablation therapy. Because this type of interventional therapy is not readily available to most veterinary patients, medical management is usually the treatment of choice. Pharmacological slowing of the AVN conduction or the AP may terminate the arrhythmia.
  - Treatment with oral diltiazem or atenolol is the first choice for termination of AVRT or in dogs and cats. For acute management, IV diltiazem or esmolol is effective. For refractory cases, sotalol, procainamide, or amiodarone may be added because any of these drugs contribute to slowing of conduction of the AVN, and also the AP directly.
  - A posttreatment 24-hour Holter should be obtained to determine if the therapy was effective at terminating the AVRT. It also allows assessment of possible drug toxicity, which may manifest as bradycardia or pauses secondary to excessive AV block. Pauses, if they occur only during sleep or rest, are usually of no concern. Some APs in dogs with OAVRT are capable of antegrade conduction if the AVN conduction is slowed, and thus a delta wave may appear on the surface ECG after AAD administration.

BRADYARRHYTHMIAS AND CONDUCTION DISTURBANCES

The bradyarrhythmias that require treatment are usually due to sinus node dysfunction (e.g., sinus bradycardia or sick sinus syndrome [SSS]), atrial conduction disturbances causing atrial standstill, or AV node conduction abnormalities causing high-grade second, or third-degree AV block (3AVB). Ventricular conduction disturbances such as left and right bundle branch block as well as left anterior fascicular block do not warrant treatment, per se. The most common clinical signs include lethargy or syncope. Medical therapy of bradyarrhythmias is generally not very effective. For patients experiencing syncope, pacemaker therapy is considered optimal.

- Sinus bradycardia is diagnosed when the sinus node discharge rate is low (<50 beats/min) in an awake dog. Sinus bradycardia of 40 to 60 beats/min during sleep is normal. Sinus bradycardia can exist in the form of a pronounced sinus arrhythmia or regular sinus bradycardia. When pathologic, sinus bradycardia often persists during excitement or exercise. The bradycardia is either primary, which is a form of SSS (see next section), or secondary to an underlying systemic disease or drug toxicity (e.g., opioids or overdosing of BBs, CCBs, or digoxin).
- Secondary sinus bradycardia is usually caused by excessive vagal tone elicited by a systemic disease. Central nervous system disease (increased intracranial or intraocular pressure or head trauma), severe pain, or respiratory or gastrointestinal disease can all cause increased vagal tone and sinus bradycardia. A vagal maneuver (e.g., carotid sinus massage) can cause transient sinus bradycardia by the same mechanism of action. Correction of the underlying condition or discontinuation of drugs will usually resolve secondary sinus bradycardia.
- Clinical signs may be absent (incidental finding) or dogs may display weakness, exercise intolerance, or syncope. A 24-hour Holter may be required to determine the severity of bradycardia and possible association of clinical signs with the slow HR. Mild exercise intolerance is often underrecognized by owners and mistakenly attributed to "old age."
- In cats, a sinus rhythm at less than 120 beats/min can be considered bradycardia, and HRs below 100 beats/min are often associated with lethargy or syncope in cats.

Treatment of Sinus Bradycardia

- The decision to treat sinus bradycardia should be based on clinical signs and the degree of bradycardia. In patients experiencing syncope or episodic weakness, pacemaker therapy is indicated.

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- In an animal with no clinical signs, sinus bradycardia may be "waited out" with close monitoring.
- If the animal appears unstable and pacemaker therapy is not an option, medical therapy aimed at abolishing high vagal tone or enhancing sympathetic tone can be attempted for temporary support. An atropine response test may help identify patients that would benefit from such medical management. After injection of atropine 0.04 mg/kg (0.01–0.04) IM or IV, the baseline HR should increase by 50% to 100% within 5 to 10 minutes (initial worsening bradycardia caused by AV block is a normal, transient response). Patients experiencing at least a partial response to atropine may be candidates for medical management of sinus bradycardia. Treatment options include either a combination of a vagolytic drug (e.g., **propantheline bromide** 0.25–0.5 mg/kg PO BID or **hyoscyamine sulfate** 0.002–0.004 mg/kg PO BID) or a phosphodiesterase inhibitor (e.g., **theophylline** 5–15 mg/kg PO BID). Alternatively, a sympathomimetic drug could be tried as well (e.g., **albuterol** 0.02–0.05 mg/kg PO BID or TID).
- Poor efficacy, as well as adverse effects, such as anxiety, excessive panting, anorexia, or gastrointestinal signs, are significant disadvantages to these therapies.

SICK SINUS SYNDROME

- The spontaneous sinus node discharge is either slower than normal (primary sinus bradycardia) or intermittently absent (sinus arrest or exit block from the sinus node). In the latter, there are pauses of various durations without P waves or an escape rhythm. The subsidiary pacemaker tissue (AV node and Purkinje fibers) is often also abnormal, resulting in inadequate escape rhythms, such that complete asystole (pauses) can last up to 10 seconds. Some cases of SSS have "brady-tachy" syndrome; in addition to sinus pauses, supraventricular tachyarrhythmias (e.g., atrial tachycardia, flutter, or fibrillation) can be present. Miniature Schnauzers (female), Cocker Spaniels, West Highland White Terriers, and Dachshunds are overrepresented. Doberman Pinschers and Boxers are also reported to have syncope associated with long sinus pauses, suggestive of SSS.
- Clinical signs range from exercise intolerance and lethargy to syncope. In Miniature Schnauzers, West Highland White Terriers, and Dachshunds, SSS more commonly manifests with long sinus pauses and syncope, whereas in Cocker Spaniels, SSS often manifests as regular sinus bradycardia, with only mild exercise intolerance, which is often underrecognized by owners and mistakenly attributed to "old age." SSS may be incidentally diagnosed during a routine preanesthesic evaluation in a geriatric dog. If these dogs are treated with a pacemaker, owners often are delighted by the return of youth and energy in their "old dogs."
- Because of the intermittent nature of the sinus pauses in some cases, a 24-hour Holter is often necessary to definitively determine the cause of clinical signs.

Treatment of Sick Sinus Syndrome

- If the animal does not have syncope and shows only mild clinical signs, medical management can be attempted (see medical treatment of sinus bradycardia). In dogs with syncope, medical management is inadequate, and pacemaker therapy is always recommended.
- Mild forms of SSS (i.e., no syncope yet) tend to progress over weeks or months to the point of syncope, and pacemaker implantation is eventually required.
- Pacemaker therapy is indicated for syncopal or lethargic dogs with SSS. In dogs with intact AV node function, transvenous placement of a pacing lead in the right atrium or auricle may successfully abolish the sinus pauses. However, patients with SSS often have concomitant conduction disease in subsidiary pacemaker sites and may have AVN block later in the disease. In dogs with suspected concomitant AV node dysfunction, a single right ventricular lead should be placed or dual-chamber pacing performed In patients with brady-tachy syndrome, a 24-hour Holter should be obtained to determine the clinical relevance of the tachy episodes and need for antiarrhythmic therapy. In many cases, however, SVA spontaneously resolves once the long sinus pauses are prevented by the pacemaker.
- Therapy for tachycardias of any nature in a SSS dog should only be considered once pacing is established, to avoid worsening of bradyarrhythmias. Antiarrhythmic therapy may be necessary if the SVA persists after pacemaker implantation.

ATRIAL STANDSTILL

- Atrial standstill occurs when the atrial myocardium is not able to depolarize, and P waves cannot be identified on the surface ECG. The two main causes are (1) persistent atrial standstill or "silent atrium" caused by primary atrial muscle disease and (2) secondary atrial standstill caused by hyperkalemia (i.e., renal failure, ruptured bladder, Addison disease, or other electrolyte imbalances). Hyperkalemia alters atrial transmembrane resting potential, and the atria become inexcitable at very high plasma potassium^{+} concentration. However, in this scenario, the sinus node continues to fire, albeit at a slower rate, and the ensuing rhythm is called sinoventricular rhythm. It is reversible with correction of K concentration.
- Persistent atrial standstill is uncommon in dogs and is irreversible. Initially, dogs may have a junctional escape rhythm (HR 60–80 beats/min). However, some dogs have progressive conduction system disease (AV junction and His-Purkinje system) and eventually develop severe bradycardia (ventricular

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escape rhythm of 20–40 beats/min) and also myocardial failure (cardiomyopathy). Atrial standstill is exceedingly rare in cats.

Treatment of Atrial Standstill

- If atrial muscle disease is causing atrial standstill, pacemaker therapy is required. Because the atria are structurally abnormal, the pacing lead must be placed in the right ventricle. Unfortunately the primary cardiac muscle disease may progressively also affect the ventricles. Over time, ventricular myocardial dysfunction and significant AV valve regurgitation may develop, and pacemaker failure may ensue because of lack of capture. However, many dogs can survive several years with this condition after pacemaker implantation (median survival time 866 days in one study, with one dog is reported to have lived 10.5 years).
- For atrial standstill secondary to hyperkalemia, emergency therapy according to the severity of the hyperkalemia and bradycardia is required. Intravenous fluids are the primary treatment. This will lower the potassium values by dilution and increased excretion. Acceptable fluids include normal saline, half-strength saline with 2.5% dextrose, or 5% dextrose in water. Alternatively, potassium can be lowered by promoting entry of potassium ions back into the intracellular space. The dextrose in fluid therapy will lead to insulin secretion that will drive the potassium back into the cells. More aggressive therapy involves IV **sodium bicarbonate** (1–2 mEq/kg IV slowly over 20 minutes with ECG monitoring) to drive potassium back into the cell. Alternatively, slow IV administration of 0.25 to 0.5 U/kg of regular insulin IV coupled with IV dextrose 1 g of 50% dextrose per unit of insulin (diluted 1:1) can be administered. Monitoring for hypoglycemia is required after this treatment. If hyperkalemia is due to hypoadrenocorticism, the insulin dose should be reduced to 0.25 U/kg as these patients are predisposed to hypoglycemia. **Calcium gluconate** (0.5–1.0 mL/kg of a 10% solution) may be given by very slow IV to counter the effects of hyperkalemia. This has no effect on the actual potassium concentration. This is "cardioprotective" because the increased extracellular Ca^{2+} makes more sodium channels available for activation.

ATRIOVENTRICULAR CONDUCTION ABNORMALITIES

Atrioventricular Block, First Degree

- Prolonged conduction time through the AV node results in an increased PR interval of greater than 0.13 second in dogs and greater than 0.09 second in cats. There are normal P waves and QRS complexes, conducting at a 1:1 ratio. No treatment is required.

Atrioventricular Block, Second Degree

- There are normally conducted P waves and QRS complexes, but intermittently P waves are blocked (i.e., not followed by a QRS complex). In a Mobitz type I (Wenckebach) AV block, the PR interval gradually prolongs before a P wave is blocked. A Mobitz type II AV block demonstrates a consistent PR interval before a blocked P wave occurs. A Mobitz type II AV block may represent a more advanced degree of conduction abnormality that occurs in the AV junction, His bundle, or below. Occasional single blocked P waves are of no clinical significance and may be attributed to high vagal tone.
- In "high-grade" second-degree AV block, there are several consecutive blocked P waves. Clinical signs depend on the number of blocked Ps (length of ventricular asystole). In cases with intermittent high-grade second-degree AV block, a 24-hour Holter may be required to make a definitive diagnosis. If clinical signs such as lethargy or syncope are observed, pacemaker therapy is indicated.

Atrioventricular Block, Complete or Third Degree

- None of the P waves conduct through the AV node; thus the atrial and ventricular activities are independent (complete AV dissociation). The atrial rate (PP interval) is faster than the ventricular (escape) rate, differentiating complete AV block from AV dissociation caused by accelerated idioventricular rhythms. The escape rhythm is usually regular and the rate ranges from 20 to 60 beats/min, depending on the origin (AV junction or ventricle). In cats with complete AV block, the ventricular escape rhythm varies from 60 to 120 beats/min.
- Complete AV block is a primary abnormality of the AV conducting system (AV node). However, it is important to evaluate the animal's electrolyte and acid-base status. Systemic diseases causing hyperkalemia, such as Addison disease or urethral obstruction, can cause an AV block that is reversible with normalization of K levels.
- In cats, hyperthyroidism can cause significant AV node disease, which may or may not be reversible with normalization of thyroid levels. A third-degree AV block is often not as life threatening in cats as in dogs. Cats with collapse episodes may live for longer than 1 year without pacemaker implantation and often succumb to other systemic diseases or structural heart disease rather than the actual bradyarrhythmia.

Treatment of Complete Atrioventricular Block

- If no underlying abnormalities are discovered, a permanent cardiac pacemaker is the only effective treatment. Pacing systems that allow sensing of spontaneous P waves in the atria and subsequent timed pacing of the ventricles may be ideal (i.e., dual-chamber or single-lead atrial-sensing

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ventricular pacing systems), but single-lead RV pacing has been very successful in veterinary patients.
- Pacemaker therapy in cats is usually performed surgically via epicardial pacing, where the lead tip is sutured onto the LV free wall. Either a transthoracic or abdominal (transdiaphragmatic) approach can be used.
- Permanent pacing should be considered first-line therapy in both canine and feline patients with symptomatic bradyarrhythmias as it improves the quality of life and prolongs survival.

VENTRICULAR ARRHYTHMIAS

GENERAL REMARKS

Ventricular arrhythmias (VAs) may occur in structurally normal-appearing hearts (hereditary channelopathies) or may be associated with myocardial abnormalities such as cardiomyopathy, significant valvular disease, or myocarditis. To date, there is no medical therapy available that is known to prevent sudden death from VA (i.e., ventricular fibrillation [VF]). However, clinical signs such as syncope or episodic weakness can be alleviated in some animals with appropriate medical therapy. Clinically important VAs are most commonly identified in certain breeds, such as Boxers with arrhythmogenic right ventricular cardiomyopathy (ARVC), Doberman Pinschers and Great Danes with dilated cardiomyopathy (DCM), and German Shepherds and Rhodesian Ridgebacks with inherited ventricular arrhythmias. Dogs with congenital heart disease, such as severe subaortic or pulmonic stenosis, are predisposed to development of VAs, likely because of abnormal myocardial perfusion secondary to myocardial hypertrophy. These arrhythmias can be worse during exercise and may be exacerbated during cardiac catheterization for angiography or interventional therapy. Catheter contact with the endocardium can elicit ventricular arrhythmias and even cause VF. Other myocardial insults (i.e., myocarditis or myocardial infarcts) can result in acute, life-threatening VAs and sudden death. Increased cardiac troponin I, a biomarker to monitor the presence of acute myocardial injury, may suggest such underlying conditions. Anti-inflammatory therapy may be recommended in these cases, in addition to antiarrhythmic therapy.

Furthermore significant ventricular arrhythmias can be seen in any dog with significant underlying illness or severe electrolyte/acid-base derangements (i.e., hit by a car [traumatic myocarditis], large-breed dogs with gastric dilatation and volvulus, hemoabdomen, sepsis, hypokalemia or neoplasia [including infiltrative myocardial disease]). In some patients with ventricular arrhythmias, a cause cannot be identified.

Cats with severe forms of cardiomyopathy, hyperthyroidism, sepsis, neoplasia, or severe electrolyte imbalances may have VA. Clinical signs depend on duration and rate of the ventricular tachycardia (VT) but may include lethargy or rarely syncope if the rate is greater than 250 beats/min. Sudden death is not uncommon in cats with cardiomyopathies, presumably because of a fatal VA.

Treatment of Ventricular Tachycardia

- VT is recognized by an abnormally wide and bizarre QRS complex shape. P waves are present but not associated with QRS complexes and may be hiding in the QRS-T complexes. AV dissociation occurs because of the accelerated ventricular rate compared with the sinus rate. VT can be monomorphic (where each QRS complex is identical) or polymorphic (where the QRS complex shape varies). Rapid, polymorphic VT is considered a more unstable arrhythmia that may more likely degenerate into VF.
- From a treatment perspective, it is also important to differentiate between "fast" VT (>170 beats/min) and "slow" VT (80–160 beats/min), which is also called accelerated idioventricular rhythm (AIVR).
- Fast VT causes significantly reduced cardiac output, and clinical signs depend on the duration of the episode of VT. Affected animals may experience syncope, weakness, or no signs at all. Fast VT can degenerate into VF and cause sudden death.
- Antiarrhythmic therapy is warranted either to convert the VT to sinus rhythm or at least to slow down the rate of the VT or reduce the length of the runs.
- A baseline 24-hour Holter is essential before the initiation of therapy of fast VT to determine the percentage of ventricular ectopic beats and the duration and rate of runs of VT as well as the possible presence and length of pauses. A diary kept by the client or hospital staff documenting the activity of the dog and the exact time of observed syncope can help correlate ECG changes on the Holter with clinical signs.
- This information is critical both to institute suitable antiarrhythmic therapy and to assess drug efficacy by comparison of the posttreatment Holter with the baseline Holter.
- Accelerated idioventricular rhythm may be associated with underlying systemic disease or can occur transiently, after a gastric dilatation-volvulus (possibly secondary to endotoxemia) or hit-by-car (traumatic myocarditis) in dogs with structurally normal hearts. Cardiac troponin I (cTnI) may be elevated, indicating acute myocardial insult, or arrhythmias may be secondary to elevated circulating catecholamine levels associated with an underlying disease. AIVR usually does not require antiarrhythmic treatment, as the rate is not fast enough to cause significant hemodynamic consequences. Instead supportive care and monitoring of the underlying condition is imperative; however, if affected animals show signs of hypotension (particularly during surgery) or lethargy, then they still may benefit from treatment of slow VT.

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- Ventricular premature contractions (VPCs) are commonly observed in cardiomyopathic cats and are usually left untreated. However, sudden death is not uncommon in this population, so the importance of VPCs may be underestimated. Albeit more technically challenging, cats can also undergo Holter monitoring. It is helpful to "practice" wearing a Holter in cats, by having owners put a T-shirt on their the cat for a few hours at home, prior to a Holter appointment. Cats with severe ventricular arrhythmias warrant antiarrhythmic therapy.

Intravenous Therapy of VT

- For treatment of acute life-threatening VT, IV Lidocaine is the first drug of choice. Start with a lidocaine bolus (2–4 mg/kg IV over 1 minute) and repeat up to 8 mg/kg (total dose over 10 minutes). If effective, follow this with a constant-rate infusion (CRI) of lidocaine (30–75 µg/kg/min). A lidocaine bolus can cause a transient drop in blood pressure and can lead to vomiting or seizures. If serum potassium levels are below normal, lidocaine may not be as effective. Lidocaine is also effective in cats but should be used judiciously in this species because cats have a lower threshold for seizures associated with lidocaine.
- If lidocaine is not successful at restoring sinus rhythm or slowing VT, then procainamide IV can be added or administered instead. Procainamide is given initially as a bolus IV (5–15 mg/kg IV slowly), followed by a constant-rate infusion (25–50 µg/kg/min). Procainamide can lead to hypotension due to its negative inotropic effect, so careful monitoring of blood pressure during the infusion is recommended.
- Intravenous esmolol may occasionally be effective at terminating VT, especially in cases where high catecholamine levels may be contributing to the presence of VA. In dogs developing VT while undergoing an interventional catheterization procedure (i.e., balloon valvuloplasty for pulmonic stenosis), esmolol alone or in combination with lidocaine can be effective. Combination of esmolol with procainamide may cause a significant drop in cardiac output and hypotension. Esmolol may be safer to use than lidocaine in cats with acute life-threatening VT.
- Intravenous amiodarone (Nexterone) may be used for acute, refractory VT if lidocaine and procainamide are ineffective. It is safe (as opposed to the original IV form of amiodarone, reported in some dogs to cause anaphylaxis and sometimes sudden death) and can be administered as a 2 to 5 mg/kg IV bolus over 10 minutes, followed by a CRI of 0.8 mg/kg/h for 6 hours, and then decrease to 0.4 mg/kg/h. Nexterone typically does not result in immediate termination of VT (like lidocaine should), so it is advised to continue the IV therapy for several hours, even if the efficacy appears inadequate initially. It can be transitioned to oral amiodarone without a pause, eliminating the need for a loading-dose regimen for oral amiodarone.
- In cases of refractory VT, magnesium supplementation (30 mg/kg slowly IV) may be tried.
- See Tables 16.1 and 16.2 for drug dosages.
- Electrical cardioversion of refractory, life-threatening VT may be pursued, if drug therapies fail. However, this requires a short period of general anesthesia and an external defibrillator that can deliver shocks synchronized to the R wave.

Chronic Oral Antiarrhythmic Therapy of Ventricular Tachycardia

- The first-line oral AAD for treatment of VT in most dogs is sotalol. In young German Shepherds with inherited VA, sotalol monotherapy should be avoided, as KCB may cause worsening of VT in this condition. See Tables 16.1 and 16.2for drug dosages.
- If a dog has advanced myocardial systolic dysfunction (fractional shortening <15%), sotalol may not be well tolerated due its beta-blocker effect.
- Alternatively, mexiletine in combination with atenolol may be effective, particularly in Boxers with ARVC. The atenolol dosage can be started at the lower end, in the case of poor myocardial function. A practical disadvantage of this treatment regimen is the frequency of drug administration (mexiletine is given q8h).
- For refractory VT, a combination of sotalol with mexiletine may be effective. These drugs should be initiated in a staggered protocol (usually sotalol first, mexiletine added after 2 days) to avoid side effects such as AV block or inappetence. Because this is a treatment protocol for refractory arrhythmias, patients are usually already receiving sotalol, in which case the addition of the second drug is quite well tolerated.
- Oral procainamide or atenolol monotherapy are usually not effective for treatment of VT in dogs.
- As a last resort, oral amiodarone may be effective for treatment of refractory VT in dogs who do not respond to the regimens listed above. It has less negative inotropic effects than sotalol or atenolol, and thus can be used safely in dogs with end-stage myocardial failure (loading dosage of 5–15 mg/kg PO q12h for 2–14 days, maintenance dosage of 6–15 mg/kg PO q24h). But caution is advised when considering amiodarone therapy because of its adverse effects. Signs of toxicity include anorexia, vomiting, lethargy, and hepatic enzymes elevation. It can be combined with atenolol or mexiletine to enhance antiarrhythmic effects. Liver enzymes, complete blood cell count, and thyroid function need to be monitored every 2 months for the first 6 months, and every 6 months thereafter.
- Amiodarone toxicity in dogs

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- Amiodarone hepatopathy is reversible after a reduction of dosage or discontinuation of the drug. Overt clinical signs of toxicity resolve within a few days of stopping amiodarone. Hepatic enzyme activity gradually returns to normal within 3 months after amiodarone is discontinued or the dosage is reduced.
- Doberman Pinschers have a higher prevalence of inherent hepatopathies, and thus the incidence of amiodarone toxicity is possibly increased in this breed. Preexisting liver enzyme elevations are a contraindication for amiodarone therapy in Doberman Pinschers, unless no other alternative is available.

- A posttreatment 24-hour Holter is essential both to determine efficacy and check for possible proarrhythmic effects from an AAD. Significant worsening of VT during sotalol therapy has been documented in Boxers and German Shepherds.
- Oral sotalol is used for treatment for VT in cats. Using an 80-mg tablet, 1/8th of a tablet may provide an appropriate dose (1–2.5 mg/kg BID) for some cats. However, medication compounding (i.e., into an oral suspension or smaller tablet size) may provide more accurate dosing and improve owner compliance.

BREED-SPECIFIC ARRHYTHMIAS

Arrhythmogenic Right Ventricular Cardiomyopathy in Boxers

- Ventricular arrhythmias and myocardial dysfunction are both manifestations of ARVC in Boxers. The clinical presentation of ARVC varies from subclinical ventricular arrhythmias to arrhythmia-associated syncope or sudden death with normal myocardial function, and (rarely) predominantly systolic myocardial failure with or without VA. ARVC is inherited in an autosomal dominant pattern in Boxers. There are two different genetic mutations in Boxer dogs that can lead to ARVC. There is an association of a mutation in the striatin gene with the development of ARVC in some Boxers. Striatin is a desmosomal protein (scaffolding protein that connects cardiomyocytes), and ARVC in humans is commonly liked to various mutations in desmosomal proteins. The second mutation is known as NCSU ARVC2 and is a single nucleotide polymorphism in a regulatory gene.
- The QRS complex morphology of VA in Boxers is more commonly positive in the inferior (leads II, III, and aVF), and negative in V1 (left bundle branch block pattern), suggesting a right ventricular or high septal origin of the arrhythmia.
- Syncope is often the first clinical sign and usually, but not always, associated with rapid runs of VT. Boxers can have multiple syncopal episodes and recover. These episodes may be more common during exercise or stress.
- A subset of Boxers also has a form of SSS, wherein fainting may be caused by long pauses from sinus arrest.
- Some Boxers also have SVAs including AF, especially if they have advanced stages of myocardial dysfunction and congestive heart failure.

Ventricular Tachycardia in Boxers with Arrhythmogenic Right Ventricular Cardiomyopathy

Chronic oral antiarrhythmic therapy

- Because of the association of VT with ARVC and myocardial systolic dysfunction, an echocardiogram is recommended before specific antiarrhythmic drug recommendations are made. See Table 16.1 for drug dosages.
- Sotalol is the treatment of choice for VT in Boxers, if the myocardial function is normal or only mildly decreased. If a Boxer has significantly reduced myocardial function, sotalol therapy should be titrated up to the desired effect but may not be tolerated.
- In such cases, mexiletine in combination with atenolol can be used. Atenolol can be started at a lower dose to limit the beta-blocker effect on myocardial contractility.
- For refractory VT and recurrent syncope, the combination of sotalol with mexiletine is useful. If the dog is already receiving sotalol with inadequate success, the addition of mexiletine is usually well tolerated.
- If VT persists or the dog does not tolerate sotalol or mexiletine/atenolol, amiodarone may be beneficial. However, amiodarone hepatopathy may occur with long-term use. The monitoring of liver enzymes is recommended.
- Radiofrequency catheter ablation (RFCA) in Boxer dogs with symptomatic ventricular tachycardia has been performed. RFCA may decrease runs of VT and improve clinical signs.

ACUTE INTRAVENOUS ANTIARRHYTHMIC THERAPY

- See the section on the treatment of VT.

Ventricular Arrhythmias and Dilated Cardiomyopathy in Doberman Pinschers

- Ventricular arrhythmias, syncope, and sudden death associated with DCM are common in Doberman Pinschers. There are two genetic mutations (PDK4;DCM1 and DCM2) that have been identified. Dogs that carry both mutations are at the highest risk of developing the disease, although dogs with a mutation in either gene can develop the disease as well. DCM is characterized by a slowly progressive, clinically occult phase during which single ventricular premature contractions first appear. This phase is followed by the development of left ventricular dysfunction and usually progressively more severe

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ventricular tachyarrhythmias. The natural outcome in cardiomyopathic patients is usually either sudden death caused by ventricular arrhythmias or end-stage congestive heart failure, often associated with AF. The incidence of sudden death before the onset of congestive heart failure is between 30% and 50%.

Key Points

- VT in Doberman Pinschers has both monomorphic and polymorphic characteristics.
- Syncope or episodic weakness has been documented in Doberman Pinschers caused by VT and bradyarrhythmias such as paradoxical sinus bradycardia and cardiac asystole. Unlike Boxers with ARVC, Doberman Pinschers with VT and DCM may die suddenly during their first syncopal episode.

Ventricular Tachycardia in Doberman Pinschers with Dilated Cardiomyopathy

Chronic oral antiarrhythmic therapy

- For treatment of VT in Doberman Pinschers, sotalol may be effective (see Table 16.1 for drug dosages). A combination of mexiletine with sotalol or atenolol may be used for refractory VT. With both drug regimens, monitoring by echocardiogram is recommended to check for worsening of myocardial function because of beta blockade. In cases with only moderate VT or significantly reduced myocardial function, monotherapy with mexiletine may be beneficial because mexiletine does not affect contractility. Doberman Pinschers seem to have gastrointestinal side effects (inappetence and vomiting) from mexiletine more readily than other breeds, so the starting dose should be at the low end of the dose range.
- Amiodarone is also effective in Doberman Pinschers with significant VT and end-stage myocardial failure; however, in this particular breed, careful monitoring of liver enzyme levels is imperative because Doberman Pinschers have a high incidence of amiodarone toxicity (for more details see the section on the treatment of VT).

ACUTE INTRAVENOUS ANTIARRHYTHMIC THERAPY

- See the section on the treatment of VT.

Inherited Ventricular Arrhythmias in Young German Shepherds

- Inherited ventricular arrhythmias and a propensity for sudden death occur in young German Shepherds. This disorder has a wide phenotypic spectrum where some German Shepherds have very few ventricular premature complexes and others have frequent, rapid (rates >350 beats/min) polymorphic VT and sudden death. Dogs with documented VT are at risk for sudden death. Of German Shepherds with more than 10 runs of VT per 24 hours, approximately 50% die suddenly; this represents about 10% to 15% of the total affected populations studied. The prevalence of this disorder in the general German Shepherd population is unknown. Affected dogs have electrophysiologic abnormalities in calcium cycling and altered sympathetic innervation of the heart. Ventricular arrhythmias may be initiated by calcium sparks and after depolarizations (early and delayed) documented in the Purkinje fibers and ventricular myocytes of these dogs.
- German Shepherds have ventricular arrhythmias at 12 to 16 weeks of age, and the frequency and severity increase until 24 to 30 weeks of age. After that time, some dogs remain severely affected, whereas others show a progressive decline in the frequency of the arrhythmias. The dogs typically do not have syncope or other clinical signs. Most dogs have a low incidence of VT. If dogs reach the age of 18 months, the probability of sudden death declines markedly.

THERAPY OF GERMAN SHEPHERDS WITH VENTRICULAR TACHYCARDIA

- Dogs with mild to moderate amounts of ventricular arrhythmias do not warrant antiarrhythmic therapy. See Table 16.1 for drug dosages.
- In dogs at high risk of sudden death (>10 runs of VT/24 hours) based on a 24-hour Holter monitoring, antiarrhythmic therapy may be administered until the dog has "survived" the vulnerable time period.
- Intravenous lidocaine is effective in eliminating the ventricular arrhythmias acutely. However, monotherapy with the oral NCB mexiletine does not suppress the ventricular arrhythmias significantly. Sotalol monotherapy has proarrhythmic effects (i.e., causes increased numbers of runs of VT) in this specific breed, probably because of the action-potential prolonging effects, which can exacerbate early after depolarization-induced triggered activity.
- Combination therapy of mexiletine and sotalol may be beneficial in reducing the incidence and rate of VT in severely affected dogs; however, it is unknown if the risk of sudden death is reduced with this treatment.
- Amiodarone therapy has not been evaluated in this breed. There is a potential risk of similar proarrhythmic effects as with sotalol. Procainamide and atenolol appear to be ineffective as AADs.

VENTRICULAR ASYSTOLE

- Ventricular asystole is characterized by the complete absence of a ventricular rhythm. P waves may be present (with complete AV block), but no QRS complexes are observed. Primary asystole occurs when the Purkinje fibers intrinsically fail to generate

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a ventricular depolarization. It is usually preceded by a severe bradyarrhythmia caused by a complete heart block, SSS, or both.

THERAPY OF VENTRICULAR ASYSTOLE

- Immediate external cardiac pacing with either transthoracic, transvenous, or epicardial electrodes (temporary pacing lead) may be effective if asystole is the result of a complete AV bock. Permanent pacemaker therapy is indicated. During cardiopulmonary resuscitation, epinephrine, isoproterenol, terbutaline, or atropine can be administered IV, but little effect is to be expected.
- Secondary asystole occurs when noncardiac factors suppress the electrical conduction system, resulting in a failure to generate any electrical depolarization. Massive pulmonary embolus, hyperkalemia, hypothermia, untreated VF or VT that deteriorates to asystole, unsuccessful defibrillation, or narcotic overdoses leading to respiratory failure can lead to secondary asystole. In such cases, the final common pathway is usually severe tissue hypoxia with metabolic acidosis.
- The prognosis is usually grave for secondary asystole because external pacing is not effective in such cases.

VENTRICULAR FIBRILLATION

- No distinctive QRS complexes are identifiable on the ECG. Instead there is an irregularly undulating baseline of variable amplitude caused by a rapid and chaotic activation of the ventricles. The sinus node is usually discharging regularly, but P waves are buried in the VF waveform. There is no mechanical contraction of the ventricles during VF; thus blood pressure drops to zero instantaneously. The rhythm cannot convert to sinus rhythm spontaneously and causes death within a few minutes because of lack of cardiac output. VT can degenerate to VF in any of the described scenarios of VT (see the previous section). VF can also occur secondary to systemic disease (i.e., severe hyperkalemia). Dogs that have congenital heart disease and are predisposed to ventricular arrhythmias may have VF during cardiac catheterization for angiography or interventional therapy because of "catheter irritation" of the myocardium.

Therapy of VF

- Drug therapy is usually not effective for treatment of VF. Because there is no blood pressure or blood flow during VF, drugs administered in a peripheral vein will not reach the myocardium. Electric defibrillation is the treatment of choice for VF.
- If VF occurs secondary to electrolyte imbalances or systemic disease, then the prognosis is usually grave, despite aggressive cardiopulmonary resuscitation efforts.
- Animals with a predisposition to VAs or with VF while under anesthesia for catheter treatment of heart disease (but that are otherwise relatively healthy) may be successfully cardioverted. It is imperative that defibrillation occurs within a very short time after onset of VF (within 1–3 minutes).
- VF has a grave prognosis if not corrected within the first 3 minutes of onset. Development of myocardial ischemia during VF contributes to worsening prognosis as time goes by. Cardiopulmonary resuscitation with chest compression should be performed continuously during the arrest, until ECG cables and defibrillator are ready for use.

Transthoracic Electrical Defibrillation Procedure

- Dogs in VF become unconscious within 10 seconds because of the lack of blood flow to the brain.
- Defibrillation should be attempted using transthoracic handheld paddles or "Fast Patch" electrodes.
- Animals should be placed in dorsal recumbency, and copious contact gel should be applied to the thorax if handheld paddles are used. For optimal current flow, the chest should be shaved first, but that may not be feasible in the interest of time. The dorsal recumbent position is safest for the operator but may not be optimal for defibrillation success in deep-chested dogs. If patch electrodes are available, dogs can also be placed in lateral recumbency, which might allow the defibrillation electrodes to be closer to the heart.
- Defibrillator ECG cables or the handheld paddles placed on the thorax should be used to ascertain the presence of VF before defibrillation.
  - With a biphasic defibrillator
  - Start with 3 J/kg.
- If VF persists, increase the dose by 50 J and repeat until a maximum of 360 J.
  - With a monophasic defibrillator
  - Start with 6 J/kg.
- If VF persists, increase the dose by 50 J and repeat until a maximum of 360 J.
- Short, transient runs of VT, sinus pauses, or AV block are common following defibrillation.

Frequently Asked Questions

WHAT IS AN ACCELERATED IDIOVENTRICULAR RHYTHM, AND SHOULD IT BE TREATED?

An idioventricular rhythm is a form of VA characterized by a rate that is slow or comparable to the sinus rates (60–150 beats/min in dogs and >100 beats/min in cats). The ventricular rate usually remains within 10–15 beats/min of the concurrent sinus rate, and the cardiac rhythm "switches" back and forth between the two competing pacemaker sites. This arrhythmia may occur for several reasons, including systemic diseases (e.g., anemia, splenic hemangiosarcoma), drugs (e.g., digoxin, opioids), electrolyte abnormalities (e.g., hypokalemia), stress (postoperative), endotoxemia (post-gastric -dilatation torsion surgery), and traumatic

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myocarditis (hit by car). In general, no clinical signs are associated with idioventricular rhythms. If clinical signs are present, they are often associated with the underlying process. Idioventricular rhythms usually do not require treatment. Management of the underlying systemic disease or metabolic abnormality is required.

WHAT ARE THE ADVANTAGES OF RATE CONTROL COMPARED WITH RHYTHM CONTROL IN THE MANAGEMENT OF ATRIAL FIBRILLATION?

Rate control can be achieved on an outpatient basis with oral medication. Rate control AADs are relatively inexpensive and well tolerated. Rate control results in reduction in clinical signs associated with heart failure and significantly prolongs survival if a mean HR of <125 beats/min can be achieved.

WHAT ARE THE DISADVANTAGES OF RATE CONTROL?

Rapid activation of the atria during AF results in progressive atrial myocardial failure; elevated HR and AV dyssynchrony contributes to progressive ventricular myocardial failure (tachycardiomyopathy). Rate control should be monitored with periodic Holter recordings, acquired in the home (stress-free) environment, which increases the cost of the (lifelong oral AAD) therapy.

Long-term oral twice daily drug therapy requires owner compliance.

WHAT ARE THE ADVANTAGES OF RHYTHM CONTROL (CARDIOVERSION: ELECTRICAL OR PHARMACOLOGIC)?

The most important advantage is that sinus rhythm and AV synchrony provide optimal cardiac output, especially in the face of myocardial failure. Furthermore the sinus rhythm allows adequate changes in HR according to blood pressure and autonomic tone.

Thus tachycardiomyopathy is avoided and a reduction in clinical signs and improved exercise tolerance can be achieved. The longer a dog who previously had AF can maintain sinus rhythm, the better for longevity.

WHAT ARE THE DISADVANTAGES OF CARDIOVERSION (ELECTRICAL OR PHARMACOLOGIC)?

A main disadvantage of electrical cardioversion is the requirement of general anesthesia, hospitalization, and a defibrillator that has the SYNC feature; thus the procedure is expensive, at least up front. The second disadvantage is the risk of recurrence of AF even after successful cardioversion. Recurrence of AF in dogs is not a question of if, but when. Oral antiarrhythmic drugs are also recommended in dogs undergoing cardioversion (amiodarone pre- and post-treatment) to enhance the chance of success and reduce the risk of immediate recurrence of AF.

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Chapter 17

Cardiopulmonary Arrest and Resuscitation

Vincent J. Thawley

Cardiopulmonary resuscitation (CPR) describes a set of techniques to provide circulatory and ventilatory support following cardiopulmonary arrest (CPA). CPR encompasses both basic and advanced life support. Basic life support (BLS) includes performing external chest compressions or internal cardiac compressions to generate forward blood flow, establishing an airway, and providing manual ventilation. Advanced life support (ALS) includes drug therapy, defibrillation, and patient monitoring techniques including electrocardiogram (ECG) analysis and capnography to measure end-tidal CO_2 ($ETCO_2$) during resuscitation. The goal of CPR is to maximize blood flow and oxygen delivery to the heart and brain until return of spontaneous circulation (ROSC) is achieved and the underlying cause of the arrest may be addressed. Ideally, ALS techniques are performed simultaneously with BLS, but only once good-quality BLS has been initiated.

- *CPA* is defined as the cessation of spontaneous circulation and ventilation. Causes of CPA include primary myocardial disease (although this is rare in veterinary patients), hypotension (secondary to hypovolemia, sepsis, or drug administration), hypoxemia (secondary to lung disease or hypoventilation), metabolic derangements (e.g., severe metabolic acidosis), or electrolyte abnormalities (e.g., hyperkalemia). Recognizing that these predisposing causes of arrest may be associated with either reversible or irreversible underlying disease processes is important.
- The prognosis for patients requiring CPR is guarded, and survival to hospital discharge is generally less than 10%. The likelihood of a successful outcome is improved when an arrest is rapidly recognized and a reversible cause is identified and addressed.
- CPR is most likely to be successful when the team is prepared, the techniques are practiced, the communication is clear, and the resuscitation takes place in a well-equipped area within the hospital.

Key Point

CPR is a potentially lifesaving procedure that requires preparation, coordination, and communication to achieve optimal results.

BASIC LIFE SUPPORT

CIRCULATION

- The first priority in BLS is rapid initiation of good-quality chest compressions. Artificial circulation during CPR may be provided by performing external chest compressions or internal cardiac massage. The goal of either technique is to maximize blood flow to the coronary and cerebral vasculature.
- Myocardial perfusion pressure (MPP) is the best predictor of ROSC in human patients and animal models of CPR, and it is represented by the following equation: MPP = aortic diastolic pressure − central venous pressure.
- Cerebral perfusion pressure (CPP) drives cerebral blood flow and is represented by the following equation: CPP = mean arterial pressure − intracranial pressure.
- There are two theories describing the mechanism of blood flow during external chest compressions. The cardiac pump theory describes actual compression of the heart through the chest wall and is likely to occur in small patients (<15 kg). The thoracic pump theory describes blood flow as a result of phasic increases in intrathoracic pressure and has been documented in larger animals (>15 kg).
- External chest compressions should be performed with the patient positioned in lateral recumbency with its dorsum toward the compressor. The chest may be compressed circumferentially or directly over the heart in small patients (<15 kg) and at the widest point of the chest in larger patients (>15 kg). Sternal compressions with the patient positioned in dorsal recumbency are recommended for barrel-chested breeds such as English Bulldogs.
- The rate of chest compressions should be 100 to 120 per minute with a ratio of compression to relaxation of 50:50. Although higher compression rates have been shown to generate greater cardiac output, it is difficult to sustain higher rates for extended periods of time during CPR. Compressions should be given with enough force to decrease the diameter of the chest wall by approximately 33% to 50%, or by 25% in barrel-chested breeds positioned in dorsal

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recumbency. Full elastic recoil of the chest should be permitted between each compression as this will allow for improved venous return during the decompression phase.

- Chest compressions should be initiated as soon as possible after diagnosing cardiopulmonary arrest and performed in 2-minute cycles with minimal interruptions. Experimental animal models have shown that peak CPP is only achieved after approximately 60 seconds of uninterrupted chest compressions and that there is a rapid decrease in CPP whenever compressions are stopped.
- If a sufficient number of rescuers are present, the person performing chest compressions should be rotated every 2 minutes to prevent the development of fatigue because this has been shown to negatively affect the quality of the resuscitation effort. The brief pause in compressions as rescuers are rotated, ideally no longer than 5 seconds, is an appropriate time to assess the patient for ROSC and assess the cardiac rhythm should a continuous ECG be employed.
- Even optimal external chest compressions produce approximately 25% of normal cardiac output. Open-chest CPR and internal cardiac compression may produce 100% of normal cardiac output, with dramatic increases in blood flow to the heart and brain. Indications for open-chest CPR include pleural space disease (e.g., pneumothorax, pleural effusions, diaphragmatic hernia), pericardial effusion, penetrating wounds, chest wall trauma, intraoperative arrests, hemoperitoneum, large dogs in which closed-chest compressions are unlikely to generate effective blood flow, or prolonged resuscitations (>2–5 minutes without ROSC).
- For open-chest CPR to be performed, the heart may be accessed via a left lateral thoracotomy (or transdiaphragmatically in patients undergoing abdominal surgery). After a rapid clip and preparation of the left chest, an emergency thoracotomy may be performed in approximately 30 seconds. A skin incision is made in the fourth or fifth intercostal space and is extended through the chest wall musculature. Ventilation is temporarily suspended, and the pleural space is accessed. A rib spreader is used to retract the ribs. Transection of the rib caudal or cranial to the thoracotomy incision at the level of the costochondral junction followed by tucking the transected rib under the next adjacent rib, a technique known as shingling, has been shown in a recent canine cadaver study to improve access to intrathoracic structures such as the heart and aorta. Once the heart is exposed, the ventricles may be compressed with one or two hands depending on the size of the patient. A rate of 100 to 120 compressions per minute is recommended. It is often easier to perform direct cardiac compression once an incision has been made in the pericardium (below the level of the phrenic nerve). The pericardium should be incised before cardiac compressions are initiated if pericardial effusion with tamponade is encountered.
- Open-chest CPR allows for compression or cross-clamping of the descending aorta to direct blood flow to the heart and brain and avoids additional volume loss in cases of abdominal hemorrhage. In the absence of an atraumatic vascular clamp, the aorta may be manually compressed, or a Penrose drain or red rubber catheter may be tightened around the aorta. When appropriate, aortic flow may be gradually restored (over 5–10 minutes).
- Open-chest CPR requires that appropriate facilities and expertise be available for postresuscitation care and management of the emergency thoracotomy.

AIRWAY

- Once chest compressions are underway, the next priority is securing an airway. Orotracheal intubation is generally performed in a routine fashion, and this may be facilitated by the use of a laryngoscope or a stylet for the endotracheal tube as well as an assistant to exteriorize the tongue. Have suction available if secretions or blood obscure visualization of the glottis. In situations where the glottis cannot be visualized, the larynx may be directly palpated and the endotracheal tube may be guided by feel. Intubation performed with the patient in lateral recumbency is suggested as this positioning allows for concurrent chest compressions.
- In rare situations, an emergency tracheostomy is required. This technique may be performed in less than 30 seconds after rapidly clipping and prepping the ventral cervical region. A midline incision is performed and sharp dissection is used to expose the trachea. Care is taken during dissection to remain on midline (between the sternothyroideus muscles) to avoid vascular structures. Once the trachea has been isolated, a transverse incision is made between cervical rings (approximately 50% of the diameter) and a cuffed tracheostomy tube is inserted. A standard endotracheal tube may also be used in this situation.
- Once an airway is established, it is important to confirm correct tube placement. This may be done by direct visualization, cervical palpation, auscultation of lung sounds, and observing chest wall movement. The use of $ETCO_2$ monitoring in this situation is also useful because tracheal gas is always higher in CO_2 than esophageal gas; however, lack of detectable $ETCO_2$ does not preclude endotracheal intubation because delivery of carbon dioxide to the lungs is compromised in the setting of cardiopulmonary arrest. Once placement is confirmed, it is vital to secure the endotracheal tube, as inadvertent tube dislodgement is very common in an arrest situation. During the resuscitation attempt, endotracheal tube

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placement should be assessed periodically to ensure that it has not become dislodged.
- Airway problems during an arrest (i.e., inability to auscult lung sounds, chest wall not moving with ventilation) should prompt rapid reevaluation of endotracheal tube placement. It is also important to ensure that the cuff has been inflated because this is often the source of problems. If airway problems have been ruled out, difficulty ventilating the lungs during an arrest suggests severe pleural space, airway, or parenchymal disease.

BREATHING

- The patient's lungs should be manually ventilated with 100% oxygen. Methods for providing positive pressure ventilation in an arrest situation include the use of an Ambu-bag or an anesthesia machine.
- Respiratory rate should be approximately 10 breaths per minute with an inspiratory time of 1 second. It should be noted that excessive ventilation often occurs during CPR and should be avoided. It has been shown in animal models that faster respiratory rates, long inspiratory time, and high tidal volumes result in a higher mean intrathoracic pressure, decreased myocardial perfusion pressure, and decreased survival.
- Normal chest wall motion should be observed and peak pressure of less than 40 cm H_2O should be maintained if possible to reduce the risk of pulmonary barotrauma. Problems causing decreased pulmonary compliance or diminished chest wall motion may include airway obstruction, severe parenchymal disease, or pleural space disease (e.g., pneumothorax, pleural effusion, diaphragmatic hernia, mass lesions).
- In the event that intubation is not feasible, breaths may be delivered via the use of a tight-fittng face mask at a ratio of 30 chest compressions followed by two rapid breaths, with immediate resumption of compressions. If a tight-fittng face mask is not available, mouth-to-snout ventilation may be considered if there is low risk to the rescuer; otherwise, chest compression-only CPR is recommended.

Key Point

Basic life support measures are the basis for all resuscitation efforts. These techniques are easily learned and are effective in maintaining ventilation and artificial circulation in patients with CPA.

ADVANCED LIFE SUPPORT

INITIATE PATIENT MONITORING: ELECTROCARDIOGRAPHY AND END-TIDAL CO_2

- ECG monitoring is integral to providing advanced life support. The course of action taken during CPR depends on the cardiac rhythm that is present. As discussed below, changes in the cardiac rhythm during the course of an arrest often dictate changes in therapy (Fig. 17.1).
- As the ECG is susceptible to motion artifact, accurate rhythm diagnosis can only be determined during the brief pause in chest compressions when compressors are rotated.
- $ETCO_2$ monitoring is an easily applied and extremely useful monitoring tool in CPR. If ventilation is constant, $ETCO_2$ is linearly related to pulmonary blood flow and, by extension, cardiac output. As with myocardial perfusion pressure, higher $ETCO_2$ during CPR has been shown to correlate with increased likelihood of successful resuscitation. In addition, because $ETCO_2$ is a surrogate marker for pulmonary blood flow, marked increases in this parameter serves as a useful indicator of ROSC. Unlike ECG, $ETCO_2$ is not affected by motion artifact and may be monitored during chest compressions.
- Achieving an $ETCO_2$ of at least 18 mm Hg may be associated with a higher likelihood of ROSC.

VENOUS ACCESS FOR DRUG AND FLUID THERAPY

- Rapid access to the circulation is vital in CPR. Central venous access (e.g., jugular vein) is ideal because drug circulation times are significantly lower when compared with peripheral venous sites. Because of the low-flow state that occurs during CPR, large flush volumes (at least 5–10 mL of nonheparinized 0.9% NaCl) are necessary, especially when peripheral catheters are used. Short, large-bore catheters are ideal, as these provide the highest flow rates for drug and fluid administration.
- Surgical cut down should be performed immediately if the initial attempt at percutaneous vascular access is not successful. Surgical cut down involves making a skin incision adjacent and parallel to the long axis of the vein (usually jugular, cephalic, or saphenous) to be isolated. Blunt dissection with a hemostat is used to expose the vein, and an intravenous catheter is introduced. Cut-down catheters should be secured with sutures and bandaged appropriately.
- Intraosseous (IO) access is an alternative to peripheral venous access, especially in small puppies, kittens, and exotic species. The intertrochanteric fossa of the femur, proximal humerus, and proximal tibia are readily accessible sites to obtain IO access.
- Intratracheal (IT) administration is an excellent method to deliver drugs when intravenous access is not available. Most drugs used in CPR (with the exception of sodium bicarbonate) can be delivered by this route. When drugs are delivered intratracheally 2 to 10 times the intravenous dose is administered, and the drug is delivered through a red rubber catheter placed through and beyond the tip

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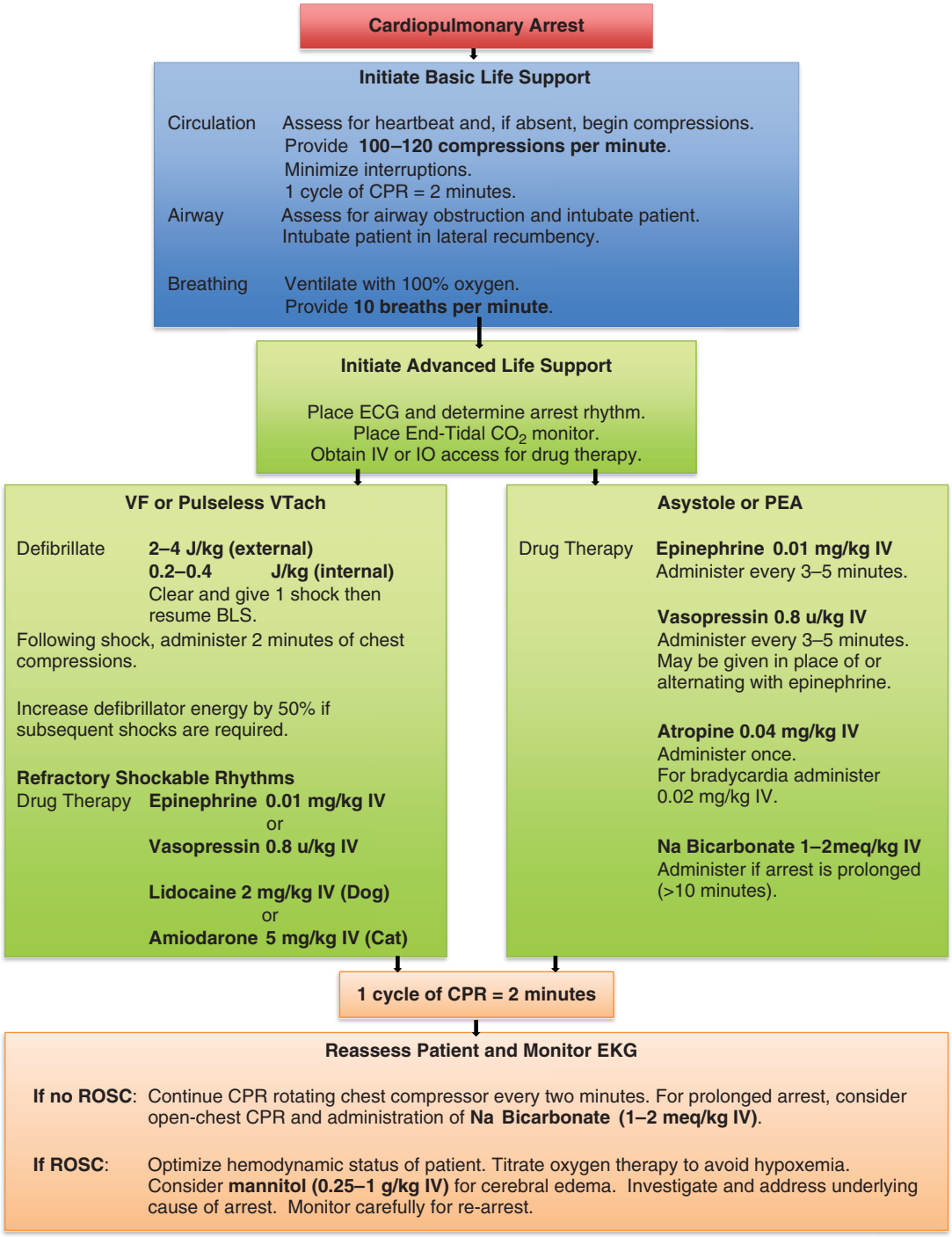


Fig. 17.1 Algorithm for performing cardiopulmonary resuscitation (CPR) in veterinary patients. *BLS*, Basic life support; CO_2, carbon dioxide; *ECG*, electrocardiogram; *IO*, intraosseous; *IV*, intravenous; *PEA*, pulseless electrical activity; *VF*, ventricular fibrillation; *pVTach*, pulseless ventricular tachycardia. (Adapted from Burkitt-Creedon JM, Boller M, Fletcher DJ, et al: 2024 RECOVER Guidelines: Updated treatment recommendations for CPR in dogs and cats. J Vet Emerg Crit Care 2024;34(Suppl 1), 2024.)

of the endotracheal tube (at the level of the carina). Air may be used to flush the catheter.

- Intracardiac drug administration is not recommended in CPR because there is a risk for inadvertent laceration of the lung or coronary vasculature, as well as the potential for intramyocardial drug administration (which may exacerbate arrhythmias or ischemia in the case of epinephrine).

SEDATION/ANALGESIA REVERSALS

- For CPA that occurs following administration of sedative or analgesic medications, prompt

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administration of reversal medications may increase the likelihood of achieving ROSC.

- Commonly used reversal agents include naloxone (for opioids) at 0.04 mg/kg (dogs and cats, IV), flumazenil (for benzodiazepines) at 0.01 mg/kg (dogs and cats, IV), or atipamezole (for alpha-2 agonists) at 0.05 mg/kg (dogs and cats, IV),
- See Table 17.1 for drug therapy guidelines.

ELECTROCARDIOGRAM EVALUATION

- As the ECG is susceptible to motion artifact when chest compression is underway, the best time to evaluate the ECG is during the brief pause at the end of a 2-minute CPR cycle when compressors are rotated. The rhythm is analyzed to determine if it is a perfusing rhythm, a shockable rhythm, or a nonshockable rhythm.
- If the ECG shows consistently repeating complexes that reliably produce pulses in the absence of chest compressions, this is a perfusing rhythm and ROSC has been achieved. If the rate is adequate, CPR efforts are suspended and postresuscitation care (discussed below) is initiated.
- Nonshockable rhythms include pulseless electrical activity (PEA, consistently repeating complexes at a rate <200/min that do not produce pulses) and asystole (ECG flat line). If asystole is the rhythm diagnosis, the ECG leads should be checked to ensure they have not disconnected from the patient. Nonshockable rhythms are more common than shockable rhythms in small animal patients experiencing CPA.
- Shockable rhythms include pulseless ventricular tachycardia (consistently repeating complexes at a rate >200/min that do not produce pulses) and ventricular fibrillation (unorganized ECG electrical activity).

MANAGEMENT OF NONSHOCKABLE RHYTHMS (PULSELESS ELECTRICAL ACTIVITY AND ASYSTOLE)

- Vasopressor therapy is utilized to optimize perfusion to the heart and the brain.
- **Epinephrine** is a potent alpha and beta catecholamine receptor agonist. Experimental studies have shown that it is the alpha (vasoconstrictor) effects rather than the beta (chronotropic/inotropic) effects that are most important in achieving ROSC. The benefit is due to the increased peripheral resistance created by adrenergic stimulation and the resultant

Table 17.1 Guidelines for Drug Therapy and Initial Defibrillator Settings During CPR

DRUG (CONC.)	WEIGHT (LB)	5	10	20	30	40	50	60	70	80	90	100
	WEIGHT (KG)	2.5	5	10	15	20	25	30	35	40	45	50
	DOSE	ML										
Epi (1:1000)	0.01 mg/kg	0.025	0.05	0.1	0.15	0.2	0.25	0.3	0.35	0.4	0.45	0.5
Atropine (0.54 mg/mL)	0.04 mg/kg	0.2	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Lidocaine (20 mg/mL)—Dog)	2 mg/kg	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Sodium bicarbonate (1 mEq/mL)	1 mEq/kg	2.5	5	10	15	20	25	30	35	40	45	50
Calcium gluconate (100 mg/mL)	50 mg/kg	1	2.5	5	7.5	10	12.5	15	17.5	20	22.5	25
Magnesium sulfate (4 mEq/mL)	0.2 mEq/kg	0.1	0.25	0.5	0.75	1	1.25	1.5	1.75	2	2.25	2.5
Vasopressin (20 units/mL)	0.8 unit/kg	0.1	0.2	0.4	0.6	0.8	1	1.2	1.4	1.6	1.8	2
Amiodarone (50 mg/mL)—Cat)	5 mg/kg	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Naloxone (0.4 mg/mL)	0.04 mg/kg	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Flumazenil (0.1 mg/mL)	0.01 mg/mL	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
External defibrillation (monophasic defibrillator)	4–6 J/kg	10	20	40	60	80	100	120	140	160	180	200
External defibrillation (biphasic defibrillator)	2–4 J/kg	5	10	20	30	40	50	60	70	80	90	100
Internal defibrillation	0.2–0.4 J/kg	1	2	3	3	4	5	6	7	8	9	10

CPR, Cardiopulmonary resuscitation.

Adapted from Burkitt-Creedon JM, Boller M, Fletcher DJ, et al: 2024 RECOVER Guidelines: Updated treatment recommendations for CPR in dogs cats. *J Vet Emerg Crit Care* 2024;34(Suppl 1), 2024.

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increase in aortic pressure that leads to an increase in myocardial perfusion pressure and a more successful resuscitation. Current guidelines in the veterinary literature suggest the use of epinephrine at a dose of 0.01 mg/kg. High-dose epinephrine therapy is no longer recommended as there is evidence to suggest that this may in fact have deleterious side effects due to adrenergic overstimulation. Epinephrine can be given IV, IT, or IO, and a shortcut to calculate the volume for administration is 0.1 mL/10 kg. The dose may be repeated every other cycle of CPR.

- **Vasopressin** is a noncatecholamine vasopressor drug. Potential advantages of vasopressin (compared with epinephrine) include efficacy in the presence of acidosis, lack of potentially harmful beta effects, and a longer half-life. The role of vasopressin in CPR is still being investigated, and current guidelines suggest that vasopressin can be used either in lieu of or alternating with epinephrine. The dose of vasopressin is 0.8 units/kg (dogs and cats), and the dose may be repeated every other cycle of CPR.
- **Atropine** is a vagolytic drug that abolishes parasympathetic tone. It is indicated in patients with bradycardias (as may occur in vagal events), as well as in pulseless electrical activity and asystole. It should be noted that high doses of atropine may cause a profound tachycardia in patients with perfusing rhythms, and that the dose is often reduced by 50% to 75% in these situations. The dose of atropine is 0.04 mg/kg (dogs and cats) and can be given by intravenous (IV), IT, or IO routes. Current guidelines suggest to administer atropine only once, and early in the CPR effort, for patients with nonshockable rhythms.
- **Sodium bicarbonate** is not recommended for use in *all* arrest situations. It is indicated, however, in patients with a preexisting severe metabolic acidosis, patients with severe hyperkalemia, and in prolonged (>10 minutes) arrest scenarios. The dose for sodium bicarbonate is 1 to 2 mEq/kg (dogs and cats, IV, IO). A shortcut to calculate the dose is 1 mL/kg of a standard 1 mEq/mL solution. Sodium bicarbonate should not be given intratracheally, as it will inactivate surfactant and have deleterious effects on pulmonary function. Administration of sodium bicarbonate to a patient that is not appropriately ventilated during CPR may lead to hypercapnia and respiratory acidosis. Sodium bicarbonate should not be used in patients with known hypoventilation.

MANAGEMENT OF SHOCKABLE RHYTHMS (PULSELESS VENTRICULAR TACHYCARDIA AND VENTRICULAR FIBRILLATION)

- Electrical defibrillation is the only effective method to convert ventricular fibrillation to a perfusing cardiac rhythm. Defibrillation is also indicated in patients with pulseless ventricular tachycardia. See Table 17.1 for defibrillation guidelines.
- Immediate defibrillation is recommended only when the duration of cardiopulmonary arrest caused by ventricular fibrillation or pulseless ventricular tachycardia is 4 minutes or less; in which case BLS is initiated and continued only as long as it takes to charge the defibrillator. Otherwise, a 2-minute cycle of chest compressions before defibrillation is recommended. The initial dose of energy for defibrillation is 2 to 4 J/kg (biphasic defibrillator) or 4 to 6 J/kg (monophasic defibrillator).
- The defibrillator must be used properly to minimize risks to members of the resuscitation team. It is strongly recommended that the patient be placed in lateral recumbency for both CPR and defibrillation. Attempting to defibrillate a patient in dorsal recumbency may allow the patient's limb to contact a team member, which could lead to the unintentional delivery of current to a staff member and a potentially harmful situation. Most defibrillators have attachments for an accessory flat paddle (often called a posterior paddle) that may be placed under the patient, with the handheld paddle placed over the heart on opposite sides of the chest wall.
- Use large amounts of contact gel and press the paddles firmly onto the patient's thorax so that the current is delivered through the chest rather than "arcing" across the surface of the skin. Arcing of current is inefficient and may be dangerous, especially if alcohol has been placed on the patient. The smell of burning hair should alert the clinician that the current is likely arcing; applying more contact gel and repositioning the paddles are warranted before administering another shock. Because of the risk of combustion during defibrillation, alcohol (to increase ECG contact) should not be used during CPR. ECG contact gel is a much safer alternative.
- Clear communication during defibrillation is also important to ensure safety. The operator must inform the other resuscitation team members of an impending defibrillation attempt and confirm that no member of the team is in contact with the patient or table before delivering a shock. Because of the safety concern, a standard protocol is followed for each defibrillation. This protocol is as follows: (1) confirm ventricular fibrillation or pulseless ventricular tachycardia, (2) apply contact gel, (3) confirm current to be delivered and charge defibrillator, (4) halt ongoing CPR, (5) call "Clear," (6) confirm that all personnel are clear of the patient (especially limbs) and table, (7) deliver current, and (8) immediately resume chest compressions for a full 2-minute cycle after which the rhythm can be evaluated to determine if another shock is indicated.
- If an initial shock is not successful, CPR is resumed for 2 minutes before defibrillation is attempted again if a shockable rhythm persists. The initial

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defibrillation energy may be doubled for the second and subsequent shocks.
- Mechanical defibrillation via a precordial thump can be attempted if electrical defibrillation is not available; however, this technique is thought to be of little benefit in most situations.
- For ventricular fibrillation or pulseless ventricular tachycardia that is refractory to the first defibrillation attempt, **lidocaine** (2 mg/kg) may be administered to dogs and amiodarone (5 mg/kg IV) may be administered to cats. Amiodarone should be diluted in 5% dextrose before administration and may cause hypotension. Use of lidocaine may suppress a functional escape rhythm in the postarrest period, should ROSC be achieved. Additionally, epinephrine or vasopressin may be used every other CPR cycle. Esmolol (0.5 mg/kg IV loading dose, followed by a continuous infusion at 50 microgram/kg/min) may also be considered.

ANCILLARY THERAPIES

- **Intravenous fluids** can be administered in shock doses (90 mL/kg in dogs and 60 mL/kg in cats, given in aliquots to effect) to patients who are hypovolemic. Intravenous fluids may also be useful to help flush drugs from peripheral sites into the central circulation. It should be noted, however, that myocardial perfusion pressure may be reduced by significant increases in central venous pressure and that bolus fluid therapy may be counterproductive in patients who are euvolemic or volume overloaded at the time of arrest.
- **Calcium gluconate** is also not routinely recommended in every arrest situation because its use may exacerbate ischemia-reperfusion injury. It is indicated in patients with symptomatic hyperkalemia or hypermagnesemia, in patients with known hypocalcemia, and for treatment of calcium channel blocker toxicity. The dose of calcium gluconate is 50 to 100 mg/kg (dogs and cats, IV, IO).
- **Magnesium sulfate** is indicated in patients with known hypomagnesemia and in some ventricular arrhythmias (e.g., torsade de pointes). The dose of magnesium sulfate is 30 mg/kg (dogs and cats, IV, IO).
- Charts with guidelines for drug dosing during CPR are available from the Veterinary Emergency and Critical Care Society (www.veccs.org).

MONITORING CARDIOPULMONARY RESUSCITATION EFFORTS

- Patient monitoring during CPR can be difficult, and some standard techniques can be potentially misleading in an arrest situation.
- Palpation of femoral pulses during chest compression is an encouraging finding; however, the presence of pulses (and a discernible pulse pressure) does not necessarily correspond to adequate arterial blood pressure or perfusion pressures. Direct arterial pressure measurement is ideal, although this is generally only feasible in patients with a previously placed arterial line.
- As mentioned earlier, ECG monitoring is vital during CPR because this often dictates the type and timing of intervention. ECG findings must always be interpreted in the light of physical examination parameters. This is especially important when an apparent escape rhythm is present. The presence of auscultable heart sounds and palpable pulses indicates ROSC.
- $ETCO_2$ monitoring is invaluable as it provides a real-time efficacy monitor for resuscitative efforts. Failure to achieve an $ETCO_2$ of at least 18 mm Hg should prompt the team to evaluate and optimize the BLS/ALS strategy. A sustained increase in $ETCO_2$ to >35 mm Hg is suggestive of ROSC; however, this should be confirmed by pulse palpation or auscultation.
- Blood gas analysis may be misleading during CPR. Despite the low-flow state and global tissue ischemia that occurs, arterial blood gas results may appear relatively normal after equilibration with alveolar gas (especially with the hyperventilation that frequently occurs during CPR). On the other hand, venous blood gas results reflect the metabolic and respiratory acidosis that characterizes the local tissue environment in the face of hypoperfusion and decreased clearance of metabolic byproducts. Therefore venous blood gas results are more useful in the monitoring of CPR. It should be noted, however, that elevated venous CO_2 may result from inadequate blood flow and local tissue hypoperfusion rather than true hypoventilation.

Key Point

Advanced life support techniques include the implementation of patient monitoring, drug therapy, and defibrillation. These interventions are based on the circumstances unique to each arrest and provide options to augment the effectiveness of basic life support.

SPECIAL SITUATIONS

ANESTHETIC ARRESTS

- In general, anesthetic-related arrests are rare; however arrests that occur in conjunction with anesthesia are usually rapidly recognized, and some retrospective veterinary studies demonstrate that these patients are the most likely to be successfully resuscitated.

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- Steps to take in an anesthetic-related arrest include initiating standard CPR, turning off gas anesthesia and flushing the anesthetic circuit, opening the pop-off valve if it is closed, and reversing injectable anesthetic agents with naloxone (for opioids) at 0.04 mg/kg (dogs and cats, IV), flumazenil (for benzodiazepines) at 0.01 mg/kg (dogs and cats, IV), or atipamenzole (for alpha-2 agonists) at 0.05 mg/kg (dogs and cats, IV).
- Immediate open-chest CPR should be performed in patients undergoing thoracotomy and should be considered in patients undergoing celiotomy (via a transdiaphragmatic approach).
- Possible underlying causes such as hypoventilation, hypoxemia, hypotension, or cardiac arrhythmias should be investigated and corrected immediately.

VAGAL EVENTS

- Vagal events, characterized by bradycardia, hypotension, and collapse, may occur in critically ill patients, especially in conjunction with coughing, retching, vomiting, or straining to defecate. In extreme cases, bradycardia may be profound and lead to asystole.
- **Atropine** is the treatment of choice in patients with symptomatic bradycardia, and it should be noted that significant (although transient) tachycardia is often seen in patients with perfusing rhythms given a full arrest dose (dogs and cats, 0.04 mg/kg IV, IM, IT). Because of this effect, the atropine dose can be reduced to one-fourth to half of the arrest dose (dogs and cats, 0.01–0.02 mg/kg IV, IM, IT) in patients with palpable pulses.
- Respiratory arrest may accompany these events, and prompt intubation and manual ventilation are indicated.
- Most patients with a witnessed vagal arrest respond remarkably well to prompt intubation, ventilation, and atropine administration. Full CPR should be instituted if no response to initial therapy occurs.

POSTRESUSCITATION CARE

PREVENTING REARREST

- Many patients who are initially resuscitated have an additional episode of CPA within the first few hours, and often the first few minutes, after ROSC.
- A rapid search for underlying causes of the arrest must be undertaken, and these should be addressed immediately. Special emphasis should be placed on finding reversible disease processes, such as drug-induced hypotension, hypovolemia, hypoventilation, anemia, or electrolyte abnormalities, because these situations are most likely to result in successful outcomes when appropriately treated.

CEREBRAL PROTECTION

- Cerebral ischemia (and subsequent reperfusion) may lead to long-term neurologic dysfunction in patients with CPA and subsequent resuscitation. This process has led to the creation of the acronym CPCR, which stands for *cardiopulmonary cerebral resuscitation*, and reflects the importance of neurologic outcome when the success of resuscitation is assessed.
- One measure to limit progressive neurologic injury in post-CPA patients is head elevation to 30 degrees. This elevation should be accomplished by elevating the entire chest, neck, and head to avoid acute kinking of the neck and possible jugular vein compression.
- **Mannitol** can be given at a dose of 0.25 to 1.0 g/kg IV (dogs and cats) over 20 minutes to treat cerebral edema, improve cerebral microvascular flow, and provide free radical scavenging effects. The diuretic effect of mannitol should not be overlooked and may lead to inadvertent hypotension if appropriate IV fluid therapy is not provided.
- Strategies to optimize the hemodynamic status of the patient, including judicious use of IV fluids, vasopressors, or positive inotropes, should be used and titrated to maintain normotension and to achieve resolution of lactic acidosis.
- Ventilatory status should be evaluated either by $ETCO_2$ or ideally by blood gas analysis, and normocapnia should be maintained. This monitoring helps control increases in intracranial pressure created by hypercapnia-induced cerebral vasodilation and prevents hypocapnia-related cerebral vasoconstriction and diminished cerebral blood flow. Many patients who have protracted periods of CPA do not ventilate effectively in the immediate (<24 hours) postarrest period and require mechanical ventilation to maintain normocapnia.
- Induced hypothermia has been shown to be beneficial in improving neurologic outcome following CPR in humans. Although this is difficult to translate to clinical veterinary patients, overzealous rewarming of mildly hypothermic patients is not recommended.

INTENSIVE CARE

- Patients resuscitated from CPA may have a range of postresuscitation syndromes affecting multiple organ systems. The severity of these abnormalities depends on the duration of the arrest and the condition of the patient before the episode.
- In addition to neurologic dysfunction, postarrest patients often have significant cardiovascular (arrhythmia, myocardial dysfunction, impaired myocardial contractility, hypotension), renal (acute kidney injury), and gastrointestinal (shock gut) sequelae. The low-flow state during CPA and CPR

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creates global ischemia followed by subsequent reperfusion, which may result in systemic inflammation (systemic inflammatory response syndrome), activation of the coagulation cascade, and disseminated intravascular coagulation. There is also the possibility that CPR has created iatrogenic injury (rib fractures, pulmonary contusion) or has resulted in additional management concerns (posthoracotomy or posttracheostomy patients).

- Intensive monitoring and supportive care are required to address these conditions, as well as conditions underlying the arrest. It is common for post-CPA patients to require vasopressor therapy, mechanical ventilation, or other advanced therapy to survive the postarrest period and be discharged from the hospital.

Key Point

Postresuscitation care is essential to the ultimate success of CPR. Intensive monitoring and supportive care are necessary to identify and address the underlying cause of CPA as well as to manage postresuscitation syndromes.

Frequently Asked Questions

WHEN SHOULD CPR NOT BE PERFORMED?

The decision to perform CPR can be difficult, especially when this decision needs to be made in a crisis situation. Retrospective studies have shown that survival to discharge is generally less than 10% for patients with a full CPA. In general, the greatest chance for a successful outcome involves a patient in which a cause for the arrest can be rapidly identified and treated. This is not often the case for patients with advanced or multisystemic diseases. Although many of these patients can be initially resuscitated, survival to discharge is extraordinarily unlikely. Speaking to an owner about a resuscitation code (full CPR, limited CPR, or Do Not Resuscitate) is recommended whenever a critically ill patient with an advanced disease is admitted to the hospital. In this way, futile resuscitation efforts can be avoided, and appropriate end-of-life decisions can be made.

WHAT IS THE NEUROLOGIC OUTCOME OF VETERINARY PATIENTS SURVIVING CPA?

As mentioned previously, the survival of patients with CPA is poor. In addition, many veterinarians have concerns about the potential for neurologic dysfunction in those patients who do survive to hospital discharge. Although this is a major concern in people that have received CPR, a retrospective study demonstrated that 16 of 18 veterinary patients who survived CPA were neurologically normal at the time of hospital discharge and that 1 of the remaining 2 patients was normal within 2 months.

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Chapter

18

Emergency Management and Critical Care

Kari Santoro Beer

HEART FAILURE

- Congestive heart failure (CHF) (commonly regarded as "backward" failure)
  - Patients with CHF generally show respiratory signs (caused by pleural effusion or pulmonary edema) or abdominal distension (caused by ascites).
- Low-output failure (commonly regarded as "forward" failure)
  - Animals with low-output heart failure have adequate intravascular volume but reduced cardiac output and most commonly have signs of weakness or collapse that are typically caused by dilated cardiomyopathy or pericardial effusion.
  - The term *myocardial failure* is used to denote the presence of reduced myocardial contractility (e.g., dilated cardiomyopathy).

CARDIAC RHYTHM DISTURBANCES

- The most common cardiac arrhythmias causing emergency presentations are severe bradyarrhythmias (third-degree heart block, sick sinus syndrome) and tachyarrhythmias. These animals generally show low-output failure and signs of weakness or collapse.

THROMBOEMBOLISM

- Thromboembolic disease typically presents with acute dysfunction of the area of compromised blood supply. In cats with cardiomyopathy, this is most often the hind limbs because of an aortic saddle thrombus, although other limbs can also be affected. Pulmonary thromboembolism may be an acute cause of respiratory distress and low-output heart failure in dogs with a variety of underlying diseases. Thromboembolic disease is also encountered in animals with infectious endocarditis.

Key Point

The ultimate cardiac emergency is cardiopulmonary arrest. The management of cardiopulmonary arrest and strategies for cardiopulmonary resuscitation in small animals are covered separately (see Chapter 18).

ASSESSING CARDIOVASCULAR FUNCTION IN THE EMERGENCY PATIENT

PHYSICAL EXAMINATION

- Historical complaints that support a primary cardiac emergency are variable and include weakness, lethargy, collapse or syncope, as well as cough, tachypnea, or respiratory distress. Additional complaints, such as anorexia, vomiting, and diarrhea, are not uncommon because primary cardiovascular problems may have wide-ranging effects on all major organ systems.
- Physical examination findings consistent with primary cardiac emergencies are variable depending on the specific condition.
  - Mucous membranes may be pale secondary to vasoconstriction or cyanotic secondary to hypoxemia. Capillary refill time is commonly prolonged because of diminished cardiac output and hypoperfusion. Decreased tissue perfusion is more commonly seen in instances of low-output failure compared with congestive failure.
  - Many cases of canine and feline heart failure are accompanied by an audible murmur or gallop. Diminished heart sounds can occur in cases of pericardial or pleural effusion or severe myocardial failure, but they can also occur with severe hypovolemia.
  - Bradyarrhythmias or tachyarrhythmias are common and often associated with irregular rhythms and diminished pulse quality or pulse deficits.
  - Pulsus paradoxus, where pulse strength gets weaker on inspiration, is detected in less than 50% of patients with pericardial effusion.
  - Tachypnea and respiratory distress are often present. In cases of pulmonary edema, auscultation will commonly reveal harsh lung sounds or crackles. In cases of pleural effusion, lung sounds are commonly diminished ventrally.
  - Animals with low-output failure often have a low body temperature and depressed mentation caused by poor oxygen delivery (cardiogenic shock).
  - Jugular pulses or distension is commonly detected in animals with right-sided heart failure, including pericardial effusion.

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DIAGNOSTIC TESTS

- Diagnostic tests include electrocardiography (ECG), pulse oximetry, blood pressure measurement, thoracic ultrasound, chest radiographs, and echocardiography. In special cases, cardiac output monitoring, including the measurement of pulmonary artery occlusion pressures, can be considered.
- A lead II ECG is usually sufficient for the rapid diagnosis of most cardiac rhythm disturbances.
- Pulse oximetry provides a useful estimate of hemoglobin saturation and arterial oxygen content, findings that may be further evaluated by arterial blood gas analysis.
- Blood pressure measurement may be accomplished with Doppler, oscillometric, or invasive techniques and reflects cardiac output and vasomotor tone. It is useful in the recognition of shock states and in monitoring therapeutic interventions.
- Chest radiography is the ideal method to assess size and shape of the cardiac silhouette, pulmonary vasculature, pulmonary parenchyma, and pleural space. It is the gold standard for documenting CHF in the form of pulmonary edema or pleural effusion. However, advancements in the use of ultrasound during triage exam have increased utility of this modality as well.
- Echocardiography provides information about cardiac structure and function. It is typically used to confirm a suspected diagnosis, determine severity of disease, assess myocardial contractility, and detect intracardiac or proximal pulmonary artery thrombi. Echocardiography is particularly useful in assessing patients with pericardial effusion for cardiac neoplasia.
- Recent evidence has shown that some cardiac biomarkers, including natriuretic peptides and cardiac troponins, may be helpful in differentiating dyspnea caused by cardiac compared with respiratory disease, or in cases of specific cardiomyopathies.

Key Point

Animals with emergency problems related to heart disease are often not stable enough for prolonged diagnostic tests. Empiric therapy for the most likely cardiac problem based on signalment, history, physical examination, rapid thoracic ultrasound, and chest radiographs is often given. In many cases, appropriate therapy can be instituted without the need for an immediate echocardiographic examination.

EMERGENCY TREATMENT OF HEART FAILURE

- Treatment of heart failure involves the following:
  - Identification and remediation of underlying causes
  - Elimination of aggravating conditions (i.e., cardiac depressants, hypertension, arrhythmias)
  - Control of congestion
  - Improvement of myocardial contractility
  - Improvement of myocardial relaxation
  - Reduction of cardiac work
  - Reduction of pathologic remodeling and neurohormonal activation

CONGESTIVE HEART FAILURE

- CHF results from elevated cardiac filling pressures that cause pulmonary or systemic venous hypertension and extravasation of fluid into the interstitial space or a body cavity.
- The location of this fluid is dependent on the failing ventricle (left-sided, right-sided, or biventricular failure), and the subsequent signs of CHF relate to the magnitude of fluid accumulation.
- CHF is the result of many cardiac diseases including chronic valvular disease, cardiomyopathies, infectious endocarditis, myocarditis, pericardial effusion, persistent arrhythmia, or congenital cardiovascular anomalies.

LEFT-SIDED CONGESTIVE HEART FAILURE

- Left-sided congestive heart failure (LCHF) results from elevated left atrial pressure. Elevated left atrial pressure may result from mitral valve insufficiency, mitral valve stenosis, or systolic or diastolic dysfunction of the left ventricle. In dogs, LCHF causes pulmonary edema; in cats, it may be associated with either or both pulmonary edema and pleural effusion.
- Clinical signs
  - Clinical signs of LCHF result from pulmonary compromise and include tachypnea, respiratory distress, lethargy, and exercise intolerance. Dogs with pulmonary edema frequently have a cough; however, cough is rare in cats with heart failure.
  - Physical examination findings include harsh lung sounds and crackles in patients with pulmonary edema. Cats with pleural effusion usually have dull lung sounds in the ventral lung fields, especially when compared with the degree of respiratory effort. Severely compromised patients will have respiratory distress, and cyanosis is not uncommon. Most patients with LCHF will be tachycardic and usually have abnormalities on auscultation such as a heart murmur, gallop rhythm, or an arrhythmia; cats can exhibit bradycardia.

Diagnostics

- Confirmation of LCHF is made with chest radiographs to document the presence and severity of pulmonary edema or pleural effusion.
- In dogs, pulmonary edema tends to be most evident at the perihilar region, although all lung lobes may be affected in severe cases. Pulmonary edema in cats does not follow this pattern, and the location of

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affected lung tissue is variable. Pleural effusion in cats may be found either with or without concurrent pulmonary edema. Other changes supportive of LCHF include cardiomegaly, evidence of left atrial enlargement, and dilated pulmonary vasculature, particularly of the pulmonary veins.

- ECG analysis can show a variety of changes, including prolongation of P-wave duration (P mitrale), increased R-wave amplitude or duration, left-axis shift, bundle branch block, supraventricular or ventricular premature complexes or tachycardia, or atrial fibrillation.
- Echocardiography provides definitive information regarding the size of the cardiac chambers as well as information on systolic and diastolic cardiac function.
- Many patients that have LCHF have severe respiratory distress and cannot tolerate a full diagnostic workup. Because of this, therapy is often instituted before obtaining radiographs or an echocardiogram. In these cases, the decision to treat CHF is based on the history, clinical signs, and physical examination findings at presentation. If access to a portable ultrasound machine is available, additional information may be obtained from a brief screening examination of the thorax for the presence of pleural effusion or grossly recognizable changes in cardiac structure (e.g., dilated left atrium, myocardial chamber dimension, or wall thickness) or function (e.g., markedly diminished fractional shortening). Rapid thoracic ultrasound (TFAST, VetBlue) may also show evidence of B-lines or ultrasound lung rockets, which may be supportive of pulmonary edema (versus contusions or pneumonia); LA:Ao ratios can also be assessed.

Treatment

- Emergency treatment of LCHF involves the use of diuretics, vasodilators, and, in some cases, inotropic agents. In addition, oxygen therapy is vital in patients with compromised pulmonary function, and some patients may benefit from the judicious use of anxiolytic drugs. Cats with significant pleural effusion require therapeutic thoracocentesis.

Diuretic therapy

- The goal of diuretic therapy in the treatment of LCHF is to reduce the circulating blood volume, thereby reducing the preload of the left ventricle and left atrial pressure. The primary diuretic used in the acute management of CHF is furosemide.
- **Furosemide** should be administered intravenously if possible, although the intramuscular route may be used in patients without vascular access. The subcutaneous route is not recommended because of likely hypoperfusion and decreased absorption.
- The dose is dependent on the severity of clinical signs and patient response. In dogs, an initial dose of 2 to 4 mg/kg intravenously (IV) or intramuscularly (IM) may be followed by additional doses q1–2h until the respiratory character improves. After improvement, additional doses of 2 mg/kg are typically given at 6- to 12-hour intervals dependent on clinical status. Cats tend to be more sensitive to furosemide therapy, and initial doses of 1 to 2 mg/kg IV or IM q12h followed by 1 to 2 mg/kg q8–12h are recommended after initial clinical response. Alternatively a CRI of 0.5 to 2 mg/kg/h may be used and potentially can produce greater diuresis and less potassium loss.
- Side effects of furosemide include dehydration, azotemia, hypokalemia, metabolic alkalosis, and, potentially, volume depletion.

Vasodilator therapy

- Vasodilator therapy has two purposes. First, venodilation decreases preload by providing additional vascular capacitance. Second, arterial vasodilation reduces left ventricular afterload, thereby reducing myocardial work and promoting forward flow.
- **Sodium nitroprusside** (dogs: 0.5–10 µg/kg/min constant-rate infusion [CRI]) increases the concentration of nitric oxide, a potent vasodilator, and is considered to be a balanced vasodilator providing both venous and arterial dilation. Combined with aggressive diuretic therapy, sodium nitroprusside is very effective in resolving severe pulmonary edema. Because of its potency and the potential for excessive vasodilation and secondary hypotension, blood pressure should be monitored closely during infusion.
  - In all cases, the initial dose should be at the low end of the range, and the dose increased based on clinical response and blood pressure. In general, mean arterial pressure should be maintained above 60 mm Hg, whereas systolic pressures should be maintained above 90 mm Hg.
  - Sodium nitroprusside is light sensitive and may induce precipitation of coadministered IV drugs. Thus it is ideally administered through a separate, light-protected IV set and catheter.
  - Long-term nitroprusside therapy is limited by the production of molecular cyanide, although toxic doses are not usually reached until 36 to 48 hours after onset of therapy.
- **Nitroglycerin ointment (2%)** is a commonly used venodilator in small animal emergency patients. The ointment is applied topically to a clipped area on either the pinna or inguinal region and is dosed according to body size. A 1/8-inch strip is used in cats and small dogs (<10 kg), a ¼-in. strip in medium dogs (10–25 kg), and a ½-in. strip in large dogs (>25 kg). The ointment may be applied q12h for the first 24 to 36 hours of treatment and should be handled with gloves because it may be absorbed transdermally. It is important to remember that continuous use results in tolerance after 48 to 72 hours.

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- Although the use of nitroglycerin ointment is common, it should be noted that topically applied therapy is limited in severe CHF, especially given the profound peripheral vasoconstriction that exists in these patients, and efficacy is questionable.

- **Hydralazine** (dogs, 0.25–2 mg/kg subcutaneously [SQ] or IM, 2 mg/kg IV, or 0.1–0.3 mg/kg/h) is an arterial dilator that causes a marked reduction in afterload and is useful in cases of severe mitral regurgitation. As with other arterial dilators, hydralazine may be associated with hypotension and reflex tachycardia in the face of decreased peripheral resistance.

Positive inotropic agents

- Positive inotropic agents are indicated in managing LCHF associated with systolic dysfunction of the left ventricle. These drugs are administered in conjunction with diuretic and vasodilator therapy. Dilated cardiomyopathy is the most common disease producing this condition; however, myocardial failure secondary to advanced chronic valvular disease, systemic inflammatory response syndrome, sepsis, or end-stage forms of other cardiomyopathies may also result in severe systolic dysfunction.
  - **Dobutamine** (dogs, 2.5–20 µg/kg/min CRI; cats, 2–10 µg/kg/min CRI) is a beta-adrenergic sympathomimetic. Side effects of dobutamine may include tachycardia and ventricular arrhythmias. Cats may have gastrointestinal or neurologic signs associated with administration.
  - **Dopamine** (dogs and cats, 2–10 µg/kg/min CRI) should be used with caution because it has alpha-adrenergic effects at higher doses and may cause deleterious vasoconstriction and tachycardia in the face of diminished myocardial function.
  - **Pimobendan** (0.25–0.3 mg/kg, PO q12h) is a phosphodiesterase inhibitor with both positive inotropic and vasodilatory effects ("inodilator") that may be useful in LCHF once patients can tolerate oral medication.
  - **Digoxin** (0.003–0.005 mg/kg q12h, up to a maximum of 0.375 mg in dogs; 0.007 mg/kg PO every other day initially in cats) is a cardiac glycoside that increases contractility and decreases heart rate. It has a very narrow therapeutic range and can result in toxicity, which generally manifests as cardiac arrhythmias or gastrointestinal signs. The positive inotropic effect is mild. Digoxin has largely been supplanted by pimobendan for inotropic support.
  - Additional options for inotropic support include phosphodiesterase inhibitors such as milrinone. **Milrinone** (dogs, 50 µg/kg slow IV bolus followed by 0.40–0.75 µg/kg/min CRI) is a drug that has both positive inotropic and vasodilatory properties, similar to a combination of dobutamine and sodium nitroprusside. Experience with clinical use in emergency patients is limited, but milrinone may be an effective agent for the short-term management of LCHF associated with systolic dysfunction.

Oxygen therapy

- Oxygen therapy (40%–60% fraction of inspired oxygen) helps to maintain the arterial oxygen content in the face of pulmonary dysfunction (in the form of ventilation-perfusion mismatch) induced by pulmonary edema. Oxygen therapy may also reduce pulmonary vascular resistance by ameliorating hypoxic pulmonary vasoconstriction.
  - An oxygen cage is often the most effective method to administer supplemental oxygen to patients with heart failure, although other alternatives (such as nasal, mask, flow-by, hood, or intratracheal oxygen supplementation) exist. Although an oxygen cage provides a quiet environment that can achieve high concentrations of oxygen, these concentrations decrease rapidly when the cage is opened. In addition, patient monitoring can be impaired for patients in an oxygen cage. Thus an oxygen cage is less effective when patients require frequent treatments or physical examination.
  - Patients should receive oxygen supplementation until their respiratory rate and effort have improved or objective measurements of pulmonary function (i.e., pulse oximetry or arterial blood gas) have returned to normal. If possible, the fraction of inspired oxygen should be tapered over 6 to 12 hours to allow the patient to adjust to breathing room air.
  - For patients with fulminant LCHF and massive pulmonary edema, standard oxygen supplementation may not be sufficient to prevent either respiratory or ventilatory failure. In these cases, only early intubation and mechanical ventilation will provide the respiratory support necessary to sustain life. Mechanical ventilation is a significant commitment for both the owner and clinician; however in many cases, mechanical ventilation may be weaned after only a short period (1–2 days) following aggressive medical management of CHF.
  - The use of anxiolytic agents in the treatment of CHF is common in human medicine and can also be useful in veterinary patients. Low-dose **morphine** (dogs, 0.1 mg/kg IV q4–6h as needed), **butorphanol** (dogs and cats, 0.1–0.2 mg/kg IV q4h), **diazepam** or **midazolam** (dogs and cats, 0.1–0.3 mg/kg IV q4h), or low-dose **acepromazine** (dogs and cats, 0.005–0.05 mg/kg IV q6–8h) can be administered.

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Therapeutic thoracocentesis

- Cats with significant pleural effusion associated with LCHF will experience considerable benefit from therapeutic thoracocentesis. Some cats will tolerate this procedure with minimal restraint, although many cats require some degree of sedation (butorphanol 0.1–0.2 mg/kg IV in combination with diazepam or midazolam 0.1–0.3 mg/kg IV).
  - Thoracocentesis is generally performed between the seventh and ninth intercostal spaces at the level of the costochondral junction, taking care to avoid the internal thoracic arteries. Ultrasound guidance should be used if possible. The area is clipped and aseptically prepared before the procedure is initiated. Thoracocentesis in cats is generally performed with a 21-gauge butterfly catheter attached to a three-way stopcock and a 10- or 20-mL syringe. In extremely obese cats, a 22-gauge needle and extension set is used in place of the butterfly catheter. In larger dogs, an 18- or 20-gauge catheter or needle with a 60-mL syringe may be used.
  - The needle should be introduced just cranial to a rib to avoid the intercostal vessels and nerves and advanced into the pleural space. The needle should be redirected or the procedure terminated once lung tissue is felt at the tip of the needle or fluid is no longer able to be aspirated. Both sides of the chest should be aspirated because bilateral fluid accumulation is found in most cats with pleural effusion secondary to CHF. It is not uncommon to remove 200 to 300 mL of fluid from the thorax of cats with severe pleural effusion.

RIGHT-SIDED CONGESTIVE HEART FAILURE

- Right-sided congestive heart failure (RCHF) is much less common in patients that are seen in the emergency department. An exception is pericardial effusion and cardiac tamponade (see the section on pericardial effusion). RCHF results from elevated right atrial and central venous pressures (CVPs). Conditions that may result in elevated CVP include tricuspid valve insufficiency or stenosis, pulmonic valve insufficiency or stenosis, pulmonary hypertension, and right ventricular systolic or diastolic dysfunction. Rarely right-sided intracardiac masses, massive pulmonary thromboembolism, and heartworm disease with caval syndrome can also induce RCHF.
- Clinical signs of RCHF are related to the presence of pleural effusion, ascites, or peripheral edema that results from increased right atrial pressure. Patients with large-volume pleural effusion may have respiratory distress with dull lung sounds. Patients with ascites will have a distended abdomen and may have respiratory compromise. Additional indications of RCHF are the presence of distended jugular veins and prominent jugular pulses, as well as the presence of a heart murmur (particularly with a maximal intensity at the left heart base or the right side of the chest). Other changes, such as a split S_2 sound or gallop sound, are variable and depend on the underlying disease.
- Emergency therapy for RCHF consists of thoracocentesis for large-volume pleural effusions (see the previous section).
- In animals with tense ascites, abdominocentesis can be performed to reduce pressure on the diaphragm and improve ventilation. Abdominocentesis can be performed with equipment similar to thoracocentesis.
  - The procedure may be performed with the patient standing or in left lateral recumbency, which reduces the likelihood of lacerating the spleen. An area caudal to the umbilicus is clipped and prepared aseptically, and the needle or catheter is induced on or just lateral to the midline. An alternative technique uses two or more short 16- to 18-gauge catheters placed just caudal and to either side of the umbilicus. The animal remains standing during the procedure and fluid is allowed to drain passively. Debate exists about the volume of fluid that can be removed safely from an animal with ascites. Most dogs will tolerate the removal of 50 to 100 mL/kg of ascites without untoward effects.

PERICARDIAL EFFUSION

- Pericardial effusion typically results from an underlying neoplasia, such as hemangiosarcoma, heart base tumors, lymphoma, or mesothelioma. Other causes of pericardial effusion include inflammatory or infectious pericarditis, restrictive pericarditis, coagulopathy, atrial rupture secondary to chronic dilation, and blunt or penetrating thoracic trauma. Small-volume pericardial effusions associated with CHF may also occur, and this phenomenon is relatively common in cats.
- Clinical signs result from cardiac tamponade and RCHF and may include abdominal distension from ascites, tachypnea from pleural effusion, weakness, lethargy, or collapse.
- Physical examination findings include tachycardia and dull heart sounds on auscultation. In some cases, pulsus paradoxus (decrement of pulse strength that occurs during the inspiratory phase of the respiratory cycle) may be recognized. Additional physical examination findings may include jugular distension and prominent jugular pulses, abdominal distension with a palpable fluid wave, and dull ventral lung sounds if pleural effusion is present. Depressed mentation and delayed capillary refill are suggestive of cardiovascular collapse and hypoperfusion in severe cases.
- Diagnostic test results consistent with pericardial effusion and cardiac tamponade include sinus

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tachycardia with diminished complex size on the ECG, with or without the presence of electrical alternans. Electrical alternans describes an alternation in the height of the R wave of the QRS complex and results from beat-to-beat changes in the mean electrical axis as the heart moves within the fluid-filled pericardium. Chest radiographs may demonstrate an enlarged cardiac silhouette as well as distension of the caudal vena cava. Pleural effusion or evidence of metastatic lung disease may be present. The cardiac silhouette often has a classic "globoid" appearance; however, this may not be true in cases of acute pericardial effusion and can also be seen with other cardiac diseases. A brief echocardiographic examination can generally confirm the presence of pericardial effusion.

Key Point

The differentiation of pericardial effusion from pleural effusion on echocardiographic examination can be challenging. Pericardial effusion is recognized by the circular appearance of hypoechoic fluid surrounding the heart. This fluid is bordered by the hyperechoic pericardium. Cardiac tamponade is recognized as diastolic collapse of the right atrium; in some cases, an underlying cause for the effusion is seen, such as a mass involving the right atrium or atrioventricular groove. In patients with pleural but not pericardial effusion, the fluid does not encircle the heart, and lung tissue as well as mediastinal tissue may be seen within the effusion.

- Although blood tests are not often a primary diagnostic tool in the diagnosis of pericardial effusion, documenting the presence of a coagulopathy is vital in the management of those patients in which this is a primary cause of the pericardial effusion. In addition, it should be recognized that patients with more chronic pericardial effusions can develop hyponatremia and hyperkalemia. These pseudo-Addisonian electrolyte changes result from the enhanced antidiuretic hormone secretion and decreased renal perfusion that occurs secondary to decreased effective circulating volume in these patients. These abnormalities rapidly resolve with the resolution of cardiac tamponade.
- The emergency treatment of symptomatic pericardial effusion and cardiac tamponade involves volume expansion and pericardiocentesis.
  - Volume expansion using partial shock doses (30–45 mL/kg) of an isotonic crystalloid transiently increases right atrial pressure. This may improve stroke volume and cardiac output and often results in clinical improvement while steps are taken to perform pericardiocentesis.
  - Pericardiocentesis is the treatment of choice for the initial management of cardiac tamponade and is a lifesaving procedure in many cases. For pericardiocentesis to be performed, the patient is placed in lateral or sternal recumbency. Sedation may be required in some patients, and conservative doses of an opioid in combination with a benzodiazepine are generally well tolerated. Other patients require only local anesthesia.
  - An area from the third to the seventh intercostal spaces is clipped and prepared aseptically, and a local anesthetic is infiltrated over the apex beat, generally at the fifth intercostal space at the level of the costochondral junction. A small stab incision is made in the skin, and a catheter is advanced through the chest wall, just cranial to the sixth rib. In large dogs, a 14-gauge, 12-cm over-the-needle catheter is used, whereas a 16-gauge, 8-cm over-the-needle catheter is used in smaller dogs. In very small dogs or in cats, an 18-gauge, 2-in. over-the-needle catheter may be used.
  - The catheter is slowly advanced toward the pericardium. Pleural fluid may be encountered first (usually straw colored). Entrance into the pericardium is accompanied by a loss of resistance and the presence of fluid flashing back into the catheter hub. This may be recognized sooner if a syringe is attached to the needle and a slight negative pressure is applied as it is advanced or if the needle has been filled with sterile saline before the procedure.
  - Once the pericardium has been punctured, the catheter is advanced off the stylet and attached to an extension set with a three-way stopcock and syringe. The pericardial fluid is aspirated, and fluid should be immediately placed into an activated clotting time tube. This allows pericardial fluid, which should not clot, to be differentiated from peripheral blood, which should clot. The development of a clot in the sample should prompt rapid removal of the catheter system. As the pericardium is drained, additional samples should be obtained for fluid analysis and cytology.
  - The ECG should be monitored during the procedure for the presence of ventricular arrhythmias that occur when the catheter contacts the epicardium. If present, these arrhythmias may be treated by slightly withdrawing the catheter or with a bolus of lidocaine (dogs, 2 mg/kg IV). The ECG is also useful to confirm the effectiveness of the procedure because the heart rate often returns to the normal range and electrical alternans disappears as the heart is decompressed.
  - Once fluid can no longer be aspirated, the catheter is withdrawn. Confirmation of a successful pericardiocentesis may be obtained with a brief recheck echocardiogram. After the procedure, the patient should be monitored for recurrent effusion. Further management of pericardial effusion involves a complete echocardiogram and

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the consideration of more definitive therapy such as a subtotal pericardectomy or a thoracoscopic pericardial window.
- Potential complications of pericardiocentesis include arrhythmias, coronary artery laceration, and lung laceration.

Key Point

In clinically stable patients, a complete echocardiographic examination is preferentially performed before pericardiocentesis because the presence of effusion supplies useful echocardiographic contrast in the attempt to identify cardiac neoplasms. In addition, performing thoracic radiographs before pericardiocentesis (if the patient is stable) allows for better visualization of the pulmonary parenchyma to screen for metastatic disease. Pericardial fluid inevitably leaks into the pleural space after centesis and may obscure the pulmonary parenchyma.

FORWARD (LOW-OUTPUT) HEART FAILURE

- Forward heart failure is the result of impaired myocardial function and results in diminished cardiac output and cardiogenic shock. Common causes of forward heart failure include dilated cardiomyopathy and myocardial failure secondary to end-stage chronic valvular disease, systemic inflammatory response syndrome, sepsis, or doxorubicin toxicity. Significant tachyarrhythmias and bradyarrhythmias may also result in a form of forward heart failure.
- Clinical signs of cardiogenic shock include weakness, lethargy, and collapse. Respiratory signs may be seen if CHF is also present. Physical examination findings associated with forward heart failure include hypothermia, pallor, delayed capillary refill time, tachycardia, and poor pulse quality. Heart sounds may be diminished or a heart murmur and/or gallop rhythm may be heard. The presence of pulmonary edema or pleural effusion will produce characteristic changes on auscultation.
- The clinical diagnosis of forward heart failure is made by finding evidence of decreased myocardial function in combination with hypotension and clinical signs of hypoperfusion. Decreased systolic function is documented using echocardiography and is evidenced by a diminished fractional shortening, increased left ventricular end-systolic dimension, increased E-point to septal separation, and decreased aortic and pulmonic flows. Chest radiographs may document the presence and severity of concurrent CHF, and a lead II ECG will help identify significant rhythm disturbances.
- Treatment of forward heart failure involves efforts to improve myocardial performance and cardiac output. This may be accomplished by ensuring that adequate preload is present and by providing inotropic support.
  - Clinical estimates of preload may be obtained by measuring left and right ventricular filling pressures.
    - Right ventricular filling pressures may be assessed by placing a central venous catheter and measuring the CVP. Normal CVP ranges between 0 and 8 cm H_2O, although wide variability exists among patients, especially those with cardiac disease.
    - Left ventricular filling pressures may be assessed by placing a pulmonary artery catheter and measuring pulmonary capillary wedge (occlusion) pressure (PCWP). Normal PCWP ranges between 5 and 14 cm H_2O. In animals with low CVP or PCWP, judicious fluid therapy may be used to increase preload and cardiac output. If the CVP or PCWP is normal or high, fluid therapy is not likely to be of benefit and may precipitate CHF. In human medicine, controversy exists regarding the benefit of pulmonary artery catheters; this technique is not commonly used in veterinary medicine.
  - Inotropic support is indicated in cases of cardiogenic shock secondary to myocardial systolic dysfunction.
    - **Dobutamine** (dogs, 2.5–20 µg/kg/min; cats, 2–10 µg/kg/min CRI) is generally the first-line agent in dogs because of its ability to increase contractility without significantly increasing heart rate. Dobutamine may also be used in cats, although gastrointestinal and neurologic side effects usually limit utility.
    - **Dopamine** (dogs and cats, 2–10 µg/kg/min) may also be used. Dopamine should be used with caution, however, because it has alpha-adrenergic effects at higher doses and may increase afterload and negatively affect cardiac output. In patients with a pulmonary artery catheter, cardiac output can be measured by the thermodilution method. Combined with measurements of direct arterial blood pressure and calculation of systemic vascular resistance, these techniques allow for the most effective clinical assessment of hemodynamics and the response to therapy.
    - Oral **pimobendan** therapy (0.25–0.3 mg/kg PO q12h) should be considered if intravenous therapy is not possible. Alternatively, digoxin therapy can also be considered, although toxicity is possible because of the narrow therapeutic range, and it is a much weaker positive inotrope than pimobendan.
- See Chapter 16 for further discussion.

CARDIAC RHYTHM DISTURBANCES

- Cardiac arrhythmias are common in emergency patients and may be associated with alterations in

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autonomic tone or responsiveness, drug exposure, electrolyte abnormalities, impaired myocardial oxygen delivery, myocardial trauma or inflammation, or primary myocardial disease.
- In many situations, these cardiac rhythm disturbances represent the cardiac effects of a systemic disease and do not require specific treatment. In other cases, aggressive intervention is required to address unstable rhythms or life-threatening perfusion deficits.
- Physical examination findings consistent with a cardiac rhythm disturbance include bradycardia or tachycardia, an irregular rhythm on auscultation, and the identification of pulse deficits. Depressed mentation or collapse may also be encountered, and syncope may be observed in cases of acute, arrhythmia-induced decreases in cerebral perfusion.
- Confirmation of cardiac rhythm disturbances is achieved by ECG. Obtaining a lead II rhythm strip is often sufficient to diagnose most rhythm disturbances. However, a 6- or 10-lead ECG may be helpful to accurately identify and characterize complex arrhythmias.

Key Point

Cardiac rhythm disorders may be intermittent and may not be noted on a single lead II ECG. Detection of rhythm disorders sometimes requires 24-hour telemetric, Holter, or event monitoring.

BRADYARRHYTHMIAS

- Bradycardia is a relatively uncommon finding in patients seen in the emergency department.
- Clinical signs associated with bradyarrhythmias include weakness, lethargy, depression, and syncope.
- Causes of symptomatic bradyarrhythmias in emergency patients include increased vagal tone, electrolyte abnormalities, hypothermia, drug toxicities, and significant disturbances of the cardiac conduction system.
- ECG rhythms seen in patients with bradycardia include sinus bradycardia, atrial standstill, sinus arrest, and high-grade second- or third-degree AV block.

Sinus Bradycardia

- In emergency patients, sinus bradycardia is most often seen with increased vagal tone.
  - Increased vagal tone may result from intraabdominal or intrathoracic diseases, or from coughing, vomiting, retching, or straining to urinate or defecate. The Cushing reflex, which occurs secondary to head trauma or other causes of elevated intracranial pressure, also produces sinus bradycardia. In this situation, massive sympathetic discharge from the vasomotor center results in systemic hypertension and reflex bradycardia.
- Other causes of sinus bradycardia include absolute or relative drug overdoses, especially of anesthetic agents such as opioids, benzodiazepines, or alpha-2 adrenergic agonists. Cardiac or vasoactive medications, including calcium channel blockers, beta-adrenergic blockers, digoxin, and cholinergic agents may also produce sinus bradycardia either by increasing vagal tone or by reducing sympathetic tone. Hypocortisolemia, as with hypoadrenocorticism, can cause bradycardia, as can severe pulmonary parenchymal disease, likely secondary to J-receptor stimulation.
- Sinus bradycardia may also be associated with hypothermia or severe hypoglycemia (blood glucose <50 mg/dL).
- Management of symptomatic sinus bradycardia generally centers on the identification and treatment of underlying factors. In cases of hypothermia or hypoglycemia, the heart rate and clinical signs often improve markedly once these factors are identified and addressed. Hypothermia is best managed by the use of an indirect heating method such as a warm air blanket. This approach minimizes overheating or detrimental vasodilation that may occur with direct heat sources such as heating pads or hot water bottles. Intravenous fluids that have been warmed to body temperature are also appropriate in patients without contraindications to fluid therapy.
- Although neurologic signs usually predominate over bradycardia, symptomatic hypoglycemia may be treated with a bolus of 0.25 to 1 g/kg of 50% dextrose that has been diluted 50:50 with 0.9% saline. Additional dextrose bolus therapy or a dextrose infusion may be necessary while causes of hypoglycemia (such as insulin overdose, hypoadrenocorticism, paraneoplastic syndrome, systemic inflammatory response syndrome, or sepsis) are investigated.
- Increased vagal tone secondary to intrathoracic disease, intraabdominal disease, coughing, gagging, retching, or straining may cause severe sinus bradycardia that results in cardiovascular collapse or syncope. In these situations, immediate administration of a parasympatholytic agent is necessary. Because of its rapid onset of action and short half-life, atropine is preferred over other agents such as glycopyrrolate. Although the cardiac arrest dose of atropine is 0.04 mg/kg, lower doses (0.005–0.01 mg/kg) are often effective in patients with bradycardia, and these doses are less likely to be associated with rebound sinus tachycardia. Intravenous administration is ideal (rarely, a brief centrally induced exacerbation of bradycardia may occur with this route), although atropine may also be administered via intratracheal, intraosseous, or intramuscular routes. In addition to atropine administration, efforts to

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identify and correct the underlying cause of the increased vagal tone are necessary.

- Atropine is also indicated in treating sinus bradycardia associated with anesthetic agents, as are measures to decrease the depth of anesthesia and to administer specific drug-reversal agents. Opioids may be reversed with naloxone (0.02 mg/kg IV, IM, or SQ) and benzodiazepines may be reversed with flumazenil (0.02 mg/kg IV, IM, or SQ). Dexmedetomidine should be reversed with atipamezole (volume equal to that of the dexmedetomidine given, IM); atropine is not recommended. However, sinus bradycardia associated with the administration of parasympathomimetic agents is also atropine responsive. There are specific recommendations for the management of patients receiving overdoses of cardiac medications such as digoxin, calcium channel blockers, or beta-adrenergic blockers. These guidelines are discussed later.

Second- and Third-Degree Atrioventricular Block

- High-grade second- and third-degree AV block represent severe disruptions of the normal cardiac conduction system. Although structural heart diseases, such as myocardial fibrosis, inflammation, or infiltration, are thought to be responsible for most cases of severe AV block, these rhythms may also be seen in patients with systemic disease or drug toxicities. Drugs associated with second- and third-degree AV block include digoxin, calcium channel blockers, and beta-adrenergic blockers.
- Most emergency patients with high-grade second- and third-degree AV block will have clinical signs relating to decreased cardiac output, such as lethargy, depressed mentation, and syncope, which is common. Rarely, these rhythms will be documented in an otherwise asymptomatic patient. These symptom-free patients (generally cats) often have third-degree AV block with a relatively high ventricular escape rate supporting an adequate cardiac output.
- Medical management of high-grade second- and third-degree AV block consists of initial parasympatholytic therapy followed by sympathomimetic drugs.
  - Initially, **atropine** may be given at the full vagolytic dose of 0.04 mg/kg IV. This is often effective in elevating the rate of discharge of the sinus node but is only rarely effective in improving the AV block in these patients.
  - Alternate therapy consists of beta-1 agonist drugs given in an effort to accelerate the ventricular escape rate. **Isoproterenol** (dogs, 0.04–0.09 µg/kg/min CRI) is commonly used in this situation, although this drug can cause hypotension secondary to vasodilation. An alternative drug is dopamine (dogs and cats, 5–10 µg/kg/min) at the beta-agonist dose. Dobutamine is another beta-1 agonist; however, it has less of a positive chronotropic effect than dopamine.
    - Beta-1 agonists are also indicated in the treatment of beta-adrenergic blocker overdose. Although these drugs may be effective in some cases, an alternative therapy involves the use of **glucagon**. A bolus of 0.15 mg/kg IV may increase cardiac rate and contractility in dogs. If a response is noted following the bolus, then glucagon can be continued as a CRI (dogs, 0.05–0.1 mg/kg/h). Glucagon may be used in dogs with calcium channel blocker overdose at a similar dosage.
- When medical management is ineffective, artificial pacemaker therapy is indicated in the treatment of symptomatic AV block (see Chapter 22).
- Temporary transthoracic or transvenous pacing is considered emergency treatment to stabilize a patient until diagnostic tests are performed and/or definitive therapy is planned. If no underlying cause, such as a drug overdose or toxicity, is identified, patients with symptomatic AV block require the placement of a permanent pacemaker.

Sinus Arrest

- The most common cause of symptomatic sinus arrest is sick sinus syndrome (SSS), a condition characterized by periods of supraventricular tachycardia interspersed with periods of sinus arrest. Although this condition may occur in a number of breeds, it is seen most often in Miniature Schnauzers. Management is similar to patients with symptomatic AV block.

Hyperkalemic Cardiotoxicity

- Although not a primary cardiac abnormality, hyperkalemic cardiotoxicity is a frequent cause of symptomatic bradycardia in emergency patients. It is most common in cats with urethral obstruction, but it also occurs in cases of acute renal failure, uroperitoneum, hypoadrenocorticism, and reperfusion injury.
- Hyperkalemia produces a characteristic set of changes on the ECG; however, the concentration at which these changes are noted is variable. Changes rarely occur if K^+ is less than 6 mmol/L. The sequence of changes involves tented/spiked T waves; flattening of the P waves; prolongation of the PR interval; bradycardia; loss of P waves (atrial standstill); prolongation of the QRS complex; merging of the QRS and T wave complexes to form a "sine wave" pattern; and, finally, ventricular flutter, fibrillation, or asystole.
- The management of life-threatening hyperkalemia involves three phases: immediate cardioprotection, redistribution of serum potassium, and removal of potassium from the body.

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- Immediate cardioprotection is achieved through the use of intravenous **calcium gluconate** (dogs and cats, 50–100 mg/kg as a slow [3- to 5-minute] IV bolus) while the ECG is monitored. Overly rapid administration results in worsening bradycardia and potentially severe ventricular arrhythmias. The onset of action of calcium gluconate is usually rapid (within 5 minutes). The duration of effect is limited, however, and hyperkalemic ECG changes will recur unless steps are taken to reduce the serum potassium level.
- Serum potassium may be transiently lowered by redistribution into the intracellular space with **regular insulin** (dogs and cats, 0.25 units/kg accompanied by dextrose bolus of 0.5 g/kg to help prevent iatrogenic hypoglycemia). Dextrose supplementation is continued in intravenous fluid therapy for 6 to 12 hours to prevent a decrease in blood glucose later in the course of treatment.
  - Alternatives or adjuncts to regular insulin therapy include **sodium bicarbonate** (dogs and cats, 1–2 mEq/kg given over 10–15 minutes) or **terbutaline** (dogs and cats, 0.01 mg/kg IV slowly).
- Definitive management involves removing potassium from the body. This is generally accomplished by establishing urine flow (relieving urethral obstruction, placing a peritoneal drainage catheter) and fluid diuresis. If adequate urine flow is unable to be achieved, patients are considered candidates for either peritoneal dialysis or hemodialysis.

TACHYARRHYTHMIAS

- Tachyarrhythmias are common in emergency patients. Clinical signs associated with pathologic tachyarrhythmias include weakness, lethargy, and collapse. On rare occasions, tachyarrhythmias may result in sudden death. Causes of symptomatic tachyarrhythmias include increased sympathetic tone, toxicities, electrolyte abnormalities, myocardial disease or ischemia, and reentrant circuits within the myocardium. Although isolated or infrequent supraventricular or ventricular premature complexes are often seen in the emergency department, most patients with symptoms have sustained tachyarrhythmias. These rhythms include sinus tachycardia, supraventricular tachycardia (including atrial or junctional tachycardia, and atrial fibrillation), and ventricular tachycardia.

Sinus Tachycardia

- Sinus tachycardia is associated with increased sympathetic tone. Common causes of sinus tachycardia in emergency patients include hypovolemia, hypotension, anemia, hypoxemia, pain, stress, fear, and excitement. No specific treatment is usually required, and treatment is generally contraindicated as it may suppress a lifesaving compensatory response. Rather the heart rate responds when the underlying cause of the tachycardia is identified and addressed. In patients with sinus tachycardia associated with anxiety, anxiolytics may result in a decrease in heart rate.
- The use of beta-adrenergic blockers, such as **esmolol** (dogs and cats, 0.1–0.5 mg/kg IV slowly, followed by 50–200 µg/kg/min CRI) or **propranolol** (dogs and cats, slow IV boluses of 0.02 mg/kg up to 0.1 mg/kg), is indicated in cases of sinus tachycardia secondary to drug or toxin exposures or in cases where all other underlying causes of tachycardia (hypovolemia, hypoxemia, anxiety) have been addressed. Side effects of beta-adrenergic blockers include bradycardia, negative inotropy, and hypotension.

Supraventricular Tachycardia

- Supraventricular tachycardias may be atrial or junctional in origin. Atrial fibrillation is also considered to be an atrial tachycardia. Junctional tachycardias originate from within the AV node or involve reentrant circuits within the AV node. Supraventricular tachycardias are also known as *narrow-complex tachycardias*, as the QRS complex resembles the QRS associated with normal sinus complexes. In rare instances, a wide-complex tachycardia may result from a supraventricular focus because of the presence of bundle branch block in the specialized conduction system.
- Supraventricular tachycardia results from abnormalities within the atrial or junctional myocardial tissue. This may occur because of grossly identifiable diseases, such as atrial dilation in the presence of elevated filling pressures or volume overload, myocardial fibrosis in the presence of cardiomyopathy, or infiltrative diseases such as cardiac neoplasia. In other cases, including most patients with reentrant circuits, supraventricular tachycardia occurs in a heart that appears to be structurally normal. This is also the case in patients where supraventricular tachycardia is caused by electrolyte abnormalities or pharmacologic causes. Common agents that can result in supraventricular tachycardia include digoxin, caffeine, chocolate, and amphetamines or other illicit drugs.
- Patients with supraventricular tachycardia usually have symptoms such as weakness, lethargy, or collapse. Pulse quality is variable but often diminished. In cases of stimulant toxicosis, supraventricular tachycardia may be associated with excitement, hyperesthesia, or seizure activity.
- Treatment of supraventricular tachycardia involves attempting to identify and specifically treat any underlying cause of the tachycardia, such as an electrolyte abnormality or digoxin overdose. In

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addition, variations in sympathetic tone may affect conduction through the AV node, and possible causes of increased sympathetic tone should be investigated. If no underlying cause of the tachycardia can be determined, a vagal maneuver may be attempted.
  - Vagal maneuvers include firm ocular pressure or carotid massage and are undertaken in an attempt to slow conduction through the AV node. In some instances, vagal maneuvers may temporarily terminate a supraventricular tachycardia.
- Pharmacologic therapy of supraventricular tachycardia in an emergency situation involves the use of injectable antiarrhythmic agents. Initial choices for antiarrhythmic therapy include beta-adrenergic blockers, which limit the rate of spontaneous depolarization in ectopic pacemakers, and calcium channel blockers, which slow conduction through the AV node. Both classes of drug have potent negative chronotropic effects and may also have profound negative inotropic properties. These negative inotropic effects are more pronounced when the drugs are given rapidly, in high doses, or in combination with other agents that impair contractility. This consideration is especially important in patients with significant structural heart disease and limited cardiovascular reserve.
  - **Esmolol** (see the dose listed earlier) is generally preferred to other beta-blocking agents, such as propranolol, because of its short half-life that allows rapid titration of effect.
  - The use of a calcium channel blocker is an alternative to beta-blocking agents. **Diltiazem** (dogs and cats, 0.1–0.25 mg/kg slowly followed by 5–20 µg/kg/min CRI) has a less pronounced negative inotropic effect than equivalent doses of esmolol.
- Other choices for antiarrhythmic therapy of supraventricular tachycardia include procainamide and amiodarone.
  - **Procainamide** (dogs, 6–8 mg/kg slowly, followed by 20–50 µg/kg/min CRI) is a fast sodium channel blocker and can be used for both supraventricular and ventricular arrhythmias.
  - **Amiodarone** (dogs, slow [20–30 minute] bolus of 5–10 mg/kg diluted in 5% dextrose in water) acts to prolong the action potential. Amiodarone may cause significant hypotension, vasodilation, and pruritus during IV administration, and long-term use may be associated with hepatic dysfunction in dogs. Its use is generally precluded by its potential adverse effects and the availability of safer, more effective medications as noted above.

Ventricular Tachycardia

- Ventricular arrhythmias are very common in emergency patients, and ventricular complexes are recognized by a wide and bizarre QRS shape. In many cases, such as isolated ventricular premature complexes, these rhythm disturbances do not require treatment. However, treatment is recommended if there are frequent or multifocal ventricular premature complexes; if the coupling interval is very rapid, creating an R-on-T shape; or if there is sustained ventricular tachycardia (rates greater than 180 beats/min) with clinical or hemodynamic sequelae.
- An accelerated idioventricular rhythm is very common in patients with noncardiac disease. This rhythm is recognized as a regular, monomorphic ventricular rhythm that is very similar in rate to the underlying sinus rhythm, and fusion complexes may be recognized during transitions between the ventricular and sinus rhythms. In general, this rhythm does not produce significant hemodynamic abnormalities and does not require therapy.
- Ventricular tachycardias result from abnormalities within the ventricular myocardium and are caused by abnormal automaticity, triggered activity, or reentrant circuits. Ventricular arrhythmias may occur because of primary cardiac disease, such as dilated cardiomyopathy, arrhythmogenic cardiomyopathy, hypertrophic cardiomyopathy, chronic valvular disease, aortic or pulmonic stenosis, myocarditis, or cardiac neoplasia. Ventricular arrhythmias may also be seen in a variety of noncardiac conditions, especially in patients with elevated sympathetic tone or inflammatory mediators, and with electrolyte disturbances such as hypocalcemia and hypokalemia.
- As with supraventricular tachycardia, long-standing ventricular tachycardia may produce myocardial failure in patients with otherwise normal hearts. This tachycardia-induced cardiomyopathy results in chamber dilation and diminished systolic function and can result in CHF.
- Patients with symptomatic ventricular tachycardia usually have symptoms such as weakness, lethargy, or collapse. Paroxysmal ventricular tachycardia may result in syncope or episodes of near-syncope.
- Treatment of ventricular tachycardia should involve attempting to identify and specifically treat any underlying cause of the tachycardia.
- In many patients, ventricular arrhythmias respond to interventions such as fluid resuscitation in patients with hypovolemia, the administration of blood products in anemic animals, the implementation of oxygen therapy in hypoxemic patients, correction of electrolyte and acid-base abnormalities, and the use of analgesics in painful patients.
- Pharmacologic therapy of ventricular tachycardia in an emergency situation involves the use of injectable antiarrhythmic agents. Initial choices for antiarrhythmic therapy include class 1 agents such as lidocaine or procainamide. Alternatives include beta-adrenergic blockers or class III agents such as amiodarone.

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- **Lidocaine** (dogs, 2 mg/kg IV bolus up to 8 mg/kg and followed by 25–80 µg/kg/min CRI) is generally a first-line choice in dogs. Cats are much more likely to have adverse effects (gastrointestinal signs, neurologic signs including seizures), and the dose is significantly decreased to prevent these signs (cats, 0.25–0.75 mg/kg IV bolus followed by 10–20 µg/kg/min CRI).
- **Procainamide** (dogs, 6–8 mg/kg slow IV bolus followed by 20–50 µg/kg/min) may also be used to treat ventricular tachycardia, particularly if lidocaine is ineffective. Rapid administration may cause significant hypotension.
- **Esmolol** (see the dose listed earlier) is generally preferred to other beta-adrenergic blockers because of its short half-life. As with procainamide, esmolol may cause hypotension, which is secondary to its potent negative inotropic effect.
- **Amiodarone** (see the dose listed earlier) may also be used for the treatment of symptomatic ventricular tachycardia; however, vasodilation and systemic hypotension are common during administration in dogs, and amiodarone is generally only used in cases where the previously listed agents are ineffective.
- As mentioned previously, in rare cases, a wide-complex tachycardia may result from a supraventricular focus with concurrent bundle branch block in the specialized conduction system. Lidocaine is unlikely to be effective in this scenario, whereas procainamide, beta-adrenergic blockers, or calcium channel blockers are more likely to be useful in the diagnosis and management of these patients.

Key Point

The shape of ventricular premature complexes and ventricular escape complexes is identical. The use of antiarrhythmics in a patient with ventricular escapes may cause suppression of the escape rhythm with disastrous consequences.

Thromboembolic Disease

- See Chapters 9 and 10 for additional discussion.
- Cardiac emergencies associated with thromboembolic disease may either be the cause or the result of severe cardiac disease. Massive pulmonary thromboembolism may cause heart disease by significantly increasing right ventricular afterload. This process results in severe pulmonary hypertension and may precipitate acute right-sided heart failure and cardiogenic shock. This disease occurs more commonly in dogs than cats and is generally associated with an underlying hypercoagulable state that results in pathologic clot formation. Conditions associated with pulmonary thromboembolism include hyperadrenocorticism, immune-mediated hemolytic anemia, sepsis, disseminated intravascular coagulation, protein-losing nephropathy and enteropathy, heartworm disease, and neoplasia.
- More commonly, emergency patients have thromboembolic disease resulting from cardiac disease. This is especially common in cats with cardiomyopathy and associated left atrial enlargement. These patients are prone to the development of blood clots that subsequently embolize the systemic arterial tree. This condition, known as *feline aortic thromboembolism*, often causes acute limb paresis and subsequent tissue ischemia secondary to obstruction of arterial blood flow.
- Cats most commonly display paraparesis but may also have unilateral hindlimb or forelimb paresis. Cats may also embolize other organs, such as the brain, kidneys, or gastrointestinal tract. CHF may be present, although arterial embolization may occur alone. All affected cats benefit from analgesia and treatment of any concurrent heart failure while in the emergency room. Thrombolytic agents such as streptokinase or tissue plasminogen activator may accelerate clot dissolution; however, their use has not provided definitive evidence of benefit in clinical trials. In addition, these agents may cause significant complications, including severe hemorrhage and fatal reperfusion syndromes.
- Conservative therapy involves the provision of supportive care and analgesia, and some cats will reestablish arterial blood flow and regain function in ischemic limbs within 2 to 3 days. Patients with CHF, more than one limb affected, or low rectal temperatures have a worse prognosis for return of perfusion and limb function. The risk of recurrence is high, with 17% to 50% of patients experiencing a repeat episode.
  - Analgesia in the form of **fentanyl** (cats, 2–5 µg/kg/h CRI for 12–18 hours as a fentanyl patch takes effect), **butorphanol** (cats, 0.1–0.3 mg/kg/h CRI or 0.1–0.3 mg/kg IV q4h), or **buprenorphine** (cats, 0.005–0.015 mg/kg IV q6–8h) is recommended. Other opioids such as hydromorphone, oxymorphone, or morphine may also be used.
  - Anticoagulation with unfractionated or low-molecular-weight heparin may be useful to help reduce clot propagation and is generally well tolerated by cats. **Unfractionated heparin** (cats, 150–250 units/kg SQ q6–8h or as a CRI of 20–50 units/kg/h at a low fluid rate) or **low-molecular-weight heparin** (e.g., enoxaparin, 1 mg/kg SQ q6–12h) is used. Unfractionated heparin will cause prolongation of the partial thromboplastin time (PTT), and a clinical target is to achieve a PTT 1.5 to 2.5 times the normal value. Low-molecular-weight heparin activity can be monitored via a factor Xa assay. In addition, the PTT will be prolonged in patients at risk for hemorrhage secondary to low-molecular-weight heparin administration. All heparinized patients should be monitored for signs of clinical bleeding and should not have jugular venipuncture or cystocentesis performed.

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- Antiplatelet therapy with **aspirin** (5 mg/kg PO q72h) or **clopidogrel** (18.75 mg/cat PO q24h) may also decrease the risk of clot formation. Evidence suggests that clopidogrel is superior to aspirin for preventing repeat episodes in cats.
- In addition, all cats that present to the emergency department with feline aortic thromboembolism should be monitored for hyperkalemia, azotemia, hypotension, cardiac arrhythmias, and signs of cardiovascular collapse that may accompany reperfusion injury.

Frequently Asked Questions

WHY DO ANIMALS WITH PERICARDIAL EFFUSION NOT HAVE LCHF AND PULMONARY EDEMA?

Pericardial effusion results in preload reduction to the heart. The pressure that develops around the heart due to effusion prevents the heart chambers from being able to accommodate venous return to the heart. This effect is most pronounced on the right side because it is the "low-pressure" side of the heart. Therefore with severe pericardial effusion, the right side cannot fill adequately because the pericardial fluid accumulation compresses that side more readily than the left side, and right-sided heart failure develops.

WHAT IS THE PURPOSE OF OXYGEN SUPPLEMENTATION AND SEDATIVE DRUGS IN THE TREATMENT OF CHF WITH PULMONARY EDEMA?

When an animal has severe pulmonary edema, hypoxemia can result. The fluid accumulation within the lung and the hypoxemia stimulate ventilation. The hypoxemia and the fluid accumulation within the lung cause anxiety and decreased lung compliance of the lung, resulting in tachycardia, increased work of breathing because of poor lung compliance, and increased oxygen demand. Oxygen helps relieve the hypoxemia, which can in turn decrease anxiety, helping slow the heart rate. Sedative drugs help relieve anxiety, which decreases another stimulus for tachycardia. This combination of therapies will ultimately increase oxygenation of the blood, slow the heart rate, and decrease oxygen demand by the respiratory muscles, all of which are favorable effects for an ailing heart.

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Chapter 19

Anesthesia of the Cardiac Patient

Ciara Ann Barr

Anesthesia of the patient with heart disease can be a challenge. Most veterinary patients with heart disease that are presented for sedation or anesthesia do not have clinical signs of heart failure. Anesthetic protocols that are routinely used for normal patients without heart disease can result in acute decompensation in patients with heart disease. Differences between dogs and cats in the response to anesthetic and analgesic drugs can compound the complex nature of cardiac anesthesia. In addition, most patients with heart disease may be treated with a variety of cardiac drugs that may interact with anesthetic drugs. This chapter provides a general overview of anesthetic drugs that may be used in dogs and cats with heart disease. Anesthetic considerations for specific cardiac diseases are also presented.

Key Point

Suggested anesthetic protocols for dogs and cats are presented based on the functional classes of heart failure.

GENERAL PRINCIPLES

- The veterinary anesthetist must understand and recognize several factors to provide safe and effective sedation or anesthesia, including the hemodynamic changes produced by heart disease, the possible interactions between cardiac and anesthetic drugs, the presence of arrhythmias, and the potential for anesthetic drugs to predispose to the production of arrhythmias.
- Knowledge of the length of sedation or anesthesia that is desired and recognition of the need for analgesia are also important.
- Virtually all anesthetic drugs directly depress cardiac function, alter vascular tone, or modify normal cardiovascular regulatory mechanisms.
- One "magic bullet" anesthetic protocol that will safely anesthetize any dog or cat with heart disease does not exist.
- Each patient and each form of cardiac disease should be considered on an individual basis to provide the safest sedation, analgesic, or anesthetic protocol.

Key Point

The general rule of thumb is to devise an anesthetic plan that provides minimal cardiopulmonary depression and returns the patient to preanesthetic status as soon and as safely as possible.

PREANESTHETIC CONSIDERATIONS

DIAGNOSIS OF THE ETIOLOGY OF HEART DISEASE

- The decision of which sedation or anesthetic protocol to administer to a patient with heart disease should be made based on the underlying etiology of the disease. Once the cause of heart disease has been determined, specific recommendations can be provided on the choice of sedation or anesthesia.

FUNCTIONAL CLASSIFICATION OF HEART FAILURE

- Three functional classifications of heart failure can be developed based on clinical signs. The decision on whether to immediately sedate or anesthetize a patient with heart disease should begin with placing the patient in one of the three classifications.
- The first classification describes the symptom-free patient that has confirmed heart disease yet is not exhibiting clinical signs of heart failure. Patients that fulfill criteria for this classification can be anesthetized without prior stabilization.
- The second classification describes when mild-to-moderate clinical signs of heart failure are evident at rest or with mild exercise. Stabilization of clinical signs with lack of clinical signs for several days following drug therapy is recommended before sedation or anesthesia. Patients with this classification that require lifesaving emergency surgery should have cardiac drug therapy instituted immediately by parenteral administration and clinical signs optimized as much as possible before anesthesia. Continuous and aggressive monitoring will be required for this classification of patient during and immediately after sedation or anesthesia. Cardiac drug therapy should continue during anesthesia

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and surgery and in the immediate postoperative period.
- The third classification describes when advanced clinical signs of heart failure are immediately obvious. Patients severely affected can have cardiogenic shock, and death or severe debilitation is likely without therapy.

Key Points
- Anesthesia is contraindicated in this third category of patients until clinical signs are stabilized with aggressive drug therapy.
- Informed consent by the client is critical when anesthetizing all patients. Specifically, clients should be advised of the increased risk of death or severe debilitation during or immediately after anesthesia if patients in this third category of heart failure are anesthetized, even following successful, aggressive cardiac drug therapy.

ANESTHETIC RISK CLASSIFICATION

- The anesthetic risk of a patient can be determined based on physical status.
- Five categories of physical status have been developed for veterinary patients based on the classification scheme adopted in human medicine by the American Society of Anesthesiologists (ASA) (Table 19.1).
- Most clinically stable cardiac patients will be ASA I through III, depending on the presence of any other underlying disorders.

Key Points
- Unstable patients with clinical signs of cardiac decompensation and heart failure that fulfill the criteria of ASA IV should not be anesthetized until the cardiac disease has been stabilized.
- There may be patients that are seen in the ASA V category, especially those with a long history of heart disease that is currently refractory to all cardiac drugs. Stabilization of the signs of heart failure may not be possible, and death during anesthesia is more likely.

PREANESTHETIC DIAGNOSTIC EVALUATION AND LABORATORY TESTS

- The patient with heart disease should have a complete diagnostic cardiac evaluation.
- All patients should have the following diagnostic and laboratory tests before sedation or anesthesia.

PHYSICAL EXAMINATION

- Particular attention should be paid to thoracic auscultation of the heart and lungs. The character of the peripheral pulse, jugular veins, mucous membranes, and capillary refill time should be noted, and the peripheral pulse should be palpated simultaneously with thoracic auscultation of the heart sounds to detect pulse deficits.

Key Point

Remember that cats with significant heart disease may not have heart murmurs. Any cat with a heart murmur, gallop, or arrhythmia should not be anesthetized without further workup.

ADDITIONAL TESTS

- Thoracic radiography, an electrocardiogram (ECG), and an echocardiogram should be performed as well. If a complete cardiac diagnostic evaluation has been performed less than 1 to 2 weeks before anesthesia and the patient's physical status has not changed, a physical examination may be the only evaluation that requires repetition.
- Complete blood count and serum chemistries should be performed with particular attention to renal values and electrolytes.

CARDIAC DRUGS AND POTENTIAL ANESTHETIC DRUG INTERACTIONS

Diuretics

- The loop diuretic furosemide is the most commonly used diuretic in patients with heart disease. The

Table 19.1 Classification of Physical Status for Anesthetized Patients

ASA CATEGORY	DESCRIPTION OF PHYSICAL STATUS	EXAMPLE
I	Normal, healthy	No cardiac disease, elective surgery (spay, castration)
II	Mild systemic disease	Compensated heart disease (no cardiac medications), fracture without shock
III	Severe systemic disease	Compensated heart disease (cardiac medications), anemia, fever, compensated renal disease, dehydration
IV	Severe systemic disease and a constant threat to life	Decompensated heart disease, electrolyte imbalance, uncontrolled internal hemorrhage
V	Moribund patient not expected to live with or without surgery	Decompensated heart disease refractory to cardiac drugs, cardiogenic shock, septic shock, terminal malignancy

Adapted from the ASA Physical Status Classification System, © 1995–2014, American Society of Anesthesiologists (ASA).

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most common electrolyte disturbance produced by furosemide is hypokalemia. Hypokalemia can result in tachyarrhythmias or predispose to digoxin toxicity. Furosemide may promote dehydration and predispose the patient to hypotension during sedation or anesthesia.
- The potassium-sparing diuretic spironolactone can result in hyperkalemia if used alone for extended periods of time. Hyperkalemia may result in arrhythmias.
- The thiazide diuretic chlorothiazide has similar side effects as the loop diuretics with long-term use. Hypokalemia and hypomagnesemia may produce or predispose to tachyarrhythmias.

Angiotensin-Converting Enzyme Inhibitors

- Enalapril and benazepril are the most commonly used angiotensin-converting enzyme (ACE) inhibitors. Each results in arterial vasodilation that can be enhanced by acepromazine, isoflurane, and sevoflurane, predisposing to arterial hypotension. ACE inhibitors should **not** be administered within 12 hours of anesthesia as they can cause refractory hypotension. Patients undergoing emergency anesthesia within this time period should have blood pressure monitored via direct arterial catheterization with multiple vasopressors available.

Key Point

Patients receiving ACE inhibitors should **not** be anesthetized within 12 hours of administration if possible.

Digitalis Glycosides

- A common side effect of digoxin administration is ventricular arrhythmias. There is the possibility of an increase in arrhythmogenesis with concurrent use of sympathomimetics (dopamine, dobutamine, norepinephrine, epinephrine) during anesthesia. Isoflurane, sevoflurane, and opioids have not been associated with increased incidence of arrhythmias secondary to digitalis.
- Hypokalemia, most commonly caused by long-term use of loop diuretics, can exacerbate digitalis toxicity. Acute onset of hypokalemia can occur during anesthesia as a result of hyperventilation (hypocarbia and concurrent respiratory alkalosis) and can exacerbate preexisting hypokalemia caused by diuretics.

Key Point

Always obtain a blood digitalis level before anesthesia.

Vasodilators

- Hydralazine is an arteriodilator that can cause reflex tachycardia and fluid and water retention. Tachycardia secondary to use of sympathomimetics (dopamine, dobutamine, norepinephrine, epinephrine) during anesthesia can be exacerbated by hydralazine. Fluid administration must be minimized and monitored closely. Acepromazine, isoflurane, and sevoflurane may exacerbate arteriodilation and predispose to arterial hypotension.
- Prazosin causes arterial and venodilation by alpha-1 adrenergic blockade. Acepromazine is contraindicated, as arteriodilation may be excessive and produce severe hypotension. Inhalant anesthetics may exacerbate arteriodilation and may predispose to arterial hypotension.

Calcium Channel Blockers

- Diltiazem is used to treat supraventricular arrhythmias. Potential side effects include vasodilation, bradycardia, and decreased myocardial contractility. Concurrent use of acepromazine and inhalant anesthetics may exacerbate vasodilation and produce hypotension. Concurrent use of opioids and inhalation anesthetics may exacerbate bradycardia. Decreased myocardial contractility may be exacerbated by propofol and inhalant anesthetics.

Inodilator

- Pimobendan is an inodilator that is used to treat mitral valve disease in dogs and is one of the primary cardiac drugs used to treat dilated cardiomyopathy. There is a potential for hypotension when isoflurane or sevoflurane is used because of their vasodilatory effects when combined with the mild vasodilation caused by pimobendan. The increased contractility caused by pimobendan can be beneficial and thus it should continue to be administered the day of anesthesia.

Antiarrhythmic Agents

- The beta-adrenergic blocking agents propranolol and atenolol are commonly used to treat arrhythmias in dogs and cats and for treatment of hypertrophic cardiomyopathy (HCM) in cats. Potential side effects include bradycardia and decreased myocardial contractile function. Bradycardia may be exacerbated with the use of opioids and inhalation anesthetics. Decreased myocardial contractility may cause hypotension during anesthesia, may be less responsive to sympathomimetics (dopamine and dobutamine), and may be exacerbated by propofol. The risks of hypotension must be weighed against the risks of reflex tachycardia when the medication is discontinued thus it is usually recommended that beta-blockers be continued the day of surgery.

Class I antiarrhythmic agents

- Procainamide and tocainide can have side effects, including decreased myocardial contractility and tocainide can cause possible bradycardia. Decreased myocardial contractility may be exacerbated by propofol.

Nonsteroidal Antiinflammatory Drugs

- Cats with heart disease may be prescribed clopidogrel (or rarely aspirin) to potentially prevent

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thromboembolic disease. Both drugs impair platelet function. Risks of surgical hemorrhage and benefit of prevention of thrombosis associated with continued administration should be discussed between the surgeon and cardiologist.

Combination Drug Therapy

- Many patients with heart disease are administered more than one cardiac drug. The potential side effects of each drug must first be considered individually. Potential side effects of the combination of drugs should be considered next. The addition of anesthetic drugs may introduce a greater possibility of side effects. For example, a dog with compensated mitral insufficiency could have been prescribed digoxin, furosemide, and enalapril. There is a great potential for hypotension produced by excessive arteriodilation, bradycardia, or decreased myocardial contractility based on the combined side effects of each drug. Enalapril should be discontinued the day prior to anesthesia and the lowest levels of inhalant anesthetics should be used to minimize vasodilation and subsequent hypotension.

ANESTHETIC DRUG SELECTION AND SUPPORTIVE CARE

CHOICE OF EITHER SEDATION OR GENERAL ANESTHESIA

- The definition of general anesthesia is the administration of injectable anesthetics, inhalation anesthetics, or a combination to produce hypnosis (sleep), analgesia, and muscle relaxation. General anesthesia is not solely produced by the administration of inhalation anesthetics. Many useful and safe general anesthetic protocols for cardiac patients are combinations of injectable anesthetic agents.

OXYGENATION AND VENTILATORY SUPPORT

- All cardiac patients that are sedated or anesthetized should have oxygen administered in some form. Sedated patients should have oxygen delivered by mask at an insufflation rate of no less than 5 L/min. All intubated patients should be attached to an anesthetic machine to deliver 100% oxygen, regardless of whether an inhalation anesthetic is delivered.
- All cardiac patients anesthetized and maintained with isoflurane, sevoflurane, or injectable anesthetics should be intubated and have ventilatory support provided if needed. Isoflurane and sevoflurane are both potent respiratory depressants and can predispose the patient to hypoxemia or hypercarbia. Hypoxemia and hypercarbia can result in pulmonary vasoconstriction as well as the production or worsening of arrhythmias. Expired carbon dioxide should be monitored with a capnometer (see the section on monitoring and supportive care during sedation and anesthesia) to ensure adequate ventilation.

ANESTHETIC DRUGS THAT ARE CONTRAINDICATED IN PATIENTS WITH CARDIAC DISEASE

- The following use of sedatives and anesthetic drugs is contraindicated in patients with heart disease, regardless of origin. The benefits of convenience, effectiveness, ease of administration, and lower cost do not justify the use of these drugs because of the profound cardiopulmonary depression.

Alpha-2 Adrenergic Drugs

- Dexmedetomidine results in intense vasoconstriction and decreased cardiac output. Decreased heart rate is usually a result of intense vasoconstriction. Administration of an anticholinergic will greatly increase cardiac work.

Induction With Inhalation Anesthetics

- Mask induction with isoflurane or sevoflurane is not recommended in dogs and cats. Most animals become very excited during mask induction, even with adequate preanesthetic medication, which could predispose to arrhythmias and increased myocardial work secondary to the stress response. Isoflurane has a very pungent odor and may result in laryngospasm, especially in cats, though sevoflurane is less pungent. Environmental contamination when isoflurane and sevoflurane are administered by mask is a very important consideration for the safety of all personnel.

ANESTHETIC DRUGS THAT SHOULD BE USED WITH CAUTION

Preanesthetic Medication

Tranquilizers

- The phenothiazine acepromazine is considered a major tranquilizer because of the high reliability of producing mental calming. The primary cardiovascular effect is dose-dependent peripheral vasodilation, with minimal effects on contractility and respiration. Hypotension can occur and is primarily treated with intravenous fluids and, if needed, peripheral vasoconstriction agents (phenylephrine, norepinephrine). The sedative and cardiovascular effects are of long duration (4–6 hours), and the effects are not reversible. Acepromazine can be considered at very low dosages in otherwise healthy cardiac patients that would benefit from decreased afterload (e.g., mitral valve disease) but not those where this would be detrimental (e.g., HCM, aortic stenosis).

Anticholinergics

- Atropine and glycopyrrolate are primarily used to maintain heart rate during anesthesia or sedation and are generally not recommended for routine use unless used with anesthetic drugs that are likely to lower heart rate (opioids) through increased

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parasympathetic tone. The potential side effects include the production of tachyarrhythmias. The increase in myocardial oxygen consumption produced by an increase in heart rate above normal values may predispose the patient with heart disease to focal ischemia and possibly arrhythmias. There is little difference between atropine and glycopyrrolate in the effectiveness of producing an increase in heart rate, though glycopyrrolate will likely have a longer duration.

Intravenous Induction Agents

Propofol

- Propofol causes dose- and rate-dependent cardiovascular and respiratory depression and thus, if used, should be administered slowly and at the lowest dose possible. Propofol is rapidly redistributed, resulting in a rapid recovery.

Dissociatives

- Ketamine can be considered in some cardiac patients where increases in heart rate or blood pressure would be well tolerated. Transient increases in heart rate can predispose to arrhythmias, so these drugs are not recommended in patients with preexisting arrhythmias. Increased heart rate is less severe if administered after preanesthetic medication. Ketamine should not be used as the sole anesthetic agent in cats with HCM. Acute fulminate congestive heart failure has been reported in cats with HCM administered ketamine and combinations of ketamine/diazepam as the sole anesthetic. The combination of tiletamine/zolazepam (Telazol) is not recommended due to the potential long duration of action and inability to reverse the tiletamine.

Inhalation anesthetics

- Inhalation anesthetics should not be used as the sole anesthetic agent to induce (mask induction) and maintain anesthesia. Both isoflurane and sevoflurane are potent vasodilators that could lead to hypotension and increased cardiac work. Both isoflurane and sevoflurane are potent respiratory depressants. If inhalants are used to maintain anesthesia, they should be used at the lowest concentrations possible.

USEFUL ANESTHETIC DRUGS

Preanesthetic Medication

- The benefits of preanesthetic medication in patients with heart disease include reducing preoperative anxiety and stress, providing preemptive analgesia, lowering the requirement for intravenous induction agents and inhalation anesthetics, and ensuring a smooth recovery. Preanesthetic medication can be administered orally, intramuscularly, subcutaneously, or intravenously.

Oral premedication: gabapentin and trazadone

- Gabapentin, an anticonvulsant used to treat neuropathic pain, can be used at higher doses to cause mild sedation in dogs and cats. It is especially effective when combined with trazodone, a serotonin receptor antagonist and reuptake inhibitor. When administered the night prior and morning of a procedure, these medications can reduce the amount of premedication or anesthetic agent needed while minimally impacting the cardiovascular system. It is important to note that trazadone can cause serotonin syndrome when combined with other medications that increase serotonin levels, such as fluoxetine and thus should not be used in combination with these medications.

Benzodiazepines

- Diazepam and midazolam are considered minor tranquilizers because when used alone, benzodiazepines do not produce profound sedation in the normally mentated patient and can cause paradoxical excitement. Benzodiazepines may produce a profound effect on patients with advanced age or disease. The most common use is in combination with an opioid (neuroleptanalgesia). Both drugs minimally depress cardiopulmonary function. Both diazepam and midazolam are absorbed after intramuscular administration. Given that diazepam may produce more pain on injection as it is propylene glycol based, it is recommended to use midazolam for IM administration instead. Clinical effects of midazolam compared with diazepam are identical in dogs and cats. The benzodiazepine antagonist flumazenil is available if the patient is having a prolonged or dysphoric recovery.

Opioids

- The primary use of opioids in veterinary anesthesia is to provide analgesia. Most opioids do not possess profound sedative effects when administered alone; however, when used in combination with tranquilizers for neuroleptanalgesia (see following), adequate sedation can be achieved. Opioids do not affect myocardial contractility or vascular tone, which makes them very attractive for use in patients with heart disease. Opioids can cause bradycardia, which is responsive to anticholinergics such as atropine or glycopyrrolate. In dogs and cats with appropriate mentation, opioids alone do not tend to produce significant respiratory depression; however, all patients receiving opioids should be monitored closely. All effects produced by opioids can be antagonized by administrating naloxone.

Morphine

- Minimal sedation is produced when used alone in normal patients, though profound sedation can occur in compromised patients. Primary side effects

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are vomiting and bradycardia. Bradycardia is usually anticholinergic responsive. Pretreatment with maropitant 1 hour prior to administration can prevent vomiting. Rapid intravenous infusion can cause histamine release and resultant hypotension. Depression of respiration is dose dependent.

Hydromorphone

- Hydromorphone has similar clinical effects and is eight times more potent compared with morphine. They have a potential for decreased heart rate (parasympathomimetic). Vomiting is likely, though less likely than with morphine. Opioid agonists can also be used as an induction agent in compromised patients.

Methadone

- Methadone has similar clinical effects to morphine and hydromorphone; however, it is far less likely to cause nausea and vomiting and thus may be preferred in patients with a high aspiration risk.

Butorphanol

- Butorphanol is an opioid agonist/antagonist that causes minimal cardiopulmonary depression, and is unlikely to produce significant bradycardia. A "ceiling effect" occurs regarding sedation and analgesia. This means that higher doses beyond the recommended maximum dose (approximately 0.8 mg/kg) do not produce more sedation or analgesia. Vomiting is a rare side effect. It is a very poor analgesic for moderate-to-severe pain.

Buprenorphine

- Buprenorphine is a partial opioid agonist that is generally a poor sedative when used alone, though slightly more effective in a neuroleptanalgesia combination. There is minimal cardiopulmonary depression, and a ceiling effect occurs similar to butorphanol. The onset of action is 20 to 30 minutes, and there is a long duration of effect. Buprenorphine provides mild-to-moderate analgesia, and given its long duration of effect can interfere with full mu agonists. Thus is not recommended as a premedication prior to a severely painful procedure (e.g., laparotomy, thoracotomy).

Fentanyl

- Fentanyl is an opioid agonist that is 100 times more potent in producing analgesia when compared with morphine. The onset of action is very rapid, and it can be used as an intravenous coinduction agent in dogs. The duration of action is extremely short, making fentanyl an ideal agent for a continuous-rate infusion (CRI) to help maintain general anesthesia. Bradycardia is more likely to occur and responds to anticholinergic administration (preferred) or a decrease in the rate of infusion. Fentanyl may accumulate after 4 hours of infusion, thus resulting in prolonged recoveries.

Remifentanil

- Remifentanil is an ultra-short-acting opioid with a similar potency to fentanyl. It has the advantage of not accumulating regardless of duration of infusion, and thus resulting in rapid recoveries. It can cause significant bradycardia, and bolus administration should be avoided. Bradycardia can also result from CRI and can be treated with anticholinergics.

Neuroleptanalgesia

- *Neuroleptanalgesia* is defined as the effect produced by the combination of a tranquilizer and an opioid. The neuroleptanalgesia combinations that are recommended for patients with heart disease include any combination of a benzodiazepine and an opioid. Intravenous or intramuscular administration may be used to produce an effect.

Opioid-benzodiazepine combinations

- The preferred neuroleptanalgesia for patients with heart disease is the combination of an opioid and a benzodiazepine. Sedation occurs more reliably with opioid agonists (morphine, hydromorphone, fentanyl, remifentanil) as compared to partial opioid agonist (buprenorphine) combinations. It is, however, possible to see excitation following administration of a full mu agonist; thus a mu antagonist and kappa agonist (butorphanol) may be preferable in patients that are not in pain or undergoing painful procedures. Intramuscular administration produces effects within 15 minutes. Panting is a prominent feature when opioid agonists are used in dogs (not cats), and respiratory depression can be pronounced. Bradycardia is more likely with opioid agonist combinations and is responsive to anticholinergics.

Key Point

A neuroleptanalgesia combination including acepromazine will produce a more profound sedative effect than combinations with benzodiazepines; however, acepromazine has a long duration of effect, including vasodilation, and it has no reversal agent. Neuroleptanalgesia combinations including benzodiazepines may be much less effective in excited or nervous dogs and cats. The clinician must weigh the risks and benefits of including acepromazine in patients with mild cardiac disease. Acepromazine should not be used in patients with moderate-to-severe cardiac disease.

Intravenous Induction Agents

Propofol

- Propofol is classified as a phenolic compound unrelated to opioids, barbiturates, or steroid anesthetics. Propofol induction is characterized as a very rapid and smooth induction with a very rapid and smooth recovery. Noncumulative effects make propofol an ideal drug for CRIs. Transient decreases in arterial blood pressure occur and are produced by a decrease in myocardial contractility. A reflex increase in heart rate is likely. Apnea can be profound, and it is closely associated with speed of injection. Use of preanesthetic medication greatly

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reduces the dose of propofol required for induction of anesthesia and reduces the possibility of decreases in blood pressure.

Alfaxalone

- Alfaxalone is a synthetic neurosteroid solubilized in cyclodextrin, which can be used for induction or maintenance of anesthesia via infusion. Alfaxalone also causes dose- and rate-dependent cardiovascular and respiratory depression, although these effects may be less pronounced than with propofol. The dose should be minimized by concurrent use of cardiovascular sparing premedications such as benzodiazepines and opioids. Alfaxalone can be used as an infusion to maintain anesthesia in patients where total intravenous anesthesia is desired. Alfaxalone may be preferable to propofol in patients with mild cardiovascular disease, as cardiac output is typically preserved despite vasodilation due to an increase in heart rate. This study was in healthy dogs but shows that alfaxalone preserves HR as compared to propofol thereby counteracting the effects of vasodilation on MAP & CO. Alfaxalone should not be used, however, in patients with severe cardiac disease. Potential side effects in cats include opisthotonos, paddling activity, and rough recovery when higher doses are used, especially if administered without premedication.

Etomidate

- Etomidate is an imidazole derivative unrelated to barbiturates and opioids. Etomidate induction is characterized as a very rapid induction with a very rapid and smooth recovery. Induction with etomidate results in a less desirable induction and recovery if administered alone without preanesthetic medication. Severe myoclonus activity can occur when used alone. Minimal cardiopulmonary depression and minimal effect on cardiac electrical activity make etomidate an ideal intravenous induction agent for the less stable patient with heart disease after appropriate preanesthetic medication. Etomidate is prepared in a propylene glycol base and has a high osmolality and dilution is recommended prior to administration. Etomidate causes cortisol suppression thus should not be administered to septic patients, Addisonian patients or to any patients as a CRI.

Dissociatives: ketamine and tiletamine/zolazepam

- Ketamine can be used as an induction agent in patients with mild-to-moderate heart disease, but it should not be used alone. Always combine ketamine with diazepam or midazolam to minimize adverse effects of rigidity. Induction with ketamine and diazepam or midazolam will increase heart rate, maintain arterial blood pressure, and have minimal effects on respiration, although apnea has been reported. Potential side effects include myoclonus activity and rough recovery. Cats with HCM should not be administered ketamine or ketamine-diazepam as sole agents.
- The effects of Tiletamine and Zolazepam (Telazol) are similar to ketamine-diazepam when administered as an intravenous bolus for induction. There are longer and potentially rougher recoveries than ketamine-diazepam, especially when used as a sole agent without preanesthetic medication (NOT recommended for routine use). Preanesthetic medication is highly recommended before use of tiletamine-zolazepam. Higher doses will be required if no preanesthetic medication is administered, and there is a greater potential for longer recoveries. There are likely the same considerations in cats with HCM as with ketamine combinations.

MAINTENANCE OF ANESTHESIA

Inhalation Anesthetics

Isoflurane and sevoflurane

- Each inhalation anesthetic has a very similar clinical effect of maintenance of anesthesia and rapid recovery. The main cardiovascular effect is dose-dependent peripheral vasodilation, which is the primary mechanism of hypotension induced by isoflurane and sevoflurane. A general rule is to administer the lowest effective concentration of isoflurane or sevoflurane that will maintain a surgical depth of anesthesia. The use of preanesthetic medications and intravenous induction agents is highly recommended and will lower the amount of inhalant necessary to maintain a surgical depth of anesthesia. Each are potent respiratory depressants that can be additive with opioids, and mechanical ventilation may be necessary to prevent hypoxemia and hypercarbia.

Nitrous Oxide

- Nitrous oxide used in combination with oxygen cannot alone produce anesthesia. Therefore it can be used as an adjunct to inhalation anesthesia only to reduce the inhalation anesthetic requirement. However, safety and environmental considerations preclude the widespread use of nitrous oxide.

Key Point

Utilize opioids and benzodiazepines when possible to reduce the amount of inhalant anesthetic needed.

Injectable General Anesthesia

- Injectable anesthetics can be used to maintain a surgical plane of general anesthesia. The definition of general anesthesia is the production of sleep, muscle relaxation, and analgesia. All three criteria can be met effectively and safely with injectable anesthetics. Specific examples will be offered at the end of this chapter, though general concepts of using all injectable agents are offered below.

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Preanesthetic medication and propofol

- The neuroleptanalgesic combination of an opioid and a benzodiazepine is administered intramuscularly.
- The induction and CRI of propofol consists of an initial induction dose of 1 to 4 mg/kg IV, followed by a CRI administered by syringe pump at a rate of 0.14 to 0.4 mg/kg/min IV, depending on other anesthetic drugs used as preanesthetic medication and the achieved effect. Higher infusion rates may be required to maintain surgical plane of anesthesia and to maintain an endotracheal tube.
- Intermittent boluses of propofol can be used instead of a CRI for short-term maintenance of general anesthesia. Administer propofol by slow bolus at a dosage of 0.5 to 1.0 mg/kg IV following initial induction dose, depending on other anesthetic drugs used as preanesthetic medication.

Preanesthetic medication and alafaxalone

- The neuroleptanalgesic combination of an opioid and a benzodiazepine is administered intramuscularly.
- The induction and CRI of alfaxalone consist of an initial induction dose of 1 to 2 mg/kg IV, followed by a CRI administered by syringe pump at a rate of 0.07 to 0.15 mg/kg/min IV, depending on other anesthetic drugs used as preanesthetic medication and the achieved effect. Higher infusion rates may be required to maintain a surgical plane of anesthesia and to maintain an endotracheal tube.
- Intermittent boluses of alfaxalone can be used instead of a CRI for short-term maintenance of general anesthesia. Administer alfaxalone by slow bolus at a dosage of 0.25 to 0.5 mg/kg IV following initial induction dose, depending on other anesthetic drugs used as preanesthetic medication.

Preanesthetic medication and ketamine-diazepam

- The neuroleptanalgesic combination of an opioid and a benzodiazepine is administered intramuscularly.
- The induction dose of ketamine is 2 to 5 mg/kg combined with 0.1 to 0.4 mg/kg of midazolam or diazepam. In general, one-quarter to one-third of the initial induction dose can be administered as an intermittent bolus, depending on other anesthetic drugs used as preanesthetic medication.

Preanesthetic medication and fentanyl

- The neuroleptanalgesic combination of an opioid and a benzodiazepine is administered intramuscularly.
- Deep sedation can be maintained with a loading dose of fentanyl of 2 to 5 µg/kg IV followed by a CRI of 0.2 to 0.4 µg/kg/min. This technique may provide sufficient deep sedation for minimally invasive procedures, but may necessitate intubation and ventilation.

SPECIES DIFFERENCES (DOG COMPARED WITH CAT) IN ANESTHETIC DRUG EFFECTS

Tranquilizers

- Compared with dogs, cats are less responsive to the mental calming effects of an equivalent dose of acepromazine when it is used as a single agent.

Opioids

- Cats are more likely to become excited from the effects of opioids and, at times, to neuroleptanalgesic combinations of diazepam and an opioid.
- Cats do not have as profound sedative effects from neuroleptanalgesic combinations. Some dogs become laterally recumbent after certain neuroleptanalgesic combinations, whereas cats rarely respond in the same manner. The general rule is that an effective neuroleptanalgesia in cats occurs when the cat assumes sternal recumbency, is very amenable to mild restraint, and has mydriasis.
- Vomiting occurs less frequently in cats with the neuroleptanalgesia combination, though it occurs frequently with hydromorphone used alone.
- Dogs have miosis when an opioid is administered, and cats have mydriasis.

Dissociatives

- The dissociatives were historically the primary class of anesthetic drugs recommended for chemical restraint in cats. However, given the potential for increased myocardial work with ketamine or tiletamine, especially in cats with HCM, alfaxalone may be preferrable to ketamine when intramuscular chemical restraint is required.

Propofol

- There is evidence that multiple exposures (consecutive days) of cats to propofol can result in oxidative injury to feline red blood cells. One anesthetic episode of propofol (induction, CRI, or intermittent boluses) will not produce oxidative injury to feline red blood cells. Propofol should not be used as an anesthetic technique for consecutive, multiple-use therapy as in radiation therapy or daily bandage care in cats.

Alfaxalone

- Alfaxalone can be administered IM in combination with an opioid to provide chemical restraint to cats with less cardiovascular impact that ketamine or Telazol. Alfaxalone administered IM at 0.5 to 2 mg/kg combined with an opioid provides heavy sedation or general anesthesia depending on the dose and individual variability within 10 minutes. Alfaxalone should not be pre-mixed with midazolam as it can precipitate in the syringe.

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ADJUNCT TECHNIQUES

Local and Regional Anesthesia/Analgesia

- Local and regional anesthesia/analgesia techniques are highly effective at reducing the amount of inhalation anesthetic required to maintain anesthesia. Many techniques are available, and the specific technique is dependent on the location of the surgical procedure. Please refer to specific anesthesia and analgesia texts for a description of the available techniques.

Local anesthetic drugs

- Lidocaine (2%) and bupivacaine (0.5%) are the most commonly used local anesthetics. Lidocaine has a rapid onset (5 minutes) and short duration (60 minutes) of action. Bupivacaine has a longer onset (15–20 minutes) and duration (4–6 hours) of action as well as a higher risk of cardiotoxicity with high plasma concentrations (ex. accidental IV injection). All nerve types are blocked with local anesthetics. Therefore regional analgesia techniques such as lumbosacral anesthesia will result in temporary rear limb paralysis and segmental vasodilation.

Opioids

- Morphine can be used in lumbosacral epidural techniques for prolonged analgesia. However, morphine should not be used alone to provide surgical anesthesia because morphine only provides analgesia and does not completely block sensation. The onset of action is up to 1 hour, and analgesia has been reported to be up to 12 to 24 hours. Movement of limbs is maintained because motor nerves are not affected by morphine. Epidural morphine administration may be associated with urinary retention; thus urine output should be monitored for 24 hours following administration.

Infiltration techniques

- Lidocaine (2%) can be infiltrated subcutaneously to a maximum dose of 4 mg/kg in dogs and 2 mg/kg in cats. Lidocaine can be diluted to 1% to obtain more total volume to block a larger area.
- Liposomal bupivacaine (NOCITA) can be used at surgical closure to provide up to 72 hours of local anesthesia to the site.

Regional techniques

Lumbosacral epidural: dogs

- Anesthesia or analgesia is produced caudal to the umbilicus.
- **Morphine** used as a sole agent is administered at 0.1 mg/kg (diluted to 0.1 mL/kg with sterile saline depending on the concentration available). Analgesic effects should be expected primarily during the postoperative period if the surgery is not lengthy and should not be relied on during surgery. There is no sensory or motor blockade produced by epidural administration of opioids. Effects are limited to long-acting analgesia without effects on motor activity.
- **Lidocaine (2%)** used as a sole agent is administered at Š4 mg/kg (0.2 ml/kg) before surgery. Minimal residual analgesia occurs following surgery, caused by the short duration of action.
- **Bupivacaine (0.5%)** used as a sole agent at 1 mg/kg (0.2 mL/kg) should be administered 15 to 20 minutes prior to surgery. Residual analgesia and motor blockade should be expected if surgery is completed within 6 hours of epidural administration.
- A combination of **morphine** and **bupivacaine (0.5%)** can be used to provide surgical and postoperative analgesia. Morphine (0.1 mg/kg) is diluted with bupivacaine (0.5%) to provide a final volume of 0.2 mL/kg.
- Occasionally an epidural technique in dogs results in the appearance of cerebrospinal fluid in the spinal needle. There is no cerebrospinal fluid within the epidural space; therefore the spinal needle has entered the subarachnoid space. Either the anesthetist can remove the spinal needle and attempt the procedure again or half the agents can be administered in the subarachnoid space. Administration of local anesthesia in the subarachnoid space is called *spinal anesthesia*.
- A common complication of epidural anesthesia is inadvertent needle puncture of a blood vessel. The local anesthetic combination should not be administered if blood enters the spinal needle.

Lumbosacral epidural: cats

- A major anatomic difference in cats compared with dogs is that the spinal cord terminates in the sacral vertebral segments in cats compared with the caudal lumbar (L4–5) in dogs. Lumbosacral epidural techniques are more difficult in cats and the chance of entering the subarachnoid space is more likely in cats. Administer half the volume of local anesthetic if cerebrospinal fluid is obtained in the spinal needle. There is also the possibility of spinal cord injury in cats. As a result, sacrococcygeal epidural may be a preferred technique. With this technique, the epidural space is entered between the sacrum and first coccygeal vertebrae. At this level, there is no chance of damaging the spinal cord or entering the subarachnoid space.
- The combinations of local anesthetics and opioids used in dogs are the same for cats.

Intercostal nerve blocks

- Regional anesthesia for a lateral thoracotomy can be obtained by placing the local anesthetic at the dorsal-most aspect of the intercostal nerves at the site of incision and two intercostals spaces, cranial and caudal. The maximum dose of bupivacaine (2 mg/kg) should not be exceeded.

Intrapleural analgesia

- Regional anesthesia for a lateral thoracotomy can be obtained by placing the local anesthetic within

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the pleural space after surgery. **Bupivacaine** (0.25%; 1.5 mg/kg undiluted) is administered through a thoracostomy tube or by a pleurocentesis puncture, and the patient is then placed surgery side down for 15 to 20 minutes to permit the adequate onset of action of bupivacaine.

Key Point

Intrapleural administration of bupivacaine is painful in the nonanesthetized patient and should be administered with extreme caution in conscious patients.

Neuromuscular Blocking Agents

- Neuromuscular blocking agents (NMBAs) block effects of acetylcholine at the neuromuscular junction, resulting in complete paralysis of all skeletal muscle when administered at recommended doses. Use of NMBAs is reserved for specific instances in which muscle paralysis is required, such as for external pacing during placement of a permanent pacemaker. Ventilatory support and use of anesthetic drugs to produce unconciousness are mandatory when using NMBAs.
- **Atracurium** (0.25 mg/kg IV initially and 0.1 mg/kg IV for repeated administration) is a short-acting NMBA with a duration of action from 20 to 25 minutes. Atracurium is metabolized by Hoffman degradation in plasma and does not require hepatic metabolism or renal excretion. Hypothermia and acidosis will prolong the effect of atracurium. Atracurium administration may be associated with histamine release; however, hypotension and tachycardia are not typically observed clinically. **Cis-atracurium**, the cis-isomer of atracurium, results in less histamine release and thus may be a safer alternative to atracurium. **Cis-atracurium** has a similar duration of effect and is dosed at 0.1 mg/kg IV initially and 0.05 mg/kg IV if repeated dosing is required.
- All patients receiving **NMBAs** should be monitored closely for signs of insufficient depth of anesthesia (rapid changes in heart rate and blood pressure) as well as depth of neuromuscular blockade. A nerve stimulator should be utilized to monitor train of four (TOF) as an indicator of depth of neuromuscular blockade. If inadequate recovery of motor function is noted (TOF < 90%), patients should be reversed. Reversal is accomplished with neostigmine (0.02 mg/kg IV), which can cause bradycardia; thus atropine (0.02 mg/kg IV) may be administered in conjunction.

MONITORING AND SUPPORTIVE CARE DURING SEDATION AND ANESTHESIA

- There are two aspects of monitoring during anesthesia: anesthetic depth and cardiopulmonary parameters. Anesthetic depth is best monitored by assessment of jaw tone and eye position. An adequately anesthetized patient has moderate jaw tone and a ventral-medial eye position. A deeply anesthetized patient has extremely loose or no jaw tone and a central eye position. A clear sign of inadequate anesthetic depth is gross, purposeful movement. Heart rate, respiratory rate, and jaw tone can all increase before movement and should be monitored continuously. The use of monitoring devices to assess cardiopulmonary parameters is highly dependent on several factors, including the severity of cardiac disease, the length of anesthesia, and the procedure being performed. Minimal equipment will be required for sedation and short procedures compared to anesthesia for major surgical procedures, both cardiac and noncardiac. Physical parameters of heart rate, respiratory rate, mucous membrane color, capillary refill time, and pulse character should be monitored at regular intervals of no more than 5 minutes during anesthesia and sedation of any duration, even if monitoring equipment is used.

Key Point

Gross purposeful movement is not possible when patients are paralyzed with NMBAs; thus monitoring anesthetic depth is challenging and it is essential that adequate analgesia is provided.

NONINVASIVE MONITORING

Electrocardiography

- Continuous ECG monitoring should be performed in all patients with heart disease during sedation and anesthesia of any duration. The decision to continue ECG monitoring during the postoperative period should be determined based on the procedure and the status of the patient.

Arterial Blood Pressure Measurement

- Indirect methods are less accurate than direct measurements. However, monitoring the trends of indirect arterial blood pressure can provide valuable information. The two indirect methods are Doppler ultrasound and oscillometric.

Doppler ultrasound method

- Systolic arterial blood pressure can be obtained by means of the Doppler method. Accuracy of obtained values is highly dependent on several factors (cuff size, skin thickness, contact of crystal, positioning of limb, vasoconstriction); therefore trends in blood pressure are monitored. The advantage of Doppler ultrasound is that active arterial blood flow can be heard at all times.

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Key Point

Do not make therapeutic decisions regarding fluid therapy solely on any indirect method of blood pressure monitoring in cats and small dogs, especially in cats with hypothermia.

Oscillometric method

- Systolic, diastolic, and mean arterial blood pressure and heart rate are determined. Mean arterial pressure is the most accurate, though values can be under- or overestimated. Accuracy of obtained values is highly dependent on several factors (cuff size, skin thickness, contact, or positioning of cuff in relation to the artery, choice of artery, positioning of limb, vasoconstriction); therefore trends in blood pressure are monitored. Oscillometric blood pressure monitoring may be inaccurate in small dogs, cats, and animals in states of hypotension or with arrhythmias, despite advances in technology.

Pulse Oximetry

- Pulse oximetry provides indirect determination of arterial oxygenation. Active pulsation of an arterial bed is required to determine oxygenation. Pulse oximetry is inaccurate in states of hypotension and peripheral vasoconstriction (hypothermia, pain). Pulse oximetry can be very accurate in the ideal circumstance. However, falsely low readings may not equate to pulmonary dysfunction, especially under general anesthesia after an extended period of time. Factors that will decrease the pulse oximetry reading accuracy include a cold, dry tongue during general anesthesia, pressure exerted on the tongue, and interference from room light. Any question about low pulse oximetry readings should be confirmed with an arterial blood gas sample.

Capnometry

- Capnometry determines the partial pressure of exhaled carbon dioxide, which is closely related to arterial partial pressure of carbon dioxide. It indirectly provides information on cardiac output. Exhaled carbon dioxide is dependent on adequate perfusion of the lungs (delivery of carbon dioxide to the lungs). Hypoventilation (increased partial pressure of carbon dioxide in arterial blood) can be detected on a breath-by-breath basis. Capnometry requires intubation in most cases for the most accurate values, though tight-fitting facemasks or nasal cannulas can provide an environment to obtain information on ventilatory status.

INVASIVE MONITORING

- The more invasive and complicated surgical procedures should incur more invasive monitoring techniques. Although the noninvasive techniques can provide general trends, invasive monitoring can provide more accurate data concerning cardiovascular function. Direct monitoring is less likely to provide false-negative values and is less likely to fail during anesthesia.

Direct Arterial Blood Pressure Measurement

- A catheter placed in a peripheral artery (dorsal pedal most common) requires specialized monitoring equipment and technical skill. It is technically more difficult to place a peripheral arterial catheter in cats and small dogs (<5 kg). The femoral artery is much larger and less difficult to place a catheter in, although maintaining the catheter is much more difficult. It is not recommended to place a catheter in the lingual artery because of the possible complication of a hematoma that could cause postoperative airway obstruction.

Key Point

Technologic advances in multiparameter monitoring devices have provided veterinarians the ability to perform invasive, accurate blood pressure monitoring.

Central Venous Pressure

- The central venous pressure (CVP) monitors right-sided heart function and is an indicator of intravascular volume. CVP uses a properly placed jugular catheter with the tip in the thoracic cavity. Inexpensive equipment (manometers) can be used to measure CVP. The same device used to monitor direct arterial blood pressure can be used to monitor CVP. CVP can be a valuable tool during anesthesia or in the postoperative period to detect early cardiac failure or fluid overload.

Arterial and Venous Blood Gas

- Arterial blood gas monitoring provides information on ventilation ($PaCO_2$) and oxygenation (PaO_2). Venous blood gas monitoring from a central vein (jugular, cranial vena cava, pulmonary artery) provides indirect information on perfusion of tissues and cardiac output. Arterial and venous blood gas monitoring combined with cardiac output information can be used to calculate oxygen delivery variables (see later). Devices used for blood gas analysis are affordable and are commonly being used in clinical practice. The lingual vein can be used during general anesthesia to collect blood for analysis. Though venous, the values are closely related to arterial values because of the close association with capillary beds.

Advanced Cardiovascular Monitoring: Cardiac Output

- Cardiac output monitoring utilizes pulmonary artery catheterization to obtain cardiovascular values that can provide information regarding

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ventricular function (see Table 19.1). Pulmonary artery catheterization is not without risk. Methods to measure cardiac output via less invasive means are available but are not yet routinely utilized.

Key Point

- Cardiac output is not synonymous with arterial blood pressure. In Table 19.1, cardiac output is a determinant of the calculation for blood pressure.

FLUID THERAPY

- Most patients with heart disease that are anesthetized with inhalation anesthetics will require intravenous fluid support. The fluid of choice for the patient with heart disease is usually a balanced isotonic crystalloid fluid (Plasmalyte, Lactated Ringers Solution). The rate of fluid therapy administration, however, is far more important than the type of fluid administered. The rate should be less than the recommended fluid rate during anesthesia of normal, healthy patients (3–5 mL/kg/h). A general rule would be to decrease the fluid rate to approximately one-fourth to one-third of the rate for a normal patient, yielding a rate of 1 to 2 mL/kg/h. Routine procedures that require less than 1 hour of general anesthesia may not require IV fluid support. Less stable patients with heart disease and patients anesthetized for emergency surgery with signs of heart failure should have CVP measured to aid in monitoring fluid therapy. Colloid fluids should be used with caution in patients with heart disease, especially if a bolus of colloids is to be administered.

VASOPRESSORS AND INOTROPES

- Patients with heart disease may require blood pressure support under general anesthesia due to the cardiovascular depressant effects of many anesthetics. Often dopamine, a mixed alpha-beta agonist is used to improve cardiac contractility at lower rates (5–10 g/kg/min) or cause vasoconstriction at higher rates (10–20 µg/kg/min). Tachyarrhythmias or bradyarrhythmias can be noted with dopamine and may require treatment or conversion to a different vasopressor or positive inotrope. Dobutamine, a beta agonist, may be employed to improve cardiac contractility; however, it can be associated with vasodilation and thus is unlikely to improve blood pressure without the addition of a vasopressor. Tachyarrhythmias may be noted with higher rates of dobutamine infusion. Norepinephrine has primarily alpha-agonism effects, with mild beta agonism and thus may be added as a second-line vasopressor if needed in significantly hypotensive patients. Caution should be taken not to overly vasoconstrict patients with poor systolic function.

AFTERCARE

BASIC NURSING CARE

- Maintain body temperature by means of external warming devices such as warm water blankets, forced air blankets, or other devices that will raise the external temperature.
- Reduce stress and anxiety by providing a quiet, dry, comfortable environment.

OXYGEN THERAPY

- Some patients may require oxygen by facemask, nasal cannula, oxygen cage, or incubator until completely recovered from anesthesia or sedation to maximize oxygen delivery parameters.

ELECTROCARDIOGRAPHIC MONITORING

- Monitor cardiac rate and rhythm continuously until the patient is completely recovered from anesthesia or sedation. Some anesthetic drugs (ketamine and inhalation anesthetics) can predispose to cardiac arrhythmias.

CARDIOVASCULAR MONITORING

- The decision to monitor blood pressure and CVP during the postoperative period should be determined by the severity of heart disease, the patient's stability, the reason for surgical intervention, and the cardiovascular status during anesthesia and surgery. Some patients will not require further monitoring, although some patients, such as a dog with dilated cardiomyopathy undergoing surgery to correct gastric dilatation-volvulus, may require all available monitoring. Invasive postoperative monitoring may be required in some patients.

ANALGESIA

- Always provide analgesia if an invasive procedure or surgery was performed. Preemptive analgesia should be always practiced. Preemptive analgesia is defined as analgesic techniques that are applied before surgical stimulation. Incorporating analgesic agents (opioids) in the preanesthetic medication is the easiest method of preemptive analgesia. Analgesia should be performed based on pain scoring for at least 12 to 24 hours after surgery. More invasive surgeries, such as fracture repairs, will require analgesia for a longer period of time.

Opioid Analgesia Techniques

- Opioids alone or in combination with the local anesthetic lidocaine and the dissociative drug ketamine can be used to provide analgesia in the postoperative period in dogs and cats. Many combinations exist, and it is the decision of the clinician as to which CRI to administer. Decisions can be made based on severity of postoperative pain and clinician experience.

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- Morphine, hydromorphone or methadone can be used as intermittent boluses every four hours as needed based on pain scoring. Fentanyl can be administered as a constant at a rate of 2 to 12 µg/kg/h.
- In patients requiring longer-acting mild-moderate analgesia, buprenorphine can be administered every 6 to 8 hours based on pain score. Two long-acting formulations are currently licensed for cats to provide longer-term pain control. Simbadol is a concentrated formulation of buprenorphine that provides 24 hours of analgesia in cats. Zorbium is a long-acting transdermal solution labeled for cats that can provide up to 4 days of pain relief.

Opioid combinations: dog and cat differences

Dogs: Fentanyl-Lidocaine-Ketamine

- Fentanyl, lidocaine, and ketamine can be combined in a single bag or administered individually via syringe pump. The advantage of individual administration is that it allows titration of each drug as necessary. For example, a patient may require a higher dose of fentanyl that would be limited by lidocaine toxicity or dysphoria associated with ketamine. Infusion rates for fentanyl are 2 to 12 µg/kg/h. Infusion rates for ketamine are 0.25 to 0.5 mg/kg/h. Infusion rates for lidocaine are 25 to 50 µg/kg/min. If administering in a single bag, the following drugs are administered to a 1-L bag of 0.45% NaCl or 0.45% NaCl and 2.5% dextrose: lidocaine (2%; 20 mg/mL; 15 mL) and ketamine (100 mg/mL; 0.6 mL). The volume of fentanyl (50 µg/mL) will vary based on the rate of fluid administration. A volume to provide a CRI of 3 µg/kg/h should be prepared for intraoperative and postoperative administration.

Cats: Fentanyl-Ketamine

- CRIs of opioid combinations for cats are prepared and administered differently than for dogs. Cats tend to be more likely to have side effects from lidocaine. Therefore lidocaine is not used in the combinations. Cats can become excited or show signs of dysphoria from high-dose opioid combinations.

Key Point

Opioid agonists should not be avoided because of possible side effects of excitement or dysphoria in cats.

ANESTHETIC CONSIDERATIONS FOR SPECIFIC CARDIAC DISEASES AND RECOMMENDED ANESTHETIC PROTOCOLS

RECOMMENDED FOR ALL PATIENTS

- Preoxygenation with 5 L/min oxygen with a facemask or "blow by" method before induction of anesthesia is used to maximize arterial oxygenation and oxygen delivery before administration of induction drugs. ECG monitoring before induction of anesthesia is also recommended. Ventilatory support should be provided to all patients maintained with inhalation anesthesia to reduce adverse effects of hypoventilation with an anesthesia ventilator with close monitoring of the end-tidal carbon dioxide concentration in the expired air ($ETCO_2$).
- The choice of anesthetic protocol should be based on a holistic assessment of the patient, including the patient's ASA classification (see previously), the specific cardiac disease, the procedure to be performed, comorbidities, temperament, and mentation. There are several differences to be noted regarding specific cardiac diseases, anesthetic drug effects, and cardiovascular support. Therefore, a short discussion of the specific cardiac diseases is followed by the choice of anesthetic protocol based on the ASA classification. Anesthetic techniques (including injectable techniques) for medical and minor surgical procedures are also discussed, followed by techniques for major surgical procedures within each ASA classification.

COMMON CARDIAC DISEASES AND ANESTHESIA TECHNIQUES FOR DOGS

Mitral Valve Insufficiency

- Mild arterial vasodilation from anesthetic drugs can result in a decrease in the regurgitant fraction across the mitral valve and maximum cardiac output.
- Supraventricular and ventricular arrhythmias are common sequelae to mitral regurgitation, and the ECG should be monitored at all times. Extremes in heart rate (bradycardia or tachycardia) can result in decreased cardiac output.

Dilated Cardiomyopathy

- Inotropic support with dobutamine or dopamine is recommended for any major surgery regardless of ASA status if the dog is not receiving pimobendan. Dogs receiving pimobendan may not need inotropic support, although if hypotension persists, dobutamine or dopamine may be needed. Tachycardia may predispose to ventricular arrhythmias, and atropine or gylcopyrollate should be used with caution. Mild arterial vasodilation can maximize cardiac output.

Congenital defects

Aortic and pulmonic stenosis

- Cardiac output is highly dependent on heart rate, and inotropic agents contribute little to no increase in cardiac output. Tachycardia may predispose to ventricular arrhythmias, and the judicious use of atropine or glycopyrrolate is recommended. The dose of atropine or glycopyrrolate should be decreased by half. Significant bradycardia can result in severe decreases in cardiac output.

Patent ductus arteriosus and ventricular septal defect

- Pulmonary overcirculation results in a rapid uptake of inhalation anesthetic and more rapid changes in

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anesthetic depth. There may be a delay in distribution of intravenous anesthetics, although a clinical effect is likely not evident. Mild arterial vasodilation may reduce the amount of blood flow across the patent ductus arteriosus or the ventricular septal defect; however, extreme caution should be taken to prevent significant systemic hypotension as the direction of shunting can reverse, leading to severe hypoxemia.

ASA II Patients: Medical Procedure or Minor, Minimally Invasive Surgical Procedures

Injectable anesthesia technique

- The preanesthetic medication of choice would be **butorphanol alone** (0.4 mg/kg IM) or a neuroleptanalgesic combination of **acepromazine** (0.01 mg/kg IM) and **butorphanol** (0.4 mg/kg IM). Prophylactic use of atropine is not recommended to minimize production of tachyarrhythmias.
- Induction and maintenance of anesthesia can be achieved by means of **propofol** (2–6 mg/kg IV) for induction followed by either CRI (0.14–0.4 mg/kg/min) or intermittent bolus (0.5–1.0 mg/kg IV).
- Alternatively, **alfaxalone** can be used for induction (1–3 mg/kg) and maintenance either with a CRI (0.07–0.14 mg/kg/min) or intermittent bolus (0.25–0.5 mg/kg).
- Physical parameters, temperature, ECG, SpO_2, capnography, and noninvasive blood pressure can be used for monitoring during the procedure.

Inhalation anesthesia technique

- Preanesthetic medication and induction are as previously described.
- Isoflurane or sevoflurane in oxygen at the lowest effective dose can be used to maintain anesthesia, using opioids intraoperatively as needed. Ventilation should be monitored closely and mechanical ventilation available if necessary.
- Physical parameters, temperature, ECG, SpO_2, capnography, and noninvasive blood pressure can be used for monitoring during the procedure.

Key Point

Potent opioid agonists are usually not required for these types of minor procedures.

ASA II Patients: Major Surgery

- An injectable anesthesia technique to maintain anesthesia is usually not required. These patients are considered very stable before anesthesia and surgery and should be able to tolerate inhalation anesthetics as the primary technique to maintain anesthesia.
- A neuroleptanalgesic combination of **acepromazine** (0.01 mg/kg IM) or **midazolam** (0.4 mg/kg IM) and **hydromorphone** (0.1 mg/kg IM) can be used for preanesthetic medication. **Anticholinergics** should be available to treat bradycardia if noted following premedication.

Key Points

- Potent opioid agonists are preferred over opioid agonist/antagonists to provide adequate preemptive analgesia.
- Adjuncts to general anesthesia can be used to minimize the use of inhalation anesthetics. A CRI of fentanyl and lidocaine with or without ketamine should commence at the beginning of surgery. An epidural injection of a combination of bupivacaine-morphine should be administered before the beginning of surgery for procedures caudal to the umbilicus. Other local anesthetic techniques can be dictated by the surgery site. An intermittent bolus of opioid agonists may also be necessary during surgery. Ventilation should be provided if hypoventilation or hypoxemia occurs during anesthesia and surgery.

ASA III or IV Patients: Medical Procedure or Minor, Minimally Invasive Surgical Procedures

- A neuroleptanalgesic combination of **midazolam** (0.4 mg/kg IM) and **butorphanol** (0.4 mg/kg IM) is administered as the preanesthetic medication. Atropine is not recommended to minimize production of tachyarrhythmias.
- Induction can be achieved with **alfaxalone** (1–3 mg/kg IV) followed by either a CRI (0.07–0.14 mg/kg/min) or intermittent bolus (0.25–0.5 mg/kg). Alternatively induction and maintenance of anesthesia can be achieved with **propofol** (2–6 mg/kg IV) for induction followed by either CRI (0.14–0.4 mg/kg/min) or intermittent bolus (0.5–1.0 mg/kg IV). **Etomidate** (1–2 mg/kg IV) should be used as the induction agent if cardiac arrhythmias are present. Multiple administrations and CRI are not recommended because etomidate may cause lysis of red blood cells and cortisol suppression. Etomidate can also cause myoclonus, pain on injection and vomiting. Administration with a benzodiazepine reduces the incidence of myoclonus. Administration of etomidate diluted or concurrently with IV fluids may reduce the incidence of pain on injection. Endotracheal intubation should be performed to protect the airway, supply supplemental oxygen, and ventilate if necessary.
- Physical parameters, temperature, ECG, SpO_2, capnography, and noninvasive blood pressure can be used for monitoring during the procedure.

Key Point

An inhalation anesthetic technique for a medical or minor surgical procedure is not indicated for ASA III-IV cardiac patients.

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ASA III or IV Patients: Major Surgery

- An injectable anesthetic technique with low-dose inhalation, only if necessary, is recommended over a technique that relies on an inhalation anesthetic to maintain anesthesia.

Key Points

- Anesthesia of ASA III or IV patients for major surgery will require intensive monitoring of anesthesia, cardiovascular parameters, and the patient.
- Neuroleptanalgesic combination of **midazolam** (0.2 mg/kg IV) and **fentanyl** (2–5 µg/kg IV) should be administered as preanesthetic medication. Atropine (0.02 mg/kg IV) may be necessary if bradycardia occurs.
- Induction with **etomidate** (1–2 mg/kg IV) is preferred. If **etomidate** is unavailable or contraindicated, such as with sepsis, intubation can be attempted with an opioid induction consisting of **fentanyl** (5–10 µg/kg IV), **midazolam** (0.2 mg/kg IV), and **lidocaine** (1–2 mg/kg) in dogs. In cats, lidocaine is not administered. If this combination is not sufficient to allow intubation, **alfaxalone** may be titrated to effect (0.5–2 mg/kg IV).
- A CRI of fentanyl (10–30 µg/kg/h) administered by a dedicated syringe pump is recommended to maintain a surgical plane of anesthesia.
- Ventilation should be provided during anesthesia and surgery if hypoventilation or hypoxemia occurs. Lidocaine and ketamine can be administered for analgesia in separate infusions. Epidural analgesia for procedures caudal to the umbilicus is recommended. Other local anesthetic techniques are dictated by the location of the surgery.
- Blood pressure (direct is preferred over indirect measurement methods), ECG, capnometry, pulse oximetry, temperature, and physical parameters are continuously monitored.

Canine Pericardial Disease: Pericardiocentesis

ASA I or II dogs that require pericardiocentesis

- Dogs not showing signs of pericardial tamponade may only require an infiltration of local anesthetic (lidocaine) in the skin and intercostals musculature at the site of needle puncture for pericardiocentesis.
- Sedation techniques for dogs not compliant to local anesthesia only include a neuroleptanalgesia combination of **midazolam** (0.2–0.4 mg/kg IV) and **butorphanol** (0.2–0.3 mg/kg IV). Atropine should only be used if bradycardia occurs.

ASA IV dogs: emergency pericardiocentesis

- Dogs that require an emergency pericardiocentesis typically have signs of collapse, right-sided heart failure, or ventricular arrhythmias all related to pericardial tamponade. Most dogs will require only an infiltration of local anesthetic (lidocaine) in the skin and intercostals musculature at the site of needle puncture for pericardiocentesis. Occasionally dogs will require sedation in addition to local anesthesia. The neuroleptanalgesia combination of **midazolam** (0.2–0.4 mg/kg IV) and **butorphanol** (0.2 mg/kg IV) is recommended. Cardiac output is highly dependent on heart rate during pericardial tamponade, and butorphanol is least likely to decrease heart rate. Atropine is not recommended. An induction agent may be necessary in addition to sedation in some instances. **Etomidate** (1–2 mg/kg IV) is the drug of choice as there are minimal to no cardiopulmonary effects. Multiple administrations and CRI are not recommended because etomidate causes cortisol suppression.

COMMON CARDIAC DISEASES AND ANESTHESIA TECHNIQUES FOR CATS

Hypertrophic Cardiomyopathy

- The most common cardiac disease in cats is HCM, which is characterized primarily as diastolic dysfunction with normal ventricular contraction. Increases in heart rate and ventricular tachyarrhythmias caused by anesthetic drugs are best avoided.

ASA I and II cats with hypertrophic cardiomyopathy: medical procedure or minor, minimally invasive surgical procedures

- Sedation with **butorphanol** (0.3 mg/kg IM) and **alfaxalone** (0.5–2 mg/kg IM) may be sufficient for imaging or minor medical procedures. If an IV catheter is already in place, these doses can be reduced by half and administered IV to achieve heavy sedation. General anesthesia can be induced with **alfaxalone** (0.5–2 mg/kg IV) and **midazolam** (0.2 mg/kg IV). Alfaxalone administered as either a CRI (0.07–0.14 mg/kg/min) or by intermittent bolus (0.25 mg/kg IV) can be used for longer procedures.
- **Propofol** (1–3 mg/kg IV) can be administered if additional anesthesia is required. Propofol administered as either a CRI (0.14–0.4 mg/kg/min) or an intermittent bolus (0.5 mg/kg IV) can be used for longer procedures.
- Physical parameters, temperature, pulse oximetry, ECG, capnography, and noninvasive blood pressure should be monitored during the procedure.
- Isoflurane or sevoflurane in oxygen is administered at the lowest effective dose to maintain general anesthesia if required.

Key Points

- Physical parameters, temperature, ECG, noninvasive blood pressure, and capnometry can be monitored during the procedure.
- Buprenorphine is not potent enough to produce the level of sedation required.

ASA II PATIENTS: MAJOR SURGERY

- Injectable anesthesia is usually not required for maintenance because patients are considered very

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stable before anesthesia and surgery and should be able to tolerate inhalation anesthetics as the primary technique to maintain anesthesia.

- Premedication with **methadone** (0.3 mg/kg IM) and **alfaxalone** (0.5–2 mg/kg IM) will provide heavy sedation. General anesthesia can be induced with **propofol** (2–4 mg/kg) or **alfaxalone** (0.5–2 mg/kg IV) and **midazolam** (0.2 mg/kg IV).

Key Points

- Potent opioid agonists are preferred over opioid agonist/antagonists to provide adequate preemptive analgesia.
- Some cats may be able to be intubated without use of an induction agent IV. Propofol (1–2 mg/kg IV) can be used to effect to permit intubation.
- Ventilation should be always provided during anesthesia and surgery. A CRI of fentanyl (0.2–0.4 µg/kg/min) or dexmedetomidine (0.25–1 µg/kg/h) is recommended to provide analgesia and minimize use of inhalation anesthetics.
- Sacrococcygeal epidural analgesia for procedures caudal to the umbilicus is recommended.

ASA III or IV Patients: Medical Procedure or Minor, Minimally Invasive Surgical Procedures

- A neuroleptanalgesic combination of **midazolam** (0.2–0.4 mg/kg IV) and **butorphanol** (0.2–0.3 mg/kg IV) is administered as preanesthetic medication with an induction agent such as alfaxalone, propofol, or etomidate available in case paradoxical excitement occurs.
- **Aflaxalone** (0.5–2 mg/kg) or **propofol** (2–4 mg/kg IV) can be used for induction. Alfaxalone administered as either a CRI (0.07–0.14 mg/kg/min) or an intermittent bolus (0.25 mg/kg IV) can be used for longer procedures. Alternatively propofol administered as either a CRI (0.14–0.4 mg/kg/min) or an intermittent bolus (0.5–1.0 mg/kg IV) can be used if additional time is required. **Etomidate** (1–2 mg/kg IV) should be used as the induction agent if the patient is hemodynamically unstable. Multiple administrations and CRI are not recommended because etomidate causes cortisol suppression. Endotracheal intubation may be necessary and supplemental oxygen should be administered with this technique.
- Physical parameters, temperature, pulse oximetry, ECG, and noninvasive blood pressure should be monitored during the procedure.

ASA III or IV Patients: Major Surgery

- An injectable anesthetic technique with low-dose inhalation, only if necessary, is recommended over a technique that relies exclusively on an inhalation anesthetic to maintain anesthesia.

Key Points

- Anesthesia of ASA III or IV patients for major surgery will require intensive monitoring of anesthesia and the patient.
- Ideally the patient will be amenable to IV catheter placement without IM premedication. If an IM premedication is needed for an anxious or fractious patient, a combination of **methadone** (0.4 mg/kg IM) and **alfaxalone** (0.5–1 mg/kg IM) can be used to provide heavy sedation.
- If the patient has an IV catheter in place, **fentanyl** (2.5–5 µg/kg IV) and **midazolam** (0.2 mg/kg IV) can be administered as coinduction agents with **etomidate** (1–2 mg/kg IV diluted 1:1 with saline).
  - **Propofol** (2–4 mg/kg IV) or **alfaxalone** (0.5–2 mg/kg) can be used if needed.
- A CRI of fentanyl (10–20 µg/kg/h) administered by a dedicated syringe pump is recommended to reduce the amount of inhalant anesthetic necessary.
- Ventilation should be provided at all times during anesthesia and surgery.
- Locoregional anesthesia is recommended whenever possible to further reduce the amount of inhalant anesthetic needed.
- Blood pressure (direct is preferred over indirect measurement methods), ECG, capnometry, pulse oximetry, temperature, and physical parameters should be monitored during surgery.

Frequently Asked Questions

WHY SHOULD INHALANT ANESTHETICS SUCH AS ISOFLURANE AND SEVOFLURANE BE USED WITH CAUTION TO MAINTAIN ANESTHESIA IN PATIENTS WITH CARDIAC DISEASE?

Inhalant anesthetics are potent arterial vasodilators. Severe hypoperfusion and hypotension can occur during anesthesia if isoflurane or sevoflurane are used as the sole anesthetic agent to maintain anesthesia. Hypoperfusion and hypotension can be worse if the dog or cat is receiving an ACE inhibitor such as enalapril or benazepril; thus it is essential that these medications are not administered on the day of anesthesia if at all possible. The less isoflurane or sevoflurane used, the less likely it is that adverse cardiovascular effects will occur.

WHAT ARE THE MAIN DIFFERENCES IN THE CLINICAL EFFECTS OF OPIOIDS IN DOGS AND CATS?

We learned long ago in our profession that cats were not small dogs. The extreme popularity of opioids for anesthesia in dogs and the advent of advanced analgesia techniques including CRIs of opioids have led to extension of opioid use to cats. However, many clinicians have been disappointed because cats do not respond in the same way to the drugs as dogs, and opioids are possibly detrimental to cats, producing hyperexcitability, opioid-related hyperthermia, and dysphoria. These effects usually cause the clinician to discontinue the opioid. Hyperexcitability and dysphoria can be treated with acepromazine or low-dose dexmedetomidine and the hyperthermia will subside when the opioid is discontinued. The clinician must weigh management of clinical

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side effects with removing the source of adequate analgesia. Opioid use in cats is essential to provide sufficient analgesia for many procedures. Opioids are extremely safe in cats with HCM, as the myocardial contractility is not affected and the heart rate is reduced. The most effective use of opioids in cats is with the concurrent use of other sedatives. The more potent the sedative, the better the clinical effect. Cats administered alfaxalone with an opioid are better sedated than when a benzodiazepine is administered. The combination of a benzodiazepine and opioid in a cat could result in excitement. Clinicians should realize that cats will not be as heavily sedated (compared with dogs), and recoveries could be rough as well. Again, overall, the safety of opioids should outweigh these concerns.

WHAT WOULD BE THE MOST IMPORTANT CARDIOPULMONARY SIDE EFFECT OF ANESTHESIA FOR THE PATIENT WITH CARDIAC DISEASE?

The two most concerning side effects are hypotension and respiratory depression. It is essential that blood pressure be monitored when patients undergo anesthesia, and that hypotension is treated appropriately by reducing inhalant anesthetic levels when possible and utilizing vasopressors and positive inotropes when necessary. The opioids, injectable and inhalation anesthetics, are all respiratory depressants. Dogs and cats with cardiac disease undergoing anesthesia should have ventilation maintained either manually or with an anesthesia ventilator to eliminate the effects of respiratory depression. Monitoring with techniques such as capnometry and pulse oximetry will lead to early diagnosis and treatment of respiratory depression. The use of NMBAs requires the use of ventilation.

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Chapter

20

Cardiac Surgery

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Cardiac surgery is indicated for management of a variety of congenital and acquired cardiac conditions in small animals. Some cardiac surgeries are performed with curative intent, whereas others are regarded as palliative. It is important that all parties (surgical team, primary-care veterinarian, and animal owner) have a clear understanding of the expectations and risks of cardiac surgery. Increasingly image-guided cardiac interventions are available to treat cardiac conditions in animals. Often these interventions offer an alternative to classic cardiac surgery and should be part of the discussion when formulating a treatment plan. Hybrid procedures that combine image-guided catheter-based techniques with minimally invasive surgical approaches are emerging as an additional option. Treatment options should be discussed by the "heart team" consisting of the medical cardiologist, interventional cardiologist, cardiac surgeon, anesthesiologist, and criticalist. Factors to consider include relative invasiveness, operating time, experience of the operator, availability of specialized equipment, patient size, patient age, comorbidities, procedural risk, efficacy, and cost. Cardiac surgeries include closed cardiac surgeries performed on the beating heart, brief circulatory arrest cardiac surgeries performed during venous inflow occlusion, and open cardiac surgeries performed during cardiopulmonary bypass.

PATENT DUCTUS ARTERIOSUS

Based on poor long-term prognosis, PDA closure is indicated in dogs and cats with left-to-right PDA, preferably early in life. PDA closure in younger animals is performed with curative intent. Older animals should undergo surgery as soon as possible after the diagnosis. Animals with secondary myocardial failure and functional mitral regurgitation may still benefit from PDA closure. Animals with pulmonary hypertension can undergo PDA closure so long as pulmonary artery pressures have not reached systemic levels. Closure of a fully right-to-left PDA is contraindicated. Treatment with a phosphodiesterase 5 inhibitor such as sildenafil can lower pulmonary artery pressures sufficiently to reestablish left-to-right shunt in some cases, especially cats.

Transcatheter ductal occlusion is now an established option for PDA closure in dogs. Given its decreased invasiveness and lower risk, transcatheter ductal occlusion is generally preferred over surgical PDA ligation in most dogs.[1,2] Limitations of transcatheter ductal occlusion include small patient size and nontapering ductal anatomy that does not allow anchoring of an occluder. The cost of the device or lack of imaging equipment can sometimes also be limiting. Dogs that are not candidates for transcatheter ductal occlusion for whatever reason become candidates for surgical ligation. Surgical ligation is the preferred option for PDA closure in cats in many centers.

Surgical PDA ligation is accomplished through a left fourth thoracotomy in dogs and a left fourth or fifth thoracotomy in cats. A lateral thoracic radiograph can be reviewed to determine the appropriate intercostal space. The lungs are rotated caudally to expose the base of the heart. The vagus nerve courses over the ductus arteriosus and serves as an anatomic landmark for identification of the ductus arteriosus. The vagus nerve is isolated at the level of the ductus and gently retracted dorsally or ventrally with one or two sutures. Rarely a persistent left cranial vena cava may overlie the ductus arteriosus. In this case, the vein should be isolated and retracted with the vagus nerve. The ductus arteriosus is isolated for ligation outside of the pericardium. Right-angled forceps are first passed transversely behind the ductus to establish a dissection plane. A dissection plane between the aorta and cranial ductus can also be established by angling the forceps 45 degrees caudally. The forceps are passed medial to the ductus in a caudal to cranial direction. Blunt dissection medial to the ductus by opening and closing the forceps should be strictly avoided. Rather, the forceps should be allowed to find their way around the ductus by careful manipulation of the forceps considering the three-dimensional shape of the ductus. Once the tip of the forceps can be visualized through the adventitial tissues between the aorta and cranial ductus, passage of the forceps can be safely completed. Heavy silk ligatures are grasped and passed around the ductus. The ductus arteriosus is closed by slowly tightening and tying the silk ligatures.

Surgical mortality rates of 0% to 7% have been reported in retrospective studies involving 50 or

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more dogs.[1,3–6] PDA ligation is curative in the majority of cases when it is performed at an early age (<6 months of age).[6,7] Resolution of secondary functional mitral regurgitation is possible but may not be entirely reversible in older animals and large-breed dogs.[8]

VENTRICULAR SEPTAL DEFECT

Ventricular septal defect (VSD) is a congenital defect resulting from incomplete development of the membranous or muscular ventricular septum. Perimembranous defects are most common in companion animals and are bordered by the central fibrous body and the inlet, trabecular, or infundibular (outlet) portion of the muscular septum. These defects are medial to the septal leaflet of the tricuspid valve, inferior to the crista supraventricularis, and closely associated with the conduction tissues of the heart. Inlet perimembranous VSD is associated with the spectrum of defects associated with atrioventricular septal defect. Muscular defects are less common, bordered entirely by muscle, and occur within the inlet, trabecular, or infundibular muscular septum. Doubly committed subarterial VSD occurs high in the infundibular septum, where the conjoined leaflets of the pulmonary and aortic valve form part of the rim of the defect.

Surgical intervention for VSD is indicated for high-flow shunts to prevent progressive heart failure or pulmonary hypertension. Aortic insufficiency is also an indication to consider surgery. Several parameters suggest that a VSD is hemodynamically significant, including radiographic evidence of pulmonary vascular enlargement, radiographic and echocardiographic evidence of left ventricular chamber dilation, and $Q_P/Q_S > 1.5$.

PULMONARY ARTERY BANDING

Pulmonary artery banding is an effective palliative option for VSD. The goal of pulmonary artery banding is to increase right ventricular systolic pressure and thereby decrease the pressure gradient driving the shunt flow. A method for placement of a dilatable pulmonary artery band has been described in cats.[9] A dilatable pulmonary band allows the option of transcatheter balloon dilation if the band becomes too tight as immature animals grow. Pulmonary artery banding is accomplished through a left fourth thoracotomy. The pericardium is opened and sutured to the thoracotomy incision. The pulmonary artery is separated from the aorta by a combination of sharp and blunt dissection. A polytetrafluoroethylene (PTFE) tape is passed around the pulmonary artery dorsal to the pulmonary valve. A pursestring suture is placed in the pulmonary artery downstream from the band, and a catheter is introduced for measurement of pulmonary arterial pressures during tightening of the band. The tape is progressively tightened by sequential placement of hemostatic clips on the band. Optimal banding is judged when pulmonary systolic arterial pressure distal to the band is decreased ideally to <30 mm Hg and the increase in systemic arterial pressures just begin to plateau. Intraoperative epicardial echocardiography evaluation of shunt flow can also be used to guide band tightening. After final tightening of the band, a 6-0 mattress suture is placed in the tape just behind the last clamp. The hemostatic clips should not protrude beyond the width of the tape to avoid late puncture of the pulmonary artery.

OPEN REPAIR FOR VENTRICULAR SEPTAL DEFECT

Open repair of VSD in dogs is performed with curative intent during cardiopulmonary bypass. Perimembranous defects are accessed through a right fifth thoracotomy and right atriotomy. Arterial cannulation for cardiopulmonary bypass is in the left femoral or carotid artery. Bicaval venous cannulation is employed to allow isolation of the right atrium. The aorta is cross-clamped and antegrade cardioplegia administered. The VSD is accessed through a right atriotomy. The septal leaflet of the tricuspid valve is retracted to expose the defect. Pledget-reinforced mattress sutures are placed around the circumference of the septal defect. The defect is closed with PTFE or Dacron cardiovascular graft. The major conduction bundles are located close to the caudal margin of the defect and are susceptible to injury during suture placement. Sutures in this portion of the defect should be placed partial thickness away from the caudal margin of the defect. Perimembranous defects typically are bordered by tricuspid valve annulus and do not have a dorsal margin. In this case, graft closure is completed by passing sutures through the tricuspid annulus into the atrial side of the valve. The atriotomy is closed with a continuous mattress suture pattern oversewn with a simple continuous pattern. Infundibular and doubly committed subarterial defects can be accessed through the outflow portion of the right ventricular wall via a median sternotomy. Cannulation for cardiopulmonary bypass and closure is similar to perimembranous defects. Closure of ventriculotomy is by pledget-reinforced mattress sutures.

Long-term palliation of VSD after pulmonary artery banding has been reported in dogs and cats for up to 7 years after surgery.[9,10] The most serious early complication is overtightening of the band, leading to reversal of shunt flow and hypoxemia. This complication can be largely avoided by measurement of systemic and pulmonary arterial pressures and shunt flow during the procedure. Progressive obstruction and late reversal of shunt flow can result from growth of the animal, progressive fibrosis of the pulmonary artery band, progressive right ventricular hypertrophy, or any combination of these. Definitive closure of VSD under cardiopulmonary bypass is considered curative in humans so long as it is undertaken prior to the

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development of severe pulmonary hypertension or secondary myocardial failure.[11] Although reports in dogs are limited,[12] a similar expectation after definitive closure of VSD in dogs is reasonable.

ATRIAL AND ATRIOVENTRICULAR SEPTAL DEFECT

Atrial septal defects (ASDs) are classified as sinus venosus defects, coronary sinus ASD, ostium secundum (fossa ovalis type) ASD, or patent foramen ovale. Sinus venosus defects are located dorsal in the atrial septum at the junction of the cranial or caudal vena cava and are often associated with anomalous pulmonary venous return. Coronary sinus ASD or unroofed coronary sinus results from incomplete separation between the coronary sinus and the left atrium due to the persistence of a left cranial vena cava. Ostium secundum ASD (fossa ovalis type) occurs in the middorsal portion of the atrial septum. Patent foramen ovale is usually a secondary defect caused by increased right atrial pressure associated with concurrent right heart defects.

Atrioventricular septal defect (AVSD) represents a spectrum of malformations involving the septum primum, the inlet portion of ventricular septum, and the atrioventricular (AV) valves. AVSDs are classified as having communication at the atrial level, ventricular level, or both with a common AV valve. AVSD with communication at the atrial level consists of an ostium primum defect and typically a trifoliate left AV valve with bridging leaflets directed towards the inlet portion of the ventricular septum.[13] The latter, previously known as a mitral cleft, can result in significant left AV valve regurgitation. Complete AVSD with atrioventricular communication, also known as an endocardial cushion defect, consists of an ostium primum defect, inflow ventricular septal defect, and a single AV valve with bridging leaflets that are common to the right and left ventricle.

Surgery to correct an ASD is based on clinical signs and the hemodynamic significance of the defect. The degree of cardiac chamber enlargement, pulmonary overcirculation on radiographs, pulmonary flow velocity, and Q_P/Q_S can be considered in evaluating hemodynamic significance,.

OPEN REPAIR FOR ATRIAL SEPTAL DEFECT AND ATRIOVENTRICULAR SEPTAL DEFECT

Open repair of ASD in dogs is accomplished with the aid of cardiopulmonary bypass through a right fifth thoracotomy. Cannulation for cardiopulmonary bypass is as described for VSD. The defect is approached through a right atriotomy. The septal defect is closed with autogenous pericardium harvested before the pericardium or PTFE cardiovascular patch. Sinus venosus defects may require concurrent correction of anomalous pulmonary venous return. This is accomplished by extending the patch closure to the right of the ostia of the anomalous veins, functionally leaving their flow on the leftward side of the patch.

AVSD repair generally requires closure of the trifoliate left AV valve prior to closing the septal defect. This is accomplished by suturing the bridging leaflets together before closing the septal defect and performing an annuloplasty if necessary (see surgical mitral valve repair). The ostium primum defect is closed with autogenous pericardium or PTFE patch. To avoid injury to the atrioventricular conduction system, the patch is extended around the coronary sinus, leaving coronary flow to the left side of the patch. The atriotomy is closed with a continuous horizontal mattress pattern and oversewn with a simple continuous pattern.

Successful surgical correction has been described for ASD and AVSD in dogs.[13–17] Open repair of ASD or AVSD can be considered curative, assuming that the magnitude of pulmonary hypertension or secondary myocardial failure is not severe at the time of surgery. For partial AVSD, the long-term outcome also depends on the magnitude of residual AV valve regurgitation.[15] As with VSD, complete AV block is a possible operative complication of open repair.

TETRALOGY OF FALLOT

Tetralogy of Fallot is a congenital heart defect that consists of pulmonary stenosis, an infundibular perimembranous VSD, a dextropostioned overriding aorta, and right ventricular hypertrophy. The long-term prognosis for tetralogy of Fallot depends on the ratio of pulmonary to systemic blood flow. Animals with resting hypoxemia and progressive polycythemia will likely succumb to the effects of the defect early in life. Surgery can be considered for animals with exertional cyanosis to lessen clinical signs and prolong life. Debilitating exercise intolerance, erythrocytosis (hematocrit > 60%), and resting hypoxemia (arterial oxygen saturation < 60%) are indications for surgery.

Palliative interventions for tetralogy of Fallot include isolated correction of the pulmonary stenosis or creation of a systemic-to-pulmonary shunt.[18,19] The goal of both palliative approaches is to increase pulmonary blood flow without creating an overwhelming left-to-right shunt. Systemic-to-pulmonary shunts increase pulmonary blood flow to a more predictable degree, and thereby improve arterial oxygen saturation while lessening the risk of excessive pulmonary overcirculation. Several systemic-to-pulmonic shunts have been devised for palliation of tetralogy of Fallot. A modified Blalock-Taussig consisting of a synthetic graft between the left subclavian artery and pulmonary artery has resulted in successful long-term palliation in dogs.[20]

MODIFIED BLALOCK-TAUSSIG SHUNT

Modified Blalock-Taussig shunt is a palliative procedure for tetralogy of Fallot in dogs >5 kg.[20] The

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magnitude of pulmonary flow is determined by the diameter of the subclavian artery, which is known to be appropriate. Modification of the classic procedure consists of placement of a synthetic vascular graft between the left subclavian and pulmonary arteries. The surgery is performed in dogs through a left fourth thoracotomy. The left subclavian artery is isolated and clamped. An end-to-side anastomosis is performed between the subclavian artery and a 5- to 6-mm ePTFE graft. A portion of the pulmonary trunk is isolated with a side-biting vascular clamp. The surgery is completed by a second anastomosis between the graft and pulmonary trunk. Clopidogrel is administered for at least 3 months after surgery. The graft should be closely monitored for thrombosis. If graft flow becomes diminished, anticoagulation therapy should be added.

OPEN REPAIR OF TETRALOGY OF FALLOT

Definitive repair of tetralogy of Fallot can be undertaken in dogs with curative intent under cardiopulmonary bypass.[21] The surgical approach in dogs is median sternotomy. Cannulation for cardiopulmonary bypass is as described for other septal defects. The aorta is cross-clamped and antegrade cardioplegia administered. The cardiac approach is via a longitudinal ventriculotomy in the right ventricular outflow tract. Patch closure of the VSD is performed, taking care to avoid injury to the tricuspid valve and conduction tissues. Infundibular pulmonary stenosis is corrected by excision of the subpulmonary muscular bands and imposition of an oval patch-graft across the stenosis. In most dogs, the obstruction will consist entirely of a subpulmonary fibromuscular band. In this case, the patch-graft is placed within the ventriculotomy without extending the incision across the pulmonary valve annulus. If valvular pulmonary stenosis is present, a transannular pulmonary patch-graft may be necessary. In all cases, preoperative planning must exclude the possibility of a prepulmonary coronary artery anomaly.

Long-term palliation of tetralogy of Fallot by modified Blalock-Taussig shunt has been reported in dogs.[20] The challenge is maintaining patency of the shunt through close monitoring and proactive antithrombotic therapies. Surgical correction for tetralogy of Fallot with long-term survival has also been reported in dogs.[21] In the author's experience, surgical correction of tetralogy of Fallot is associated with resolution of clinical signs and restoration of life expectancy after surgery.

MITRAL REGURGITATION

Mitral regurgitation (MR) is the most frequent cause of cardiovascular disability and death in dogs. Mitral regurgitation is classified etiologically as primary (organic) caused by a primary leaflet abnormality or functional (secondary) associated with cardiac remodeling. Examples of primary MR include degenerative disease, endocarditis, and congenital mitral dysplasia. Functional MR results from remodeling of the left atrium plus or minus left ventricle. Functional MR associated with ventricular dilation results from the combined effects of displacement of the papillary apparatus and dilation of the mitral annulus. Atrial dilation, such as occurring with lone atrial fibrillation, can be associated with functional MR, sometimes referred to as atrial functional MR. Carpentier classification of MR classifies MR as due to annular dilation (type I), excessive leaflet motion (type II), and restrictive leaflet motion (type IIIa and IIIb). Correction of MR takes into consideration both its etiologic and functional classification.

Degenerative MR is characterized by varying degrees of nodular thickening valve leaflets, elongation/rupture of the chordae tendineae, and dilation of the valve annulus. Mitral regurgitation results from a combination of primary leaflet prolapse/flail (type II defect) and annular dilation secondary to cardiac remodeling (type I defect). As such, degenerative MR actually represents "mixed MR" caused by primary leaflet abnormality worsened by secondary functional MR due to cardiac remodeling. In dogs with degenerative MR, leaflet prolapse/flail invariably involves the A2 segment of the mitral valve plus or minus involvement of other segments of the anterior and posterior leaflet. These understandings form the basis of interventions for degenerative MR. Congenital mitral valve dysplasia is most often associated with a combination of restrictive leaflet motion (type III defect) and secondary annular dilation (type I defect). Leaflet motion is restricted in both systole and diastole due to tethering of portions of the mitral leaflets by abnormal chordal attachments (type IIIa defect). It is important to distinguish congenital mitral dysplasia from secondary functional ventricular MR associated with dilated cardiomyopathy. The latter is caused by restrictive leaflet motion during systole due to the displacement of papillary muscles (type IIIb defect).

Dogs undergoing consideration for intervention should meet the criteria for severe MR. This could include dogs in American College of Veterinary Internal Medicine (ACVIM) stage B2 or stage C. Dogs in late-stage C/D or atrial fibrillation are considered poor candidates for mitral intervention. Criteria that support severe MR include:

- Holosystolic wall-impinging eccentric color-flow jet or
- Central color-flow jet area ≥50%
- E-wave dominant mitral inflow ≥1.0 m/s and/or E:A ratio >2
- Dense holosystolic triangular regurgitant profile on CW Doppler
- Regurgitant fraction ≥ 50% (PISA, volumetric, Doppler)
- Regurgitant volume ≥ 1.0 mL/kg

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SURGICAL MITRAL VALVE REPAIR

Surgical mitral valve repair in dogs is performed with the aid of cardiopulmonary bypass. The surgical approach is a left atriotomy via a left fifth thoracotomy. Arterial cannulation is either via the carotid or femoral artery. Venous cannulation is either via a jugular vein or direct right atrial via the right auricle, or both. A cannula is placed in the aorta for administration of cardioplegia solution. The repair is performed with the aorta cross-clamped under full cardioplegic cardiac arrest.

Several repair techniques have been described to correct leaflet prolapse/flail. Placement of artificial chordae has been most commonly employed in dogs.[22,23] Artificial chordae consist of 5-0 or 4-0 ePTFE monofilament suture. Pledge-reinforced ePTFE sutures are placed in the tip papillary muscle and then brought through the leaflet margin. Correction of prolapse/flail is focused primarily on the anterior (septal) leaflet. The most important aspect of the technique is judging the length of the artificial chordae. A general guide is that the edge of the valve leaflet should just reach the plane of the opposing annulus after tying down the annuloplasty ring.

Edge-to-edge (E-to-E) repair was first described as a surgical technique to address anterior leaflet prolapse.[24] E-to-E repair has been used in dogs.[23] It is accomplished by suturing the free edge of the prolapsing leaflet to the free edge of the facing leaflet at the point of greatest prolapse, resulting in a double orifice valve. The E-to-E technique can be combined with a more conservative annuloplasty to avoid creating mitral stenosis. The E-to-E technique can also be used to correct regurgitation in the commissural regions, which can be difficult to repair with other techniques.

Annuloplasty addresses secondary annular dilation that is an inevitable component of severe MR. Dilation of the annulus occurs over the mural and commissural regions of the annulus. The septal region of the annulus is fixed by the fibrous trigone of the heart and does not dilate. Thus it is the mural and commissural regions of the annulus that must be reduced by annuloplasty. Several annuloplasty techniques have been employed. The simplest involves plication of the annulus with running sutures in the commissural and mural portions of the annulus. In humans, ring or band annuloplasty has been shown to be more durable and is therefore preferred. A variety of rings/bands have been developed, including rigid, flexible, saddle-shaped, complete, and partial rings. Commercial annuloplasty rings can be used in larger dogs, but for small dogs, a partial "band" can be fashioned from cardiovascular graft materials such as ePTFE.[22,23] The size (length) of the ring is based on the area of the septal leaflet, with the goal of reducing the area of the annulus to the approximate area of the septal leaflet. Band annuloplasty is achieved by placing sutures into the valve annulus 1 to 2 mm back from the leaflet insertion. If a complete ring is used, sutures are placed across the base of the septal leaflet, taking care to avoid the aortic valve that lies in close proximity. If a partial band technique is used, then anchoring sutures are placed just beyond the commissural regions of the annulus. Sutures are passed through the ring/band. The ring/band is seated into a position and the sutures are tied. Annuloplasty reestablishes appropriate coaptation of the valve leaflets.

Reported operative mortality for surgical mitral valve repair is about 10% in the most experienced centers.[23,25,26] Surgery-associated morbidity is fairly high, with reported complication rates as high as 30%.[25,26] Reported complications include acute renal insufficiency, pulmonary and neurologic complications, diaphragmatic paralysis, cardiac hematoma, thrombosis, and bleeding.[23,25,26] Median hospitalization was 6 days (4–14 days) in one experienced center.[25] Short-term outcomes in dogs undergoing surgical mitral repair demonstrate feasibility;[23,25,26] however, outcome data on long-term efficacy and durability of repair and survival in dogs undergoing surgical mitral repair is limited.[22,23,25–28]

Surgical repair of mitral valve dysplasia is challenging and varies depending on the spectrum of morphologies present. Aberrant chordae tendineae that are restricting leaflet closure are divided. If division of an aberrant chorda results in leaflet prolapse, this is addressed by placement of an artificial chordae. Fusion and shortening of the chordae tendineae can be addressed by fenestration of the fused chordae and splitting of the papillary muscle to effectively lengthen the chordal apparatus. If this fails to adequately mobilize the valve apparatus, then the papillary muscle can be divided and lengthened with a large ePTFE mattress suture as described for correction of tricuspid valve dysplasia. Commissurotomy may be necessary if valvular mitral stenosis is present. Lastly a ring annuloplasty will usually be necessary to correct secondary annular dilation.

TRANSCATHETER EDGE-TO-EDGE MITRAL VALVE REPAIR

Transcatheter edge-to-edge mitral valve repair (TEER) is a minimally invasive beating-heart intervention for treatment of severe degenerative MR. The procedure is based on the original surgical edge-to-edge technique. In humans, TEER has become a successful alternative to surgical mitral repair in patients who are not good candidates for surgery. TEER in dogs is performed under general anesthesia through a minithoracotomy (3–4 cm) and transapical cardiac approach in the beating heart under fluoroscopic and transesophageal echocardiography (TEE) guidance. The device is the Canine Mitral V-Clamp (Hongyu Medical, Shanghai).

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Table 20.1 Guideline for Transcatheter Edge-to-Edge Mitral Valve Repair (TEER) Based on Anatomic Complexity

CENTER EXPERIENCE			
EXPECTATION FOR MR REDUCTION			
NONCOMPLEX IDEAL FOR TEER	**COMPLEX SUITABLE FOR TEER**	**VERY COMPLEX CHALLENGING TEER**	**TEER DIFFICULT OR IMPOSSIBLE**
BENEFIT ≥≥≥ RISK	**BENEFIT ≥≥ RISK**	**BENEFIT ≥ RISK**	**RISK > BENEFIT**
• Focal A2 leaflet prolapse • Posterior wall-hugging regurgitant jet • Coaptation gap ≤2 mm • Commissural VC width <33% of commissural mitral diameter • Preserved coaptation reserve (depth)	• A2 and P2 prolapse • Two-segment anterior leaflet prolapse • Eccentric posterior to central regurgitant jet • Coaptation gap 2–4 mm • Commissural VC width 33%–66% commissural mitral diameter • Coaptation reserve (depth) <3 mm	• A2 leaflet flail • Three-segment anterior leaflet prolapse • Two-segment prolapse of posterior leaflet prolapse • Central regurgitant jet • Vertical coaptation gap >4 mm, or horizontal coaptation gap = 2 mm • Commissural VC width >6% commissural mitral diameter • Limited coaptation reserve with wide coaptation gap • Systolic A-P diameter >20 mm • Body weight <4 kg	• P2 leaflet flail • Severe multisegment leaflet flail • Multiple disparate and wide regurgitant jets • Deep leaflet cleft(s) or perforation • Leaflet thickness >5 mm • Vertical coaptation gap >6 mm, or horizontal coaptation gap >2 mm • Posterior leaflet length <4 mm • Systolic A-P mitral diameter >22 mm • Body weight <2.5 kg

MR, Mitral regurgitation; *VC*, vena contracta.

Functional pathoanatomy of the mitral valve is an important predictor of procedural success and outcome in dogs undergoing TEER intervention. Based on currently available canine device sizes (14, 16, 18, 20 mm), midsystolic A-P mitral annular diameter should be between 14 and 22 mm on apical five-chamber echocardiographic views. Anatomic guidelines are not absolute but represent a continuum from favorable to less favorable to unfavorable (Table 20.1). Dogs with the most favorable functional anatomy can be expected to have the best outcomes in terms of reduction in MR severity and extension of life expectancy with low risk. Dogs with less favorable functional anatomy may derive benefit from TEER with less reduction in MR severity and some associated increase in procedural risk. Dogs with unfavorable functional anatomy carry a higher procedural risk compared to potential benefit and are considered poor candidates for the TEER procedure. Final assessment of functional anatomy for edge-to-edge repair should be based on preprocedural TEE.

TEER is performed under general anesthesia via a minithoracotomy and transapical cardiac approach with dogs in right lateral recumbency. The optimal intercostal space is typically the seventh intercostal space confirmed on a lateral fluoroscopic view of the cardiac silhouette and transthoracic echocardiography. A 3-cm intercostal thoracotomy is made just dorsal to the sternum. The pericardium is opened and sutured to the incision to elevate the cardiac apex. The cardiac puncture site is determined by external compression of the heart during biplane TEE of the left ventricle and mitral valve. The optimal puncture site is perpendicular to the plane of the mitral valve on both the inflow-outflow and commissural views. Two pledget-reinforced mattress sutures of 4-0 polypropylene are placed at the optimal puncture site and passed through tourniquets.

The procedure was performed under fluoroscopic and TEE visualization. The typical fluoroscopic view is 10 degrees cranial and 0 to 10 degrees left anterior oblique on the fluoroscope (90–100 degrees on the patient). Simultaneous TEE views are biplane commissural and inflow-outflow views, and three-dimensional en face view of the mitral valve. The TEE and fluoroscopic views are oriented with the aorta at 9 o'clock to provide synchronous cranial-caudal movement of the device between the TEE and fluoroscopic views.

An intravenous bolus of heparin (50 U/kg) is administered prior to entering the heart. Four V-Clamp sizes (14, 16, 18, 20 mm) were available. V-Clamp sizing is based on the midsystolic anterior-posterior (A-P) mitral valve dimension on TEE inflow-outflow view minus 0 to 2 mm. Cardiac puncture is performed with an 18-gauge needle catheter. A 0.035-inch 50-cm J-tip wire is passed into the left ventricle. The J-tip wire and dilator are exchanged for a specialized soft Nitinol basket tip device for crossing the mitral valve. The device is passed retrograde across the mitral valve and the introducer catheter is passed over the device into the left atrium. The V-Clamp delivery apparatus is passed into the left atrium via the introducer catheter. The arms of the V-Clamp are manipulated to obtain optimal medial-lateral, A-P, and rotational orientations on TEE and fluoroscopic views prior to crossing the mitral valve. The lower arms of the clamp are brought across the mitral valve, maintaining appropriate

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orientation. The lower arms of the clamp are brought up under the mitral valve to suspend the leaflets. The valve leaflets are captured by bringing the upper arms down to the valve, thereby closing the clamp. Once initial leaflet capture is confirmed based on TEE and fluoroscopy, the clamp is locked. The unclamped portion of the anterior and posterior leaflets are measured and subtracted from the preclamp lengths to assure adequate leaflet capture (ideally ≥4 mm each leaflet). The clamp is then released. Deployment of a second clamp, if needed, follows the same basic procedure with deployment as close as possible to the first clamp. An early feasibility study of TEER in dogs demonstrated promising results with low procedural risk, rapid recovery, and meaningful (50% or greater) reductions in MR severity.[29]

TRICUSPID VALVE DYSPLASIA

Tricuspid valve dysplasia (TVD) is a congenital malformation of the tricuspid valve. A spectrum of anatomic defects has been associated with tricuspid dysplasia. In dogs, the most common form consists of direct attachment of large papillary muscles to the mural leaflet and an immobile septal leaflet that is "tethered" to the ventricular septum by multiple short, fine chordae. Restrictive leaflet motion (Type IIIa) combined with severe annular dilation result in "torrential" tricuspid regurgitation (TR) in the most advanced cases. Most dogs with massive or torrential tricuspid regurgitation develop right-sided congestive heart failure early in life. Atrial fibrillation is a frequent late sequela that contributes to the progression of heart failure.

Indications for surgery include massive (grade 4/5) or torrential (grade 5/5) tricuspid regurgitation, severe right atrial and ventricular dilation, and/or evidence of hepatic venous congestion. Surgical intervention should be undertaken before congestive heart failure becomes established. Once right-sided congestive heart failure becomes medically refractory or atrial fibrillation becomes long-standing, dogs become poor candidates for surgery.

TRICUSPID VALVE REPAIR

Tricuspid valve repair is a possible option for dogs with the typical tricuspid valve dysplasia. Tricuspid valve repair is performed via a right-sided thoracotomy through the fifth intercostal space. Cannulation for cardiopulmonary bypass is femoral arterial and bicaval venous. The surgery is performed under antegrade cardioplegia with the aorta cross-clamped. The tricuspid valve accessed is a right atriotomy. Surgical repair is directed at mobilization of the mural and septal leaflets and annuloplasty to restore normal annular area. Mobilization of leaflets is accomplished by division of abnormal leaflet attachments and resuspension with artificial chordae.[30]

TRICUSPID VALVE REPLACEMENT

Tricuspid valve replacement with a bioprosthetic valve can be considered for tricuspid valve dysplasia outside of the typical spectrum of defects that are not amendable to repair or when tricuspid stenosis is present. The surgical approach and cannulation for cardiopulmonary bypass are as described for tricuspid valve repair.[31] Between 12 and 15 double-armed 3-0 or 2-0 buttressed mattress sutures are preplaced in the valve annulus. The leaflets are excised or imbricated within the mattress sutures to prevent them from interfering with the motion of the valve prosthesis. Dilation of the tricuspid annulus is corrected by taking wide bites with each mattress suture. Mattress sutures are passed through the sewing ring of the valve prosthesis. The valve is seated into place and the sutures are tied. The atriotomy is closed with a continuous horizontal mattress pattern and oversewn with a simple continuous pattern. Dogs undergoing valve replacement with a bioprosthesis should be placed on antithrombotic therapy for 3 months. The current recommended protocol combines oral apixaban and clopidogrel.[32]

The author has performed tricuspid valve repair on more than 25 dogs. Tricuspid valve repair for tricuspid dysplasia generally does not abolish tricuspid regurgitation but rather decreases torrential/massive TR to levels that can be tolerated for several years. Seven-year survival after tricuspid valve repair >75%. Reverse remodeling may further improve TR. Supraventricular tachyarrhythmias are sometimes observed as a late sequela. These are usually manageable with appropriate medical therapy and/or cardioversion. Tricuspid valve replacement abolishes tricuspid regurgitation and can be curative provided the valve prosthesis remains functional.[31] Prosthesis-associated complications include thrombosis or late inflammatory pannus and can occur in up to 50% of cases. Dogs undergoing bioprosthetic tricuspid valve replacement have remained free from clinical signs for periods of 7 years or longer.

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Chapter

21

Pacemaker Therapy

Janice M. Bright and Brianna M. Potter

"It struck me how easily excitable the myocardium is. You just touch it and it gives you a run of extra beats—so why should the heart that is so sensitive…die because there's nothing there to stimulate the chest?"

Paul M. Zoll, inventor of the first clinically successful external pacemaker. Jeffrey K: Cardiac pacing and electrophysiology at millennium's end: historical notes and observations. PACE 22;1713, 1999.

Pacemaker therapy has become a common method of treating symptomatic bradycardia in dogs and cats. Pharmacologic therapy may provide temporary chronotropic support for patients with bradycardia, but successful long-term management usually requires implantation of a permanent pacemaker. The ultimate objective of cardiac pacing is to normalize cardiac function by providing optimal heart rate and rhythm chronically.

INDICATIONS FOR PACING

- Permanent pacemaker implantation is indicated for treatment of patients with chronic, symptomatic bradyarrhythmias. In dogs, the most frequent clinical manifestations of bradycardia are syncope, collapse, exercise intolerance, or lethargy. Less commonly, bradyarrhythmia results in congestive heart failure. In cats, the most frequent complaint associated with bradycardia is syncope or related to congestive heart failure.
- The most common bradyarrhythmia necessitating pacemaker therapy in dogs and cats is advanced (high-grade second- or third-degree) atrioventricular (AV) block. Antibradycardia pacing is also indicated in dogs with sick sinus syndrome and permanent atrial standstill. Less often, permanent pacing is used to prevent vasovagal syncope.
- Most dogs needing pacemaker therapy have little to no underlying structural heart disease; however, older dogs may have some degree of degenerative mitral valve disease. In cats, AV block is frequently associated with underlying myocardial disease.

PREIMPLANTATION EVALUATION

- A standard electrocardiogram (ECG) should be obtained for definitive diagnosis of the arrhythmia. If it is unclear whether a patient's clinical signs are due to bradyarrhythmia, correlation of the clinical signs with the arrhythmia should be obtained with some form of ambulatory electrocardiographic monitoring (Holter or event monitoring). Ambulatory ECG monitoring allows the ECG to be recorded over a longer period of time, either continuously for 24 to 48 hours (Holter monitoring), continuously for up to 2 weeks (event monitor), or intermittently during weeks to months (event recording). Ambulatory ECG may also be needed to confirm intermittent bradycardia.
- After confirmation that an indication for permanent cardiac pacing exists, the most appropriate pacing system and pacing mode for the patient should be determined. Factors to be considered for this determination include specific underlying rhythm disturbance, overall physical condition, presence of associated medical problems including structural heart disease, and exercise requirements of the patient.
- Therefore all patients needing implantation of a permanent pacemaker should receive a thorough medical and cardiovascular evaluation before implantation to identify presence and severity of structural heart diseases or noncardiac diseases. Certain coexisting conditions may require additional medical treatment or may predispose the patients to complications from pacemaker implantation (such as thrombus formation). Furthermore, these conditions often affect the type and programming of the pacing system used.

COMPONENTS AND TYPES OF PACING SYSTEMS

- A permanent artificial cardiac pacing system consists of a pulse generator and one or more pacing leads. The pulse generator contains electronic circuitry and a lithium-iodide power cell (battery) sealed within a metal case with a connector block

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into which the lead(s) is (are) inserted. A pacing lead consists of an insulated wire or set of wires to conduct electrical impulses from the generator to the myocardium, but the lead also enables the generator to detect or sense native (endogenous) cardiac electrical activity.

- Modern cardiac pacemakers have sophisticated electronic circuitry capable of discharging pacing impulses of varying duration and voltage, sensing intracardiac signals, filtering signals, providing rate response functions, and storing rhythm data. Data retrieval and generator programming are done by means of telemetry with a pacemaker programmer. The programmer is also used to display real-time ECG and intracardiac electrograms and to test battery life, lead impedance, retrograde ventriculoatrial conduction, and pacing thresholds.
- The heart can be paced by epicardial lead(s) placed surgically or by permanent endocardial pacing lead(s) placed transvenously. Permanent transvenous pacing has largely replaced epicardial pacing because of its ease and safety. However, epicardial leads remain available and may be preferred when venous access is limited or when there is an associated condition that would increase likelihood of bacteremia, thrombosis, or embolism with a permanent transvenous lead (e.g., a focus of sepsis or a hypercoagulable state). Epicardial leads are often used in feline patients.
- Permanent transvenous pacing leads use either active or passive fixation for attachment of the lead tip to the endocardium. A passive fixation transvenous lead has a "collar" of tines encircling the distal tip, which anchor the lead by becoming enmeshed in the trabeculae of the right ventricle or right atrium (Fig. 21.1). An active-fixation lead has a small metal helix that exits the tip of the lead to penetrate the endocardium (see Fig. 21.1). Although the type of fixation of a transvenous lead does not appear to affect the incidence of lead dislodgement, it is wise to avoid use of passive fixation in animals with significant right ventricular (RV) dilation.
- Another consideration for selection of the pacing system is whether a unipolar or bipolar system is desired. A unipolar pacing system uses the lead tip as the cathode (negative pole) of the electrical circuit and the metal case of the pulse generator as the anode (positive pole). The impulse travels from generator to myocardium via the lead and returns to the generator via the soft tissues. A major disadvantage of unipolar pacing is the proximity of the electrical circuit to skeletal muscle, which may result in skeletal muscle twitching. Advantages of unipolar pacing include smaller diameter of the pacing lead, a single attachment site of the lead to the epicardium when epicardial pacing is used, and superior sensing of endogenous cardiac potentials. The increased sensitivity of unipolar sensing may be, however, a double-edged sword in that this sensing modality will also create a greater likelihood of electromagnetic interference (EMI). Bipolar pacing systems have two closely spaced electrodes located distally on a transvenous lead or closely adjacent at two ends of an epicardial lead (Fig. 21.2). One electrode (usually the distal electrode on a transvenous bipolar lead) is the cathode, and the other is the anode. Electrical impulses travel to the cathode from the pulse generator and return to the anode to complete the circuit. In the majority of cases, a bipolar pacing system is preferred because there is less potential for EMI with bipolar pacing and because of absence of skeletal muscle stimulation.
- Although lead length should be given consideration before pacemaker implantation, length is often determined by availability. Most leads are longer than necessary, and excess length can be accommodated within the generator pocket. However, in large- or giant-breed dogs, adequate lead length may become an important factor.

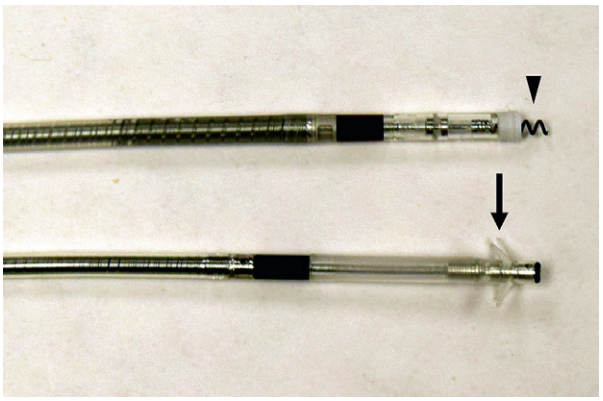


Fig. 21.1 The distal ends of two transvenous (endocardial) pacing leads are shown. The lead at the top is an active-fixation lead with the helix extruded, as it would be for attachment into the endocardium (*arrowhead*). The lead on the bottom is a passive fixation lead. Note the collar of tines (*arrow*), which become trapped within trabeculae to anchor the tip of the lead to the right ventricular (RV) endocardium.

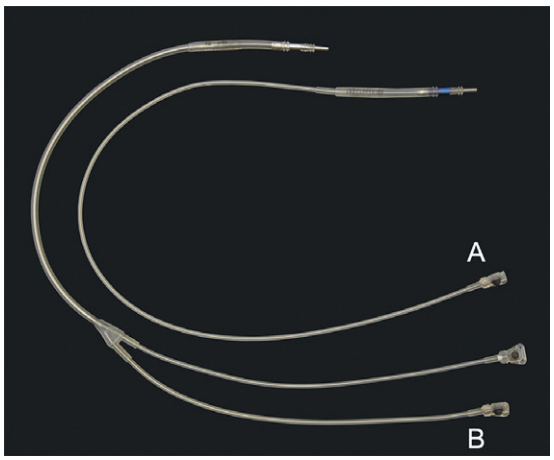


Fig. 21.2 Two epicardial pacing leads are shown. The inner lead (*A*) is a unipolar lead with a single suture-on electrode (cathode). The outer lead (*B*) is a bipolar lead with two suture-on electrodes (cathode and anode).

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Key Point

When a permanent pacing lead is selected, it is imperative that the lead be compatible with the pacing site selected (epicardial or endocardial, atrial or ventricular), that the lead and generator be of compatible size, that the lead's programmed polarity matches the generator's polarity settings, and that the lead is of sufficient length.

PACING WITH CHRONOTROPIC COMPETENCE (RATE RESPONSIVENESS)

- Initially, permanent cardiac pacing systems used in people and in dogs had a single lead that paced the ventricles at a constant rate. Later, rate-responsive pacing generators became available, which allowed the paced rate to vary depending on the activity of the patient.
- Rate response is achieved through use of sensors in the pacemaker; the most commonly used is that of an accelerometer detecting motion, which adjusts the paced rate between programmed upper and lower limits to match the level of exercise or rest. Thus rate-responsive pacing better mimics the normal physiologic response of the heart to exercise. Each pacemaker manufacturer has programming features to achieve rate-adaptive pacing. Success of patient-specific pacemaker programming can be examined through heart rate histograms, Holter analysis, Poincaré plots, and tachograms.

SINGLE-CHAMBER PACING COMPARED WITH DUAL-CHAMBER PACING

- The original goal of permanent cardiac pacing was to alleviate hemodynamic instability resulting from an abnormally low ventricular rate, and this remains the primary goal of pacemaker therapy in most veterinary patients. However, it is now recognized that cardiac output is dependent, not only on ventricular rate but also on the physiologic heart rate variation, synchrony between atrial and ventricular contraction, and ventricular activation sequence. Modern cardiac pacing has evolved in people from single-chamber to dual-chamber ventricular pacing (atrial and ventricular sensing and pacing) primarily to provide pacing with AV synchrony.
- AV synchrony is attained either by pacing the atrium or by sensing intrinsic atrial activity and tracking this activity. Either a paced atrial depolarization or an endogenous atrial depolarization triggers an AV delay, programmable in length, after which the ventricle is paced (if intrinsic ventricular activity is not sensed). Atrial synchronous pacing provides not only AV synchrony but also physiologic heart rate variation (see the discussion of pacing modes later).
- While AV synchrony may not be clinically important for many dogs and cats needing chronotropic support, pacing that provides AV synchrony will provide higher systemic pressure and lower ventricular filling pressures than single-chamber ventricular pacing. Therefore dual-chamber pacing to provide AV synchrony is likely to be important in animals with significant underlying structural heart disease or in working animals such as military dogs and agility dogs.
- Dual-chamber pacing has been utilized in veterinary medicine as a means of providing both heart rate response and AV synchrony. Disadvantages of dual-chamber pacing include its more complex programming, increased expense (when two leads are used), increased implantation procedural time, and the technical challenge of placing atrial-sensing/pacing leads in small patients.
- Normalization of the left ventricular activation sequence may be achieved by means of site-specific pacing within the right atrium and right ventricle.

SITE-SPECIFIC AND BIVENTRICULAR PACING

- The typical site for transvenous ventricular lead placement is at the RV apex because of the ease of placement and the security of passive lead fixation at this site. However, long-term pacing from the RV apex produces asynchronous ventricular contraction, impaired cardiac performance, and deleterious myocardial remodeling. Therefore alternative RV pacing sites have been proposed including septal, para-Hisian, and outflow tract sites and at times combined left ventricular and RV pacing (biventricular pacing).
- Transvenous biventricular pacing systems are feasible to implant in most medium- to large-breed dogs and should be considered for those with an expected long duration of pacing, significant structural heart disease, or heart failure.

PACEMAKER NOMENCLATURE

- Pacing nomenclature was established in 1974 and updated in 2002 for use in human medicine. This nomenclature also applies to veterinary pacemaker therapy, and awareness of it is important for understanding cardiac pacing. Pacing nomenclature classifies pacing based on the site and mode of both pacing and sensing using a series of three to five letters (Table 21.1).
- The first letter (position I) indicates the cardiac chamber or chambers in which pacing occurs: A = atrium; V = ventricle; D = dual chamber (both A and V); O = none.
- The second letter (position II) indicates the chamber or chambers in which sensing of electrical activity occurs. The letters are the same as those for the first position. (Some pacemaker manufacturers use the letter "S" in both the first and second positions to indicate that a generator is capable of pacing or sensing only a single cardiac chamber.)

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Table 21.1 Pacemaker Nomenclature for Antibradycardia Pacing

POSITION	I	II	III	IV	V
	CHAMBER(S) PACED	CHAMBER(S) SENSED	RESPONSE TO SENSING	RATE MODULATION	MULTISITE PACING
	O = None	O = None	O = None	O = None	O = None
	A = Atrium	A = Atrium	T = Triggered	R = Rate responsive	A = Atrium
	V = Ventricle	V = Ventricle	I = Inhibited		V = Ventricle
	D = Dual (A + V)	D = Dual (A + V)	D = Dual (T + I)		D = Dual (A + V)

From Bernstein AD, Daubert JC, Fletcher RD, et al: North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group: the revised NASPE/BPEG generic code for antibradycardia, adaptive-rate, and multisite pacing, *Pacing Clin Electrophysiol* 25:260–264, 2002.
Manufacturers' designation only: *S* = Single (A or V).

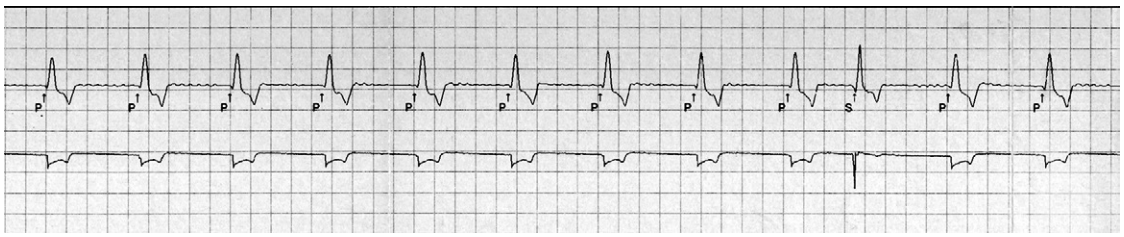


Fig. 21.3 A lead II electrocardiogram (ECG) rhythm strip (*top*) and a simultaneous intracardiac electrogram (*bottom*) recorded from the pacing lead of a canine patient with complete atrioventricular (AV) block and a bipolar VVIR pacemaker. Small arrows labeled *P* indicate pacing stimuli, and each small pacing stimulus artifact results in a paced QRS complex. The tenth QRS complex is an endogenous ventricular depolarization that is sensed by the generator, resulting in brief interruption of the paced rhythm. The sensed intrinsic activity resets the timing of the next paced beat (25 mm/s).

- The third letter (position III) refers to the mode of response to sensed electrical activity. In this position, the letter "I" indicates that a sensed electrical event inhibits the output pulse and causes the generator to recycle for one or more timing cycles. The letter "T" indicates that an output pulse is triggered in response to a sensed electrical event. When the letter "D" is in this position, it indicates that both "I" and "T" responses can occur, and this designation is limited to dual-chambered systems.
- The fourth letter (position IV) of the pacemaker nomenclature code refers to presence or absence of rate modulation. When the letter "R" is in this position, it designates that the generator has one or more sensors (such an accelerometer to sense motion) to adjust the paced heart rate independently of intrinsic cardiac activity.
- The fifth letter (position V) of the code is used to indicate whether multisite pacing is present in A = one or both atria; V = one or both ventricles; D = any combination of the atria and ventricles; O = none of the cardiac chambers. For example, a patient with a dual-chamber rate-responsive pacemaker with biventricular stimulation would be designated as having a DDDRV pacing system. The fifth letter is often omitted when describing pacing of veterinary patients because multisite pacing within the atria and ventricles is uncommon.

PACING MODES

- Currently the most commonly used pacing mode in veterinary patients and in human patients worldwide is single-chamber, ventricular-inhibited synchronous pacing either with (VVIR) or without (VVI) rate response. In this mode, the artificial pacing stimulus is delivered to the ventricle (the right ventricle if transvenous), and ventricular sensing allows sensed endogenous ventricular events to inhibit the pacemaker output. Inhibition of the output in response to endogenous ventricular activity is an important feature that prevents competitive rhythms and potentially fatal consequences of an electrical stimulus delivered during the vulnerable period of the cardiac cycle. A VVI (or VVIR) pacemaker is refractory for a specified, programmable interval after either a paced or sensed ventricular depolarization. This interval is the ventricular refractory period (VRP), and ventricular events occurring within the VRP will not reset the ventricular timing. However, an endogenous ventricular depolarization occurring after the VRP will be sensed, the generator output inhibited, and the timing cycle restarted from the intrinsic QRS complex. Thus the cardiac rhythm may be irregular on auscultation in patients with VVI or VVIR pacing, and RR intervals may vary on the ECG if there is intrinsic ventricular activity (Fig. 21.3).
- Single-chamber, atrial-inhibited pacing (AAI or AAIR) is identical to VVI pacing with the difference that pacing and sensing occur from the atrium, and pacemaker output is inhibited by sensed atrial events. The importance of maintaining both AV synchrony and synchronous ventricular contraction makes this pacing mode attractive for dogs

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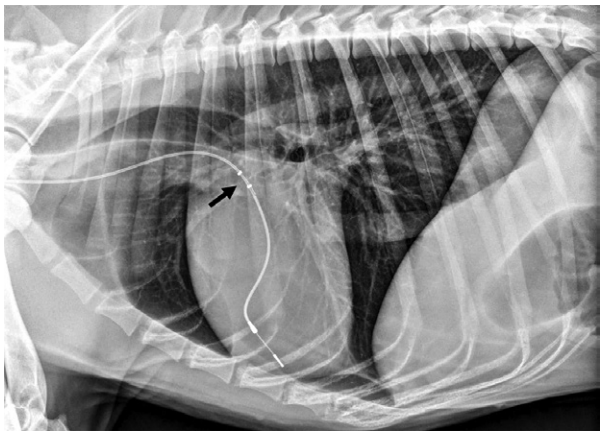


Fig. 21.4 Lateral radiograph obtained from a canine patient with a transvenous VDD pacing system. The ventricular electrodes are within the right ventricle (passive fixation) with the cathode at the ventricular apex. The floating atrial-sensing electrodes (*arrow*), located more proximally on the lead, are within the right atrium.

with sinus node dysfunction (sick sinus syndrome), particularly those with degenerative valve disease. The obvious disadvantage of atrial-based pacing is lack of ventricular depolarization should AV block occur. Therefore despite the observation that AV block occurs infrequently in dogs with sinus node dysfunction, 24-hour ambulatory ECG monitoring before pacemaker implantation and Wenckebach testing (see pacemaker implantation) of AV conduction at the time of implantation are recommended before permanent AAI pacing.

- Atrial synchronous pacing (VDD) has been utilized and reported in dogs with AV block. With this mode, pacing occurs only in the ventricle, sensing occurs in both chambers, and ventricular output is inhibited by intrinsic ventricular activity but stimulated by ventricular tracking of sensed atrial activity. In other words, there is ventricular pacing in response to endogenous P waves. A single pacing lead with a pair of floating atrial electrodes located on the intraatrial portion of the lead to sense atrial activity is typically used (Fig. 21.4). However, VDD pacing can also be accomplished using separate atrial and ventricular leads. In the VDD pacing mode, sensed atrial events initiate an AV delay. If an endogenous ventricular depolarization occurs during the AV delay, ventricular stimulation is inhibited, and the timing cycle is reset. If no endogenous ventricular activity occurs, then a paced ventricular beat occurs at the end of the AV delay, resetting the timing cycle. If no atrial event occurs, the pacemaker escapes with a paced ventricular depolarization at the lower rate. In other words, with VDD pacing the patient will be paced VVI in absence of sensed atrial activity. VDD pacing is appropriate only for animals with AV block and normal sinus node function. If a single VDD pacing lead is to be used, the patient must be large enough for the atrial-sensing electrodes (located 11.5–14 cm from the ventricular electrodes) to be placed in a stable position within or closely adjacent to the right atrium. Fig. 21.5 shows an ECG recorded from a dog with a VDD pacing system.
- Dual-chamber pacing and sensing with inhibition and tracking (DDD) is a common mode of antibradycardia pacing in humans. The primary difference between this mode and VDD mode is that in DDD mode, when there is absence of sensed intrinsic atrial activity, the atrium is paced, and the atrial-paced beat is tracked by ventricular pacing. Thus with the exception of ectopic endogenous ventricular depolarizations, AV synchrony is continuously present. This mode of pacing is important in humans with both sinus node dysfunction and AV block. However, most dogs with complete AV block have a normal sinus node, making atrial pacing unnecessary. Furthermore DDD pacing alone does not provide rate response during AV sequential pacing (DDDR is required).
- Dual-chamber pacing requires optimal programming of timing intervals such as atrioventricular delay, postventricular atrial refractory period, and total atrial refractory period to limit deleterious upper rate behavior and create a more physiologic ventricular response at maximum tracking rates.

PACEMAKER IMPLANTATION

- The rate of complications associated with pacemaker implantation is inversely related to the experience of the implanter. Therefore only highly experienced, well-qualified veterinarians should attempt pacemaker implantation.
- Whereas transvenous pacemaker implantation is generally done with sedation and local anesthesia in human patients, general anesthesia is used for veterinary patients to maintain aseptic technique during the implantation.
- Application of a temporary external pacing system before induction of anesthesia is strongly recommended because a rapid and profound decrease in heart rate may occur after induction of anesthesia.
- Regardless of the type of pacing lead used, specific measurements of lead impedance, amplitude, and pacing threshold(s) should be obtained using a pacemaker system analyzer at the time of implantation. These measurements assure optimal placement of the lead for pacing and sensing. Atrial pacing to determine the Wenckebach point (the heart rate producing progressive prolongation of AV nodal conduction followed by second-degree AV block) is also recommended if the use of a permanent atrial-based pacing system is being considered.
- Radiographs or saved fluoroscopic images should be obtained immediately after implantation to document final lead position(s).

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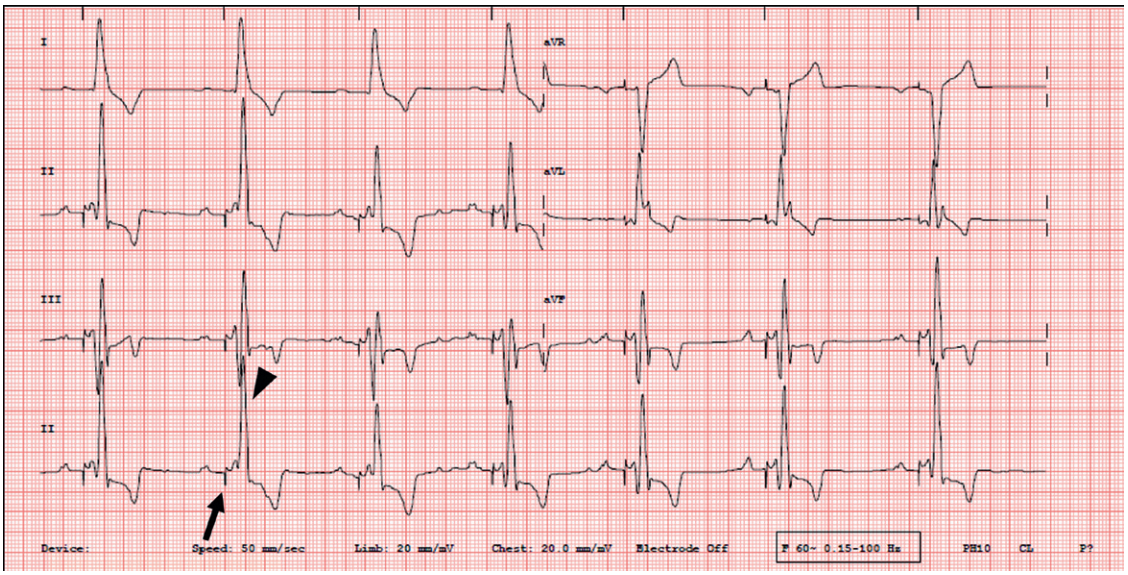


Fig. 21.5 This six-lead electrocardiogram (ECG) was recorded from a dog with a transvenous VDD pacing system implanted for treatment of third-degree atrioventricular (AV) block. The dog has normal sinus node function, resulting in P waves with sinus arrhythmia. The P waves are sensed by the generator, and after a sensed delay of 120 ms, there are pacing stimulus artifacts (*arrow*) followed immediately by paced QRS complexes that have left bundle branch block morphology (*arrowhead*) (50 mm/sec).

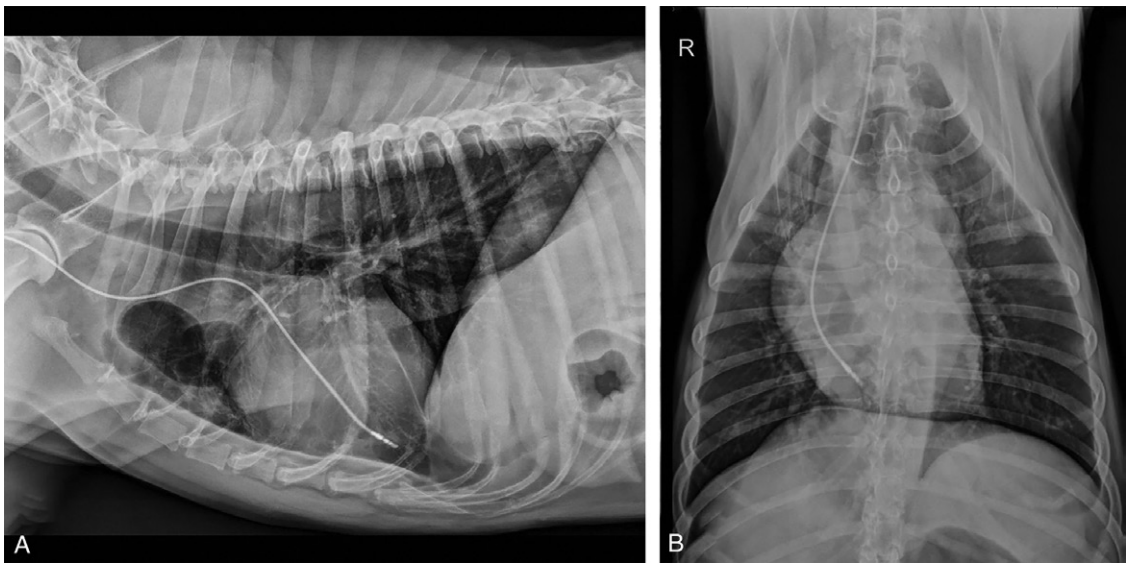


Fig. 21.6 Lateral **(A)** and ventrodorsal **(B)** thoracic radiographs showing typical placement of a transvenous pacing lead in a dog. The lead tip is in the right ventricular apex.

- A permanent transvenous ventricular pacing lead is typically inserted into the right external jugular vein as close to the thoracic inlet as possible and advanced fluoroscopically into the ventricle, leaving the lead tip in the most apical portion of the right ventricle angled toward the diaphragm (Fig. 21.6). The rare exception would be site-specific placement of a transvenous ventricular lead in the septum, para-Hisian, or RV outflow tract in an attempt to reduce mitral regurgitation and deleterious cardiac remodeling in patients with valvular disease or dilated cardiomyopathy.
- A permanent, transvenous atrial pacing lead with straight stylet is typically inserted into the right external jugular vein and advanced fluoroscopically until the lead tip is at the junction of the right atrium and caudal vena cava. The straight stylet is then replaced with a J-shaped stylet, and the lead

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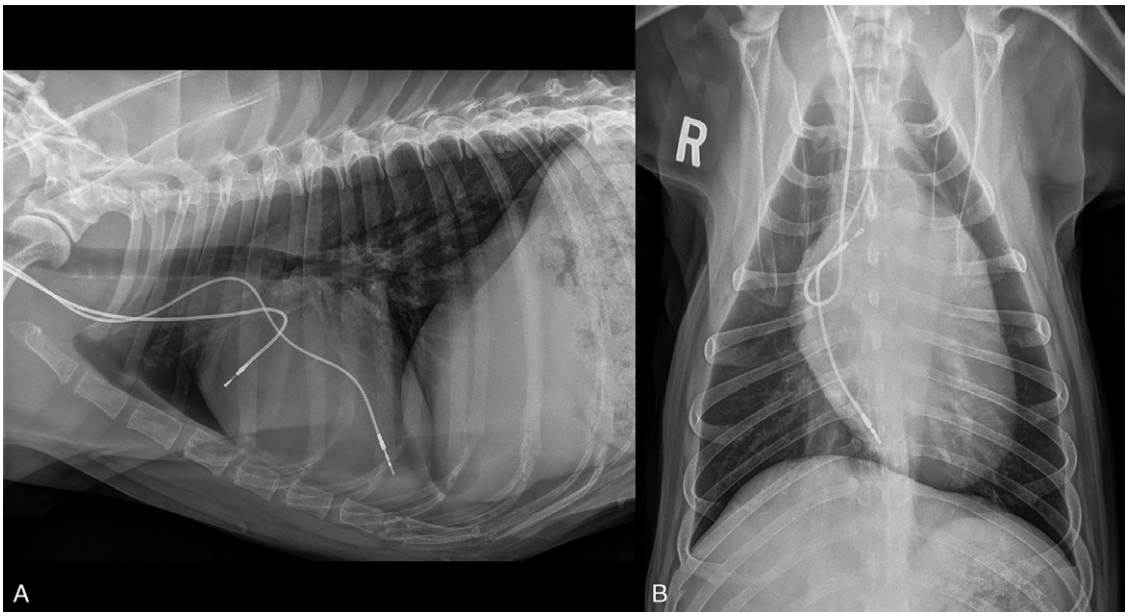


Fig. 21.7 Lateral **(A)** and ventrodorsal **(B)** thoracic radiographs from a dog with transvenous atrial and ventricular pacing leads. In this dog, the atrial lead was placed in the right auricle. (Courtesy Saki Kadotani, DVM, DACVIM [Cardiology]).

retracted to move the lead tip into the right auricle (Fig. 21.7). In small-breed dogs, active-fixation lead implantation in the interatrial septum may be necessary. If both a ventricular and an atrial lead are used, the ventricular lead is placed initially, followed by placement of the atrial lead.

- Right jugular venipuncture should not be attempted after transvenous pacemaker implantation, and a harness or gentle leader should be used instead of a neck lead to avoid damaging transvenous pacing wires.
- When a transvenous pacing system is used, the generator is usually placed into a subcutaneous pocket made on the right dorsolateral region of the neck. If a unipolar system has been used, skeletal muscle twitching in the area of the generator pocket may occur with each paced beat.
- Perioperative antibiotics are generally administered intravenously 20 minutes before beginning the implantation and repeated every 90 minutes until completion.

PACEMAKER COMPLICATIONS

- Permanent cardiac pacing, whether endocardial or epicardial, is relatively safe but not without risk of complication. Reported complications include lead dislodgement, lead perforation, infection, lead fracture, insulation break, battery issues, or programming errors. Most recent canine studies have shown relatively low complication rates (13%–33%) associated with transvenous cardiac pacing. There was no significant difference in the rate of major complications between epicardial and transvenous pacing systems. Similarly a study comparing major and minor complication rates of single-chamber pacing compared with dual-chamber pacing in dogs showed no significant differences.
- The most frequent major complication in dogs with artificial cardiac pacemakers is lead malfunction because of lead dislodgement. Lead dislodgement occurs more often with transvenous leads than with epicardial leads but is equally likely to occur with either active or passive fixation leads. Surprisingly this complication does not appear to be affected by the experience of the implanter, and it may be either acute or chronic (hours to months after implantation). There is some evidence to suggest that lead dislodgement is more common with atrial-based pacing than with ventricular pacing. Lead dislodgement may be radiographically apparent (macrodislodgement) or not radiographically apparent (microdislodgement), and radiographs showing lead position should be compared with radiographs or fluoroscopic images taken at the time of implantation (Figs. 21.6 and 21.8). Lead dislodgement often necessitates a second procedure to reposition the lead or place a second system (transvenous or epicardial). Occasionally, programming the generator to higher output or changing the pacing mode from bipolar to unipolar can reestablish effective pacing without repositioning of the lead.
- Another potential cause of lead malfunction is intermittent or complete failure of pacing because

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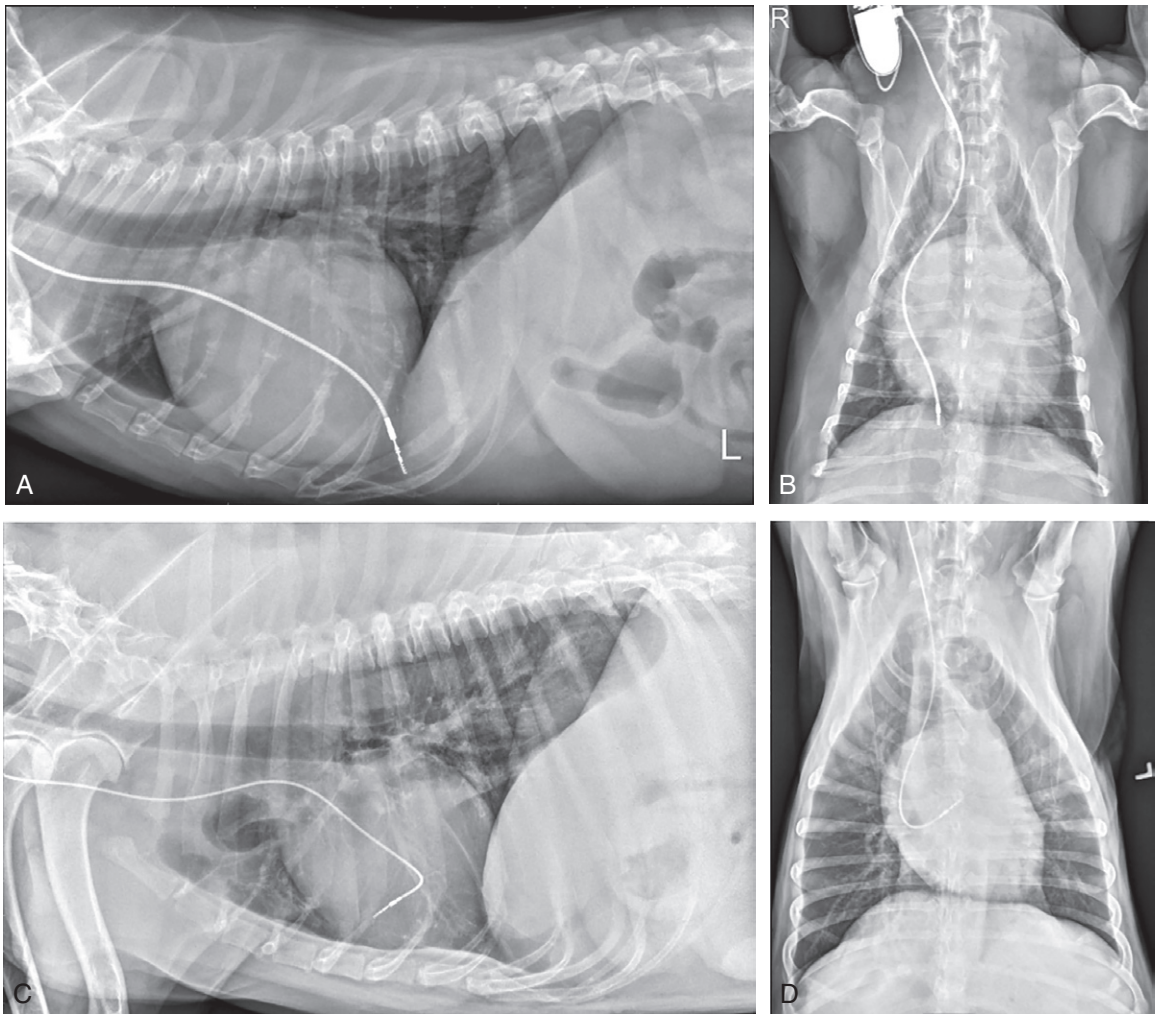


Fig. 21.8 Lateral **(A** and **C)** and ventrodorsal **(B** and **D)** thoracic radiographs from two dogs with transvenous pacing leads. Radiographs **(A)** and **(B)** were obtained from a dog with right ventricular perforation of the pacing lead. Radiographs **(C)** and **(D)** were obtained from a dog after dislodging the right ventricular (RV) pacing lead. Note that the distal tip of the lead is now located within the RV outflow tract rather than at the apex. This radiograph was taken shortly after recovery from anesthesia for pacemaker implantation.

of a loose connection at the interface of the lead and the connector block of the generator. This problem is usually the result of inadequate securing of the lead at the time of implantation. When connection of the lead to the generator is loose, manipulation of the generator or generator pocket may induce the pacing malfunction. The poor connection may also be apparent radiographically (Fig. 21.9). A second operative procedure to secure the lead pin into the generator connector block is needed to restore reliable pacemaker function.

- Lead fracture or lead insulation breaks are causes of pacemaker malfunction occasionally encountered in veterinary patients. Fractures or insulation breaks may cause impaired sensing, impaired pacing, or both. These complications may result from biting injuries, venipuncture, excessive repetitive lead motion, or traction with a neck leash. Lead fracture or lead insulation breaks are often identified by unacceptably high or low lead impedance measurements. Lead fractures may also be identified radiographically (Fig. 21.10).
- Exit block refers to failure of the pacing stimulus to depolarize the myocardium (failure to capture), a complication often caused by development of fibrous tissue at the electrode cardiac interface. Fibrosis is frequently secondary to inflammation incited at the time of implantation, and use of leads with steroid eluting tips is likely to minimize or prevent this problem. However, exit block may also occur from progression of underlying myocardial disease.
- Perforation of the right atrial or RV wall by a transvenous pacing lead is a rare complication that may occur at implantation or chronically. This complication may result in acute, fatal hemorrhage or loss of effective cardiac pacing caused by failure to capture.
- Pacemaker infections may involve the pocket, lead, or both and may cause fatal septicemia or endocarditis. In dogs, most pacemaker infections occur

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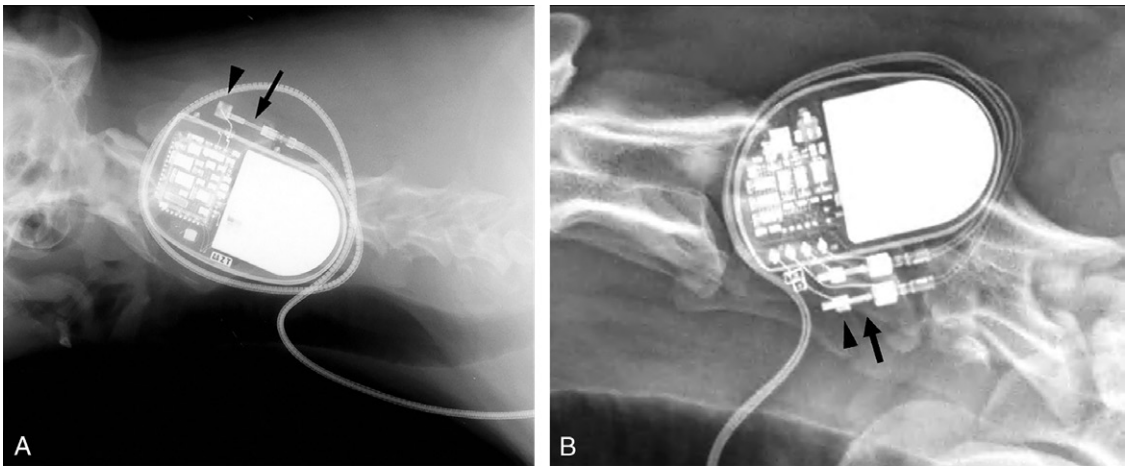


Fig. 21.9 **(A)** This radiograph was obtained from a dog with intermittent failure to pace caused by a loose connection at the interface of the unipolar lead and the connector block. The loose connection occurred because the lead pin was not adequately secured at the time of pacemaker implantation. Note that the connector pin (*arrow*) has withdrawn from the block (*arrowhead*) and is not passing all the way through the block screw. **(B)** For comparison, this radiograph shows an appropriately engaged connector pin from a patient with a transvenous VDD pacing lead. The connector pin (*arrow*) is visible beyond the connection block screw (*arrowhead*).

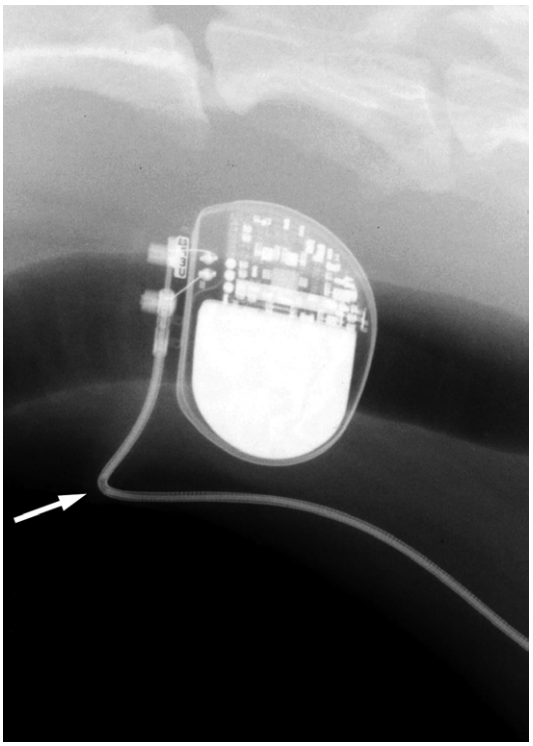


Fig. 21.10 This radiograph was taken from a dog that experienced failure of a pacemaker system because of lead fracture. The fracture is visible at the tip of the arrow.

within 3 months after implantation. With very few exceptions, treatment of an infected pacemaker or lead should consist of removal and replacement of the entire system. However, removal of a chronically implanted transvenous pacing lead is often difficult and may result in a heart wall tear, air embolism, or cardiovascular avulsion.

- Other potentially lethal pacemaker complications reported in dogs and cats include generator failure, significant arrhythmias such as ventricular asystole, atrial fibrillation, ventricular tachycardia, and ventricular fibrillation, development of congestive heart failure, cranial vena cava syndrome, and chylothorax. Extensive thrombosis and thromboembolism may also occur.
- Minor complications associated with pacemaker implantation include formation of hematomas and seromas at the generator or cervical lead site, skeletal muscle twitching at the generator site, transient or minor arrhythmias, and suture line dehiscence. For contamination or lead damage to be avoided, needle aspiration or other forms of mechanical drainage of seromas or hematomas should not be used. Alternatively conservative management with pressure, warm compresses, and prophylactic antibiotics is recommended.
- EMI is a pacemaker complication that occurs when any signal, either biologic or nonbiologic, originating outside of the heart is detected by the sensing circuitry of the pacemaker. EMI can result in inappropriate inhibition of pacing, asynchronous pacing (cardiac pacing in which impulse generation by the pacemaker occurs at a fixed rate, independent of underlying cardiac activity), damage to the generator or myocardium, or reprogramming of the pacing parameters. Sources of EMI include electrocautery, electrical cardioversion or defibrillation, magnetic resonance imaging, and electroshock therapy.

PACEMAKER PROGRAMMING AND FOLLOW-UP

- State-of-the-art pacemakers have programmable parameters that can be evaluated and altered to optimize and monitor function of the pacing system. Although there is some variation between

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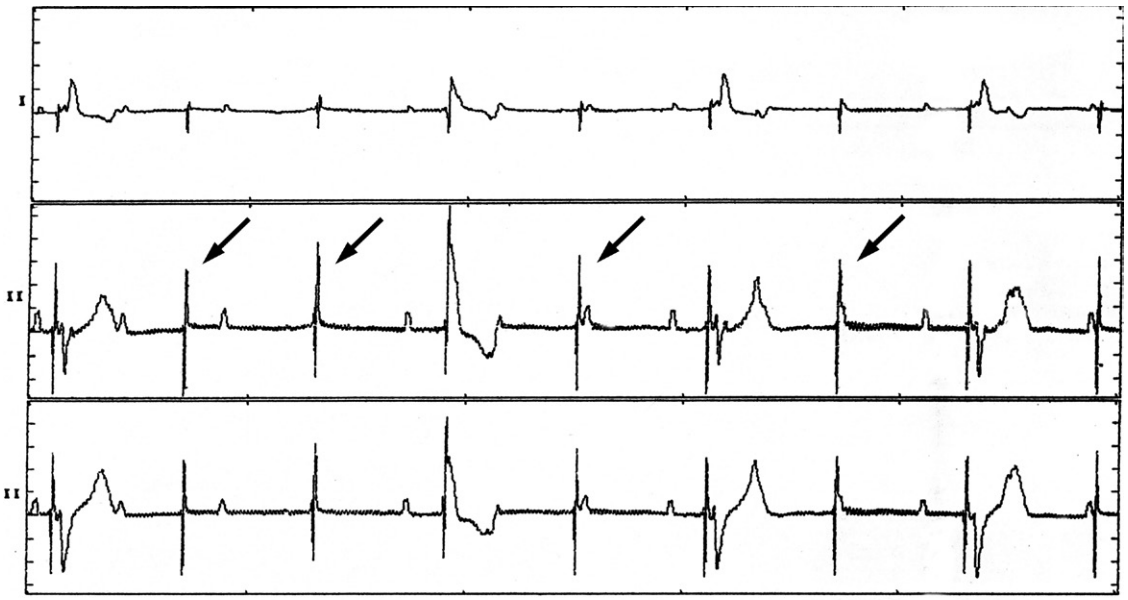


Fig. 21.11 A simultaneous lead I, II, and III electrocardiogram (ECG) recorded from a dog with complete atrioventricular block and a malfunctioning unipolar pacemaker. The ECG shows numerous pacing stimuli that do not depolarize the myocardium (intermittent failure to capture) (*arrows*).

generators, typical data obtained during a pacemaker programming and evaluating session would include output current, output voltage, lead impedance, battery status, sensitivity (sensing parameters), event records, ECG monitoring, pacing histogram, and pacing thresholds. A real-time intracardiac electrogram can be displayed with a simultaneous surface ECG to show timing of pacing and sensing on the monitor (see Fig. 21.3).

- Output programming, referring to programming of the pulse width and voltage amplitude of the pacing signals, is the most important aspect of programming that should be done routinely. Output should be high enough to provide an adequate pacing margin of safety while also maintaining output as low as possible to maximize battery longevity. Although there is no consensus regarding the best way to program output parameters, acceptable methods include doubling the threshold voltage amplitude, tripling the pulse width at threshold, and plotting strength duration curves. Because capture threshold usually increases immediately after pacemaker implantation as healing occurs, energy output should be set relatively high at implantation and then reprogrammed after healing (approximately 4–8 weeks after implant).
- Sensing parameters also need to be programmed and checked. Appropriate sensing of intrinsic cardiac activity is extremely important for proper pacemaker function, whether the patient has a single- or dual-chamber pacing system. Oversensing of T waves in canine patients is a common programming error. This problem can usually be corrected with an increase in the sensing threshold value.
- Appropriate programming of the refractory period is also essential for correct pacemaker function. If the refractory period is too long, intrinsic QRS complexes may cause multiple restarts of the refractory period, causing the generator to switch to asynchronous pacing. The recommended method for programming of the refractory period is that the refractory period should include the T wave and be slightly longer than the QT interval; however, dogs and cats with concurrent tachyarrhythmias may require programming with a slightly shorter refractory period to prevent noise reversion (an algorithm that causes the pacemaker to switch to asynchronous pacing when repetitive sensing at a high rate occurs).
- Although a pacemaker programmer is often necessary for troubleshooting and thoroughly evaluating pacemaker function, a standard ECG recording may also be helpful. The ECG appearance of paced beats differs from that of endogenous beats. A paced beat includes a pacing stimulus artifact, a depolarization wave, and a repolarization wave. The pacing stimulus artifact is typically small with bipolar pacing and relatively large with unipolar pacing (see Fig. 21.5; Fig. 21.11). The QRS shape of a paced beat will depend on location of the ventricular pacing lead; transvenous pacing from the RV apex usually produces QRS complexes with a left bundle branch block configuration in the frontal plane leads (see Fig. 21.5). If the pacing mode is VVI or VVIR, the paced beats are inhibited by sensed spontaneous beats, and the basic pacing cycle is reset.
- ECG abnormalities in patients with pacemakers may be broadly classified as failure to capture,

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failure of output or pacing, and abnormal sensing. Failure to capture is recognized as a pacing stimulus artifact without ventricular depolarization (see Fig. 21.11). Causes of failure to capture include lead dislodgement, high thresholds with inadequately programmed output, partial lead fracture, insulation defect, impending battery depletion, poor or incompatible connection of the lead to the generator, and functional noncapture (pacing stimulus during the refractory period of a spontaneous beat).

- Failure to pace is recognized as failure of the generator to deliver an appropriately timed stimulus. This problem is often caused by oversensing, but it may be caused by true failure of the generator or circuit interruption (the electrical signal does not reach the heart). Reasons for true failure to output include circuit failure, complete or intermittent lead wire fracture, intermittent or permanent loose connection to the connector block, incompatible lead, battery depletion, internal insulation fracture (bipolar lead), and lack of anodal contact (unipolar lead). When a pacemaker battery reaches the end state of depletion, either failure to capture because of reduced voltage output or failure to pace because of total battery depletion may occur.
- Sensing abnormalities, both under- and oversensing, may also be recognized on a surface ECG in many patients. Causes of sensing abnormalities include lead dislodgement or poor lead positioning, inappropriate settings, lead insulation failure, circuit failure, magnet application, malfunction of the generator (reed switch), EMI, and battery depletion.

Frequently Ask Questions

WHEN AND WHY SHOULD DUAL-CHAMBER PACING BE DONE?

Dual-chamber pacing provides AV synchrony, whereas single-chamber ventricular pacing does not. Pacing with AV synchrony reduces ventricular filling pressures and mitral regurgitation compared with VVI (or VVIR) pacing. Therefore dual-chamber pacing is preferable in patients with chronic valvular disease or myocardial disease. AV synchrony is also essential for high-level athletic activity, and therefore dual-chamber pacing should be considered in athletic dogs and working dogs. However, placement of a VDD lead or two conventional transvenous leads is technically difficult in animals weighing less than 10 kg.

WHAT ARE THE MAJOR ADVANTAGES OF ATRIAL SYNCHRONOUS PACING (VDD PACING) COMPARED WITH VENTRICULAR-INHIBITED SYNCHRONOUS PACING (VVI PACING)?

The advantages of VDD pacing include chronotropic competence based on intrinsic sinus rate and restoration of AV synchrony. A disadvantage of this pacing mode is that it is appropriate only for treatment of patients with bradycardia caused by AV block. Therefore this method of pacing cannot be used in patients with atrial fibrillation, atrial standstill, or sinus node dysfunction. Furthermore, VDD pacing is limited to patients large enough to accommodate two leads or a special VDD lead.

WHAT IS AN APPROPRIATE DIAGNOSTIC APPROACH FOR A PATIENT WITH AN IMPLANTED PACEMAKER WHO HAS A RECURRENCE OF CLINICAL SIGNS SUGGESTIVE OF BRADYARRHYTHMIA?

A physical examination and standard ECG should be done. The physical examination will confirm bradycardia if the pacemaker dysfunction is continuous. Occasionally, event recording or Holter monitoring is needed to confirm bradycardia that is episodic as a result of intermittent pacemaker dysfunction. Physical examination may occasionally reveal a cause of pacemaker dysfunction, such as significant generator migration causing traction on a pacing lead. Standard ECG is used to identify the underlying rhythm, which, in turn, may confirm sustained pacemaker malfunction. In addition, standard ECG may help identify a specific cause of pacemaker malfunction. For example, regular pacing stimuli that occur at a rate below the lower programmed rate suggest battery depletion. Pacing stimuli with appropriate timing but without capture may indicate lead dislodgement, lead fracture, lead insulation defect, or inadequate capture threshold. Survey radiographs may confirm fractures or macrodislodgements of pacing leads. Use of a pacemaker programmer to evaluate output and sensing, to test lead impedance, and to determine capture thresholds is often necessary for definitive diagnosis of the cause of pacemaker dysfunction. Holter monitoring or event recording may be needed to confirm or refute intermittent tachyarrhythmias as the cause of the clinical signs if no evidence of pacemaker malfunction is found and ECG rhythm strips during tachycardia episodes are not available from the memory of the generator.

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Chapter

22

Nutrition and Cardiovascular Disease

Lisa M. Freeman and John E. Rush

Optimal nutrition is an integral part of the overall medical treatment for cats and dogs with cardiac disease. Key nutritional concerns include maintenance of appetite and optimal body composition, avoiding nutritional deficiencies and excesses, and gaining benefits of targeted dietary supplementation. However, the same diet is not optimal for every dog or every cat with cardiac disease, and to optimize care for patients it is important to individualize the nutritional plan. General recommendations such as "feed a senior diet" or "give a renal diet" are not ideal and even general recommendations such as "feed any cardiac diet" will not optimize the overall care of these patients.

GENERAL PRINCIPLES

NUTRIENTS OF CONCERN IN CARDIAC DISEASE

Dogs and cats with cardiac disease have a variety of pathophysiologic changes, such as activation of the renin-angiotensin-aldosterone system, sodium and water retention, and, depending on stage of disease and medication regimen, electrolyte alterations. As a result, nutritional alterations are often present in animals with cardiac disease. These nutrients are considered "nutrients of concern" in animals with cardiac disease.

Common Nutritional Alterations in Cardiac Disease

- Reduced calorie intake. Once congestive heart failure (CHF) arises, alterations in appetite are common. These can include a complete loss of appetite (anorexia), a reduction in food intake (hyporexia), or changes in food preferences (dysrexia). These all contribute to reduced food intake and resulting weight and muscle loss, which are common in CHF. These changes in appetite can be the result of increased production of inflammatory mediators (e.g., inflammatory cytokines, oxidative stress), side effects of cardiac medications, or poor control of CHF signs (especially in dogs with ascites). Although many animals with cardiac disease exhibit weight loss, particularly with more advanced disease, animals with cardiac disease also can be overweight or obese (with or without muscle loss). While avoiding overweight and obesity is an important goal for healthy animals, once CHF is present, being overweight may actually be associated with a protective effect; this is known as the *obesity paradox*. This appears to be related primarily to more muscle in most overweight patients, rather than any direct benefits of excess fat.
- Increased protein catabolism resulting in muscle loss. Muscle loss (cachexia) is common (42%–69% of dogs and cats with CHF), particularly as the disease progresses. This muscle loss is unlike that seen in a healthy animal that loses weight, which primarily loses fat. If an animal with CHF does not get sufficient calories and protein, the primary tissue lost is muscle. The muscle loss in cardiac cachexia is a multifactorial process caused by decreased calorie intake, increased energy requirements, and increased production of inflammatory cytokines (e.g., tumor necrosis factor [TNF] and interleukin-1 [IL-1]). These cytokines decrease appetite, increase energy requirements, and increase the catabolism of lean body mass. The term *cachexia* does not necessarily refer to an emaciated, end-stage patient. In fact, in the early stages, it can be very subtle and even may occur in obese animals. Muscle loss usually is noted first in the epaxial, gluteal, scapular, or temporal muscles. Cardiac cachexia has deleterious effects on strength, immune function, and survival, so it is important to recognize this muscle loss at an early stage when there is a better opportunity to successfully manage it. Feeding a diet that is restricted in protein (e.g., renal diets, some senior diets) may contribute to unwanted muscle loss.
- Taurine deficiency. Taurine can play a role in the development of feline dilated cardiomyopathy (DCM) and in some cases of canine DCM. Taurine deficiency–induced DCM is now uncommon in cats but should be suspected if cats are eating diets that are vegetarian, home prepared, or designed for "intermittent or supplemental use." Some dogs with DCM also can have taurine deficiency, with Cocker Spaniels, Golden Retrievers, and Newfoundlands appearing to be predisposed.

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However in diet-associated DCM identified in dogs eating high-pulse diets (see FAQs below), taurine deficiency is uncommon.

- Omega-3 fatty acid "deficiency." In addition to serving as a source of calories and essential fatty acids, fat also can have significant benefits on inflammation, immune function, and hemodynamics. Most pet foods contain primarily omega-6 fatty acids (e.g., linoleic acid, arachidonic acids). The omega-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) normally are found in very low concentrations in the cell membrane compared with the omega-6 fatty acids, but their level can be increased by consumption of a food or supplement enriched in omega-3 fatty acids. In one study, dogs with CHF had a relative deficiency of omega-3 fatty acids, which was corrected after supplementation of omega-3 fatty acids. However, omega-3 fatty acid status in cardiac disease may differ between breeds of dogs and between species. The benefit of having a higher concentration of omega-3 fatty acids in cell membranes is that breakdown products of the omega-3 fatty acids (eicosanoids) are less potent inflammatory mediators than eicosanoids derived from omega-6 fatty acids. Increasing consumption of omega-3 fatty acids decreases the production of cytokines and other inflammatory mediators. Fish oil also reduces the production of TNF and IL-1 and improves muscle mass. In some animals, fish oil supplementation also improves appetite. Another potential benefit is that omega-3 fatty acids have antiarrhythmic effects.
- Mineral excess or deficiency
  - Sodium. Increased retention of sodium and water is well recognized in animals with cardiac disease because of activation of the renin-angiotensin-aldosterone system and other neuroendocrine systems. In contrast, hyponatremia that develops in late-stage CHF due to excessive free water retention can be associated with adverse outcomes.
  - Potassium. Depending on the stage of disease and medications being administered, animals with cardiac disease can be hyperkalemic (with angiotensin-converting enzyme [ACE] inhibitors or aldosterone receptor antagonists), hypokalemic (with loop diuretics), or normokalemic. Hypokalemia can cause overall weakness and increase the risk for arrhythmia. Hyperkalemia can reduce appetite and cause dogs to feel generally unwell.
  - Magnesium. High doses of diuretics can increase the risk for hypomagnesemia, which can increase the risk for arrhythmia. However, in animals with concurrent chronic kidney disease, hypermagnesemia may develop.
- Vitamin deficiency. B vitamin deficiencies appear to be relatively common in humans with CHF and are likely caused by urinary losses secondary to diuretic use. Similar studies have not been published in dogs with CHF, but one study in cats with cardiomyopathies showed reduced serum concentrations of vitamins B_6 and B_{12}.
- Pulses, potatoes, and sweet potatoes. Pet food ingredients associated with the form of diet-associated DCM currently under investigation by the US Food and Drug Administration (FDA) are pulses (peas, lentils, chickpeas, dry beans) and, to a lesser degree, potatoes and sweet potatoes (see FAQs below). Until more is known, we recommend avoiding diets containing any pulses, potatoes, or sweet potatoes in dogs and cats with heart disease (or, at least avoiding diets with these in the top 10 ingredients or those that have more than one of them anywhere on the ingredient list).

In addition to the above nutrients of concern, there are a number of other nutrients with potential pharmacologic effects (e.g., L-carnitine, coenzyme Q10, antioxidants, arginine) that are based on pathophysiologic changes in cardiac disease (e.g., altered myocardial energetics, increased oxidative stress, endothelial dysfunction). Some nutrients, such as omega-3 fatty acids and taurine, may act through pharmacologic and nutritional effects. For example, taurine, as an essential nutrient for cats, may have benefit by correcting a nutritional deficiency. However, taurine also has mild antioxidant and positive inotropic properties.

It is important to be aware that the potential nutrients of concern listed above do not mean that *all* cats with hypertrophic cardiomyopathy (HCM) or *all* dogs with myxomatous mitral valve disease (MMVD) require modification of *all* of these nutrients or that they should be supplemented or restricted to the same levels. This would be similar to the concept of recommending the same dose of furosemide for all dogs with MMVD, no matter what the stage of disease. Instead one should consider the underlying disease (e.g., DCM, HCM, MMVD, congenital heart disease) and the stage of disease (e.g., preclinical or mild, moderate, or severe heart failure), but also the following factors:

- Appetite: Normal, hyporexia, anorexia, or dysrexia. Dysrexia is the most common abnormal appetite finding in animals with CHF.
- Physical examination findings, especially body condition score (BCS), muscle condition score (MCS), and gain or loss in body weight.
- Laboratory results (e.g., hypoalbuminemia, azotemia, hypokalemia or hyperkalemia, hypomagnesemia).
- Concurrent diseases. Concurrent diseases are present in most animals with cardiac disease and can have significant effects on the nutritional modifications that will be optimal for that patient.
- Animal preferences.
- Owner preferences.

On the basis of all these factors, a nutritional plan can be individualized for each animal. For the

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development and execution of this plan, there are five key steps: (1) performing a nutritional assessment, (2) making individualized nutritional recommendations, (3) addressing all components of the diet, (4) communicating with the owner, and (5) reassessing nutritional needs at each recheck examination.

PERFORM A NUTRITIONAL ASSESSMENT ON EVERY PATIENT

A good nutritional assessment of the animal's current dietary intake and preferences should consider the following five questions:

1. Is the current diet contributing to the cardiac disease (or other medical conditions)? Examples include an animal with CHF eating a very high-sodium diet, a cat with DCM eating a vegetarian diet, or a dog with severe muscle loss eating a diet designed for animals with renal disease. Even if the diet is not directly affecting the cardiac disease, it could be contributing to suboptimal health (e.g., an unbalanced home-prepared diet, excessive calories contributing to obesity).
2. Is the current diet optimized for managing the cardiac disease?
3. Are all of the components of the animal's current diet known, including treats, supplements, and foods used to administer medications? The entire diet may be composed of pet food, but it more likely also includes home-prepared food, treats, table food, dietary supplements, rawhides or dental chews, and a variety of other sources of nutrients that must be addressed.
4. What are the animal's taste preferences? Recommending the preferred formulation (dry or canned), flavor, and even kibble shape and size can be helpful for feeding the hospitalized animal, as well as for making long-term recommendations.
5. Are the owners' preferences understood, such as type of food, the importance of treats and table food, and ability to administer medications?

Nutritional assessment consists of a screening evaluation and, if any "red flags" are identified, an extended evaluation should follow. The screening evaluation can quickly identify nutritional issues in the animal with cardiac disease that require further evaluation. The screening evaluation can be achieved by having the owner complete a short diet history form and by performing a physical examination.

- Diet history. Standardized diet history forms make gathering complete information more efficient and ensure that the required information is collected for every patient. A complete diet history includes the main pet food being provided (or the home-prepared diet recipe being used, if any), but also treats, table food, dietary supplements, and foods used for medication administration. Also even if the dog or cat is discharged with an optimal diet plan, one should not assume that the owner is following these recommendations at subsequent visits; ask exactly what the owner is feeding at each visit. See Fig. 22.1 for a cardiac-specific diet history form. Also see the World Small Animal Veterinary Association Nutrition Toolkit or the American College of Veterinary Nutrition websites for more general diet history forms.
- Body weight. Use the same scale at each visit to identify subtle changes in body weight, which may reflect altered nutritional status or fluid accumulation.
- BCS. Use a consistent method to assess and record BCS at each visit (either a 9- or a 5-point scale). The BCS evaluates body fat and can identify animals that are overweight or obese, as well as animals that are below ideal body weight.
- MCS. The MCS differs from the BCS in that the MCS evaluates muscle mass. Animals can be very obese (9/9) and have significant muscle loss. Conversely they can be thin (3/9) but have normal muscle mass. Evaluation of the MCS includes visual examination and palpation over the temporal bones, scapulae, lumbar vertebrae, and pelvic bones. Muscle condition is graded as normal or as mild, moderate, or severe muscle loss (Fig. 22.2), rather than using a numerical score. With an assessment of muscle condition at every visit, muscle loss can be identified at its early stages when intervention is more likely to be successful.
- Presence of other medical conditions that also require dietary modifications (e.g., gastrointestinal disease, feline lower urinary tract disease, chronic kidney disease).

If any of these issues are identified on the screening evaluation, additional diet information may be needed so that an optimal individualized nutritional plan can be developed.

MAKE INDIVIDUALIZED NUTRITIONAL RECOMMENDATIONS

The same diet will not be optimal for all animals with cardiac disease. For example, for a 9-year-old spayed female Cavalier King Charles Spaniel with MMVD, mild, well-controlled CHF, normal laboratory values, a BCS of 7/9, and no muscle loss, for which the owner prefers a combination of canned and dry food, the main nutrients of concern would be calories (avoiding any additional weight gain), protein (ensuring adequate protein intake), and sodium (mild sodium restriction <100 mg/100 kcal). The clinician can then make recommendations for several specific canned and dry options (or provide a link to a curated low-sodium diet list, such as the Tufts HeartSmart website [https://heartsmart.vet.tufts.edu/]) from which the owner could choose. However, if that same dog now had more advanced CHF, with a BCS of 4/9,

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DIET HISTORY FORM FOR ANIMALS WITH HEART DISEASE

Please answer the following questions about your pet

Today's date: _____

Pet's name: _____ Owner's name : _____

1. How would you assess your pet's appetite? (mark the point on the line that best represents your pet's appetite)
   Poor **Excellent**
   _____

2. Describe your pet's appetite over the last few weeks? (check all that apply)
   ☐Eats about the same amount as usual ☐Eats less than usual ☐Eats more than usual
   ☐Seems to prefer different foods than usual

3. Over the last few weeks, has your pet (check one)
   ☐Lost weight ☐Gained weight ☐Stayed about the same weight ☐Don't know

4. Please list below the brands and product names (if applicable) and the amount of ALL foods, treats, snack, dental chews, rawhides, and any other food item that your pet currently eats.

Food	Form	Amount	How often?	Fed since
Examples:				
Purina Dog Chow	dry	1 ½ cups	2x/day	Jan, 2011
Science Diet Adult Gourmet Beef Entree	can	½ large can	1x/day	Jan, 2012
90% lean hamburger	microwaved	3 oz	1x/week	Jan, 2011
Milk Bone medium	dry	1	2x/day	Aug, 2010
Rawhide	dry	2x6" strip	3x/week	Dec, 2011

5. Do you give any dietary supplements to your pet (for example: vitamins, glucosamine, fatty acids, or any other supplements)? ☐Yes ☐No If yes, please list which ones and give brands and amounts:

		Brand	Dose
Taurine	☐Yes ☐No	_____	_____
Carnitine	☐Yes ☐No	_____	_____
Antioxidants	☐Yes ☐No	_____	_____
Multivitamin	☐Yes ☐No	_____	_____
Coenzyme Q10	☐Yes ☐No	_____	_____
Fish oil	☐Yes ☐No	_____	_____
Flaxseed, cod liver oil, or other fatty acids?	☐Yes ☐No	_____	_____
Other (please list)	☐Yes ☐No	_____	_____
		_____	_____
		_____	_____

6. How do you administer pills to your pet?
   ☐I do not give any medications
   ☐I give them without any food
   ☐I put them in my pet's dog/cat food
   ☐I put them in a Pill Pocket or similar product
   ☐I put them in other foods (such as cheese or lunch meat; please list foods):_____

Information below to be completed by the veterinarian:

Current body weight: _____kg Ideal body weight: _____kg

Current body condition score: _____/9

Muscle Condition Score: ☐normal muscle ☐mild muscle loss ☐moderate muscle loss ☐severe muscle loss

Fig. 22.1 A sample diet history form designed to collect important nutritional information for dogs and cats with cardiac disease.

moderate muscle loss, dysrexia, hyperkalemia, and hypoalbuminemia, the nutrients of concern (and the levels to which nutrients should be modified) will be very different. In this situation, a high-calorie density and palatable diet, with increased protein and low to moderate potassium concentrations, would be ideal. Concurrent diseases also alter diet choice and are present in many animals with heart disease. The optimal diet for a cat with HCM alone is very different from that of a cat with HCM and chronic kidney disease or feline lower urinary tract disease. Finally, the animals' preferences and the owners' preferences (e.g., canned or dry, brand, type, homemade or commercial, and veterinary or over-the-counter diet) must be taken

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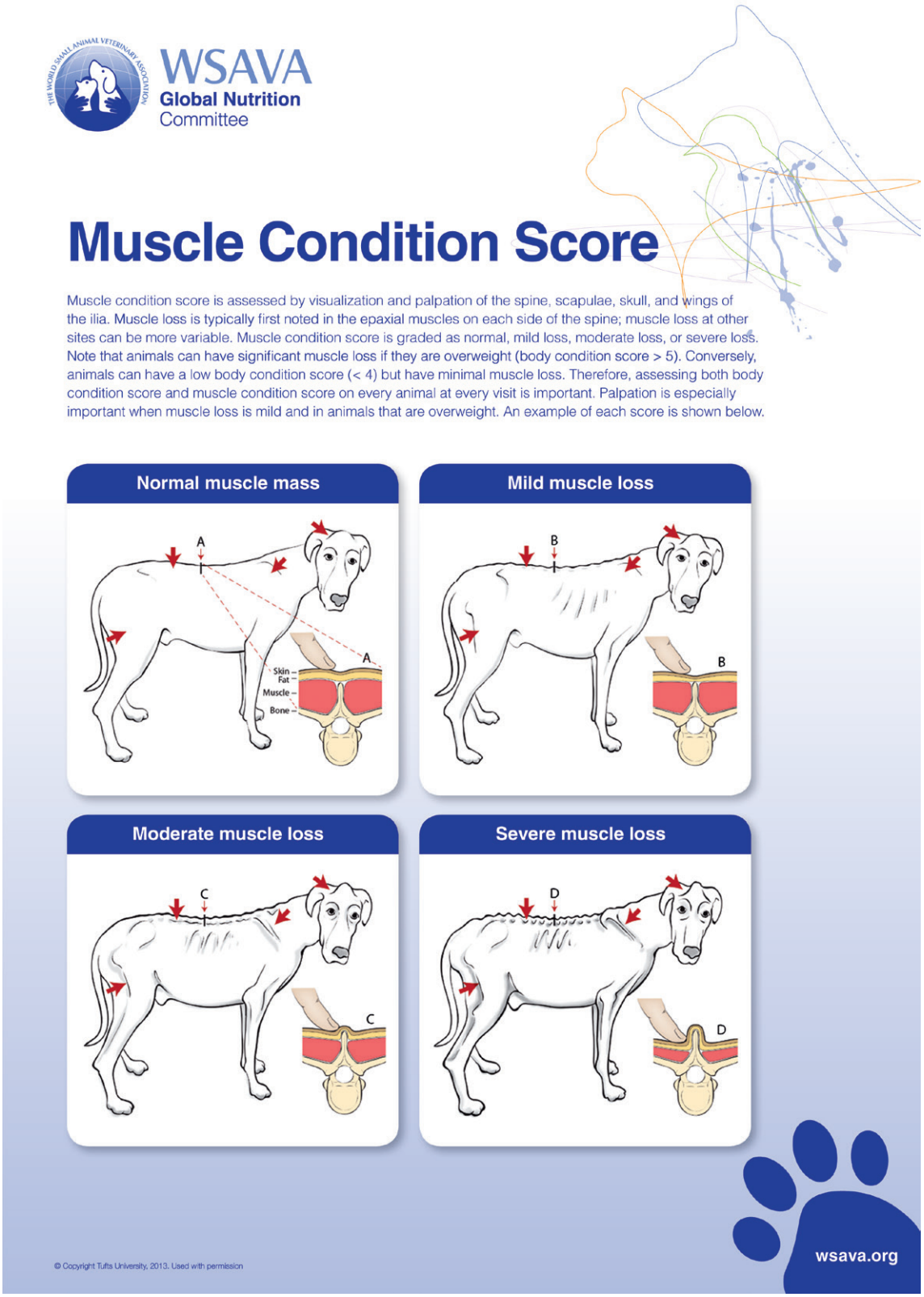


Fig. 22.2 Muscle condition score (MCS) charts for dogs and cats. Compared with the body condition score, which assesses fat stores, the MCS assesses the animal's muscle, which can be affected by disease or aging. Provided courtesy of the World Small Animal Veterinary Association (WSAVA). Available at the WSAVA Global Nutrition Committee Nutritional Toolkit website: https://www.wsava.org/nutrition-toolkit. Copyright Tufts University, 2014.

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into consideration for good compliance and successful treatment.

On the basis of patient characteristics (and owner preferences), one or more diets can be selected. One general recommendation for heart disease is to avoid protein-restricted diets (e.g., renal diets or diets with protein <5.1 g/100 kcal for dogs or 6.5 g/100 kcal for cats), unless concurrent advanced kidney disease is present. Currently several commercial veterinary cardiac diets for dogs are available in the United States. These diets are moderately to severely sodium restricted (21–50 mg/100 kcal). However, they vary widely in calories, protein, total fat, phosphorus, and the omega-3 fatty acids (EPA and DHA), among other differences (e.g., chloride, potassium, taurine, magnesium, medium-chain triglycerides, fiber). Therefore not every cardiac diet is appropriate for every dog with heart disease and recommendations must be individualized. There currently are no commercial veterinary diets designed for cats with cardiac disease in the United States. Nonetheless there are a variety of diets, both veterinary diets marketed for other diseases and over-the-counter reduced sodium diets that may have the desired properties for individual patients with cardiac disease. We usually try to offer the owner several options so that they can determine which one the animal prefers. Having a number of choices is particularly beneficial for animals with CHF in which anorexia, hyporexia, and dysrexia are common.

Avoid common mistakes in nutritional recommendations:

- Be sure to make specific diet recommendations, not just "feed a low-sodium diet" or even "feed any cardiac diet." It is difficult for owners to know which diets are low in sodium (because this is not required information on pet food label), and owners usually do not know what cutoff is specifically meant by low sodium. In addition, as noted above, cardiac diets vary widely in their nutrient levels so they may not all be ideal for an individual patient.
- Avoid the general recommendation of "feed a senior diet." Although senior diets are assumed to be low in sodium, this is not always true. Some senior diets may be appropriate, but it is very important to look at the characteristics of the individual product. There is no legal definition for a senior diet, so the levels of calories, protein, sodium, and other nutrients can vary dramatically among products from different companies. In studies of over-the-counter senior diets for dogs and cats, the sodium content ranged from 33 to 412 mg/100 kcal for dogs (the Association of American Feed Control Officials [AAFCO] sodium minimum for dogs is 20 mg/100 kcal) and 70 to 270 mg/100 kcal for cats (the AAFCO sodium minimum for cats is 50 mg/100 kcal). Therefore recommending "any senior diet" could dramatically increase the sodium intake for a dog or cat with heart disease.

ADDRESS ALL COMPONENTS OF THE DIET

Including all sources of dietary sodium in the overall diet plan is important to achieve success with nutritional modifications.

- Treats. Most dogs and many cats with heart disease receive treats. Most owners are not aware of what foods are high or low in sodium, and so they must be given very specific recommendations for appropriate treats and table food (and appropriate amounts). We typically provide a list of foods that are appropriate and foods to avoid as treats to assist the owner in wise selection. The Tufts HeartSmart website has an up-to-date listing of treats that are low in sodium.
- Foods used for medication administration. Most dog owners and many cat owners (34%) use food for medication administration, and the most commonly used foods are high in sodium (e.g, cheese, hot dogs, lunch meats). Therefore it is important to provide effective but appropriate foods for administering medications (Box 22.1).
- Dietary supplements. Ask at each visit if the owner is administering dietary supplements. If so, ensure that the supplements are safe, are not interacting

Box 22.1 Recommended Options for Medication Administration

- Place medications within low-sodium foods[a]
  - Dogs or cats
    - Meat or fish, home-cooked, without salt (not lunch meats or canned meat/fish)
    - Low-sodium canned pet food
    - Whipped cream (Reddi Wip); check label to ensure it has 0 mg sodium
    - Whipped cream cheese (check label to ensure it is <80 mg/100 kcal; many are much higher)
  - Dogs only
    - Soft fruit (e.g., banana, orange, melon, berries; *avoid grapes*)
    - Whipped vanilla cake frosting (check label to ensure it is <80 mg/100 kcal and that it does not contain artificial sweeteners or xylitol)
    - Mini marshmallows
    - Peanut butter (labeled as "no salt added" or "unsalted")
- Teach the owner to administer the pill to the animal without using foods. This may involve demonstrating the technique with the patient's own medication or an empty capsule.
- Place individual pills within an empty gelatin capsule.
- Teach the owners to use a pet piller or pet pill gun.
- Switch from pills to a compounded, flavored liquid medication from a reputable compounding pharmacy. (Note: The pharmacokinetics may be altered.)

[a]For more diet tips, see the Cummings School's HeartSmart website (https://heartsmart.vet.tufts.edu/).

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with the diet or cardiac medications, and are being administered at an appropriate dose. Animals with cardiac disease are more likely to be receiving dietary supplements than animals in the general pet population as "Internet surfing" for alternative treatments of cardiac disease is common. The clinician and pet owner should understand that dietary supplements can be marketed without proof of safety, efficacy, or quality control. Therefore careful consideration of the type, dose, and brand may be important to avoid toxicities or a complete lack of efficacy. Because there is little governmental regulation over dietary supplements, pet owners should consider selecting dietary supplements that have the Dietary Supplement Verification Program logo; this program tests supplements for ingredients, concentrations, dissolvability, and contaminants. Another resource is Consumerlab, which performs independent testing of health and nutrition products.

- Another important issue related to administration of dietary supplements is that some owners may use them in place of standard cardiac medications. Animals with severe CHF may already be receiving many pills each day, and it may not be clear to owners that it is more important to give the cardiac medications than the dietary supplements. In situations in which pill administration is becoming overwhelming for an owner, clinicians can assist the owner in prioritizing dietary supplements (or encouraging them to discontinue all unnecessary supplements).

COMMUNICATE WITH THE OWNER

- Be sure to understand "non-negotiables" with the owners (e.g., they wish to continue giving treats, they want to feed a home-cooked diet, or they have to give medications in foods). Discuss the nutritional plan and answer questions. Consider referring the owner to a board certified veterinary nutritionist for complicated cases, when multiple diseases are present, when owners have a large number of questions about nutrition, or for those who wish to feed a home-cooked diet.
- Warn owners of cyclical/variable appetite in animals with CHF. Animals with CHF often have periods of anorexia, hyporexia, and dysrexia. Altered appetite often heralds a change in CHF control or a need to change medications, but fluctuations in appetite, eating patterns, and food preferences (i.e., dysrexia) often occur even when CHF is well controlled. Owners often describe this as the animal preferring different foods than it used to, or the animal eating one food well for a week and then not wanting it anymore (but they will eat a different food). It is helpful to warn the owner about appetite and to provide multiple appropriate diet options for the owner to feed. A rotation of low-sodium diets or even rotation of appropriate foods that can be used in moderation mixed into meals (i.e., "toppers") also can be helpful. It is important to advise the owner that if the animal is also acting lethargic, is tachypneic, will not eat *any* foods, or had been eating very well and then suddenly stops eating, this usually indicates the need for reassessment and adjustment of medications.

REASSESS THE PLAN

Monitoring the nutritional plan at each visit is important. This should include the following:

- Nutritional assessment
  - Clinical signs
  - Diet history
  - Body weight
  - BCS
  - MCS
  - Laboratory testing (e.g., electrolytes, blood urea nitrogen, creatinine, albumin)
  - Others, as indicated

NUTRITIONAL RECOMMENDATIONS BASED ON SEVERITY OF DISEASE

Nutritional recommendations will be considered with the severity of heart disease to emphasize the importance of considering different stages for nutrition (Table 22.1).

- Calories. Optimal calorie intake should be a goal for all stages of disease, as previously described. Once an animal has developed CHF, weight loss should be avoided in animals that have a BCS of 6–7/9, as the weight lost will be primarily muscle, rather than fat.
- Protein. Ensuring adequate protein intake is important, as previously described. This should be a primary goal in animals with cardiac disease, particularly as CHF becomes more advanced.
- Amino acids
  - Taurine. Although most current cases of feline and canine DCM appear to be independent of taurine status, taurine deficiency still should be considered whenever the diagnosis of DCM is made. Plasma and whole blood taurine levels should be analyzed, and treatment with taurine (cats: 125–250 mg q12h PO; dogs: 250–1000 mg q12h PO) should begin concurrent with medical therapy. If the animal is eating an unconventional diet that is likely to be unbalanced, the owner also should be counseled to switch to a nutritionally balanced, low-sodium commercial pet food made by a well-established manufacturer containing well-researched ingredients.
- Fat. Some dogs and cats require restriction of fat for other medical conditions (e.g., gastrointestinal

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Table 22.1 Recommendations for Nutrients of Concern in Dogs and Cats Based on Stage of Cardiac Disease[a]

NUTRIENT	ACVIM STAGE A	ACVIM STAGE B	ACVIM STAGE C	ACVIM STAGE D
Calories	Calories sufficient to achieve and maintain BCS of 4–5 (on a 9-point scale). Try to prevent overweight/obesity and, if present, work to achieve successful weight loss.	Calories sufficient to achieve and maintain BCS of 5–6 (on a 9-point scale).	Calories sufficient to achieve and maintain BCS of 5–7 (on a 9-point scale).	
Protein	Minimum dietary protein of 5.1 g/100 kcal for dogs and 6.5 g/100 kcal for cats.		Minimum dietary protein of 6.1 g/100 kcal for dogs and 7.5 g/100 kcal for cats (unless concurrent kidney disease is present). Higher dietary protein may be desirable in animals with significant muscle loss.	
*Fat*Omega-3 fatty acids	No known benefits at this stage.	For pets with arrhythmias, late-stage B2, or if losing weight or muscle mass. Fish oil[b] (total daily dose of 40 mg/kg EPA + 25 mg/kg DHA from diet, supplements, or the combination) if taste is acceptable to the animal or animal can be pilled.	Fish oil[b] (total daily dose of 40 mg/kg EPA + 25 mg/kg DHA from diet, supplements, or the combination) if taste is acceptable to the animal or animal can be pilled.	
Minerals Sodium	<130 mg/100 kcal	<100 mg/100 kcal	<80 mg/100 kcal	
Potassium	No known benefits of supplementation at this stage.	No known benefits of supplementation at this stage unless hypokalemia is present.	*Hypokalemia*: Check dietary K^+ to ensure not a low K^+ diet. Supplement potassium gluconate if diet alone does not correct.[b] *Hyperkalemia*: Lower dietary K^+	
Magnesium	No known benefits of supplementation at this stage.	No known benefits of supplementation at this stage unless hypomagnesemia is present.	Ensure dietary intake if hypomagnesemia is present. Supplement magnesium citrate if hypomagnesemia persists.[b]	
Vitamins B vitamins	No known benefits of supplementation at this stage.	No known benefits of supplementation at this stage.	Consider supplementation of B vitamin complex if on high-dose diuretics.[b]	

ACVIM, American College of Veterinary Internal Medicine; *BCS*, body condition score; *DHA*, docosahexaenoic acid; *EPA*, eicosapentaenoic acid.
Note that all of these nutrients may not be of concern for an individual animal (e.g., if an animal has normal serum potassium concentrations, modification of dietary potassium is not warranted). Conversely if animals have concurrent disease (e.g., gastrointestinal disease, chronic kidney disease, cancer), additional nutrients will likely need to be modified. For some diet options, see the Tufts HeartSmart website or contact individual manufacturers for nutritional information. For complicated cases, consider consulting with a board certified veterinary nutritionist (www.vetspecialists.com or http://www.ebvs.eu/specialists).
[a]*Note:* Home-prepared diets are strongly discouraged unless the recipe is formulated by a board certified veterinary nutritionist (www.vetspecialists.com or https://www.esvcn.eu/college), the owner will follow the recipe exactly, and the animal/recipe will be reevaluated frequently.
[b]Consider quality control issues with all dietary supplements.

disease, pancreatitis), but for most animals, fat restriction is not recommended since most low-fat diets are lower in calories and it can be difficult to meet calorie needs. We currently recommend a daily dose of fish oil to provide 40 mg/kg of EPA and 25 mg/kg of DHA for most animals with CHF if there are no contraindications (e.g., dietary fat intolerance, coagulopathies). Commercial diets vary widely in their EPA and DHA content but most will not contain sufficient levels to meet the above dose and supplementation is necessary to achieve this omega-3 fatty acid dose (although if it is a diet containing moderate levels of EPA and DHA, this should be taken into account when calculating the supplemental dose). When fish oil supplements are used, it is important to know the exact amount of

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EPA and DHA in a specific fish oil brand because supplements vary widely. The most common formulation of fish oil is 1-g capsules that contain approximately 180 mg of EPA and 120 mg of DHA. At this concentration, fish oil can be administered at a dose of 1 capsule per 10 pounds of body weight per day to achieve the recommended EPA and DHA dosages. Fish oil supplements always should contain vitamin E as an antioxidant, but other nutrients should not be included to avoid toxicities. Cod liver oil and flaxseed oil should not be used to provide omega-3 fatty acids to dogs and cats.

- Carbohydrate. Carbohydrate restriction is not needed for most pets with heart disease unless concurrent disease is present (e.g., diabetes mellitus). In one study, reduced starch levels (along with EPA and DHA supplementation) were associated with reductions in some echocardiographic measurements in cats with asymptomatic HCM, but additional research is needed.
- Minerals
  - Sodium. Although sodium restriction is typically recommended for all stages of cardiac disease, the degree of restriction should vary with the stage of disease. We do not recommend severe sodium restriction at any stage of heart disease (Table 22.1), but especially not early in the disease because this can activate the renin-angiotensin-aldosterone system. Instead we recommend introducing the concept of sodium restriction in pets with early disease and using this time to begin to educate owners about nutrition and cardiac disease, particularly with respect to foods that are high in sodium. Most owners are unaware of the sodium content of pet and human foods and need very specific instructions regarding appropriate pet foods, acceptable treats for the animal's stage of disease, and methods for administering medications. Owners also should be counseled on specific high-sodium foods to avoid, such as baby food, bread, canned vegetables (unless labeled "no salt added"), cheeses, peanut butters, condiments (e.g., ketchup, soy sauce), lunch meats and cold cuts (e.g., ham, corned beef, salami, sausages, bacon, hot dogs), most pet treats, rawhides and bully sticks, pickled foods, pizza, processed foods (e.g., potato mixes, rice mixes, macaroni and cheese), snack foods (e.g., potato chips, packaged popcorn, crackers), and soups (unless homemade without salt). As cardiac disease progresses, additional dietary sodium restriction is often recommended.
  - Potassium. Commercial pet foods vary widely in their potassium content, and so, depending on the individual animal's serum potassium concentration, foods may exacerbate hypokalemia or hyperkalemia. Conversely diets can be selected to help manage hypokalemia or hyperkalemia. Serum potassium should be monitored regularly in animals with CHF and addressed with diet, supplementation, or both.
  - Magnesium. Serum magnesium concentrations should be measured in animals with CHF, although serum magnesium concentrations are a poor indicator of total body stores. Nonetheless serial evaluations in an individual patient may be useful, especially in patients with arrhythmias or in those receiving high doses of diuretics. Diets high in magnesium may be beneficial in a hypomagnesemic animal, although supplementation may be needed to help correct hypomagnesemia.
- Other nutrients
  - L-Carnitine. Carnitine deficiency syndromes can be associated with primary myocardial disease in humans and have been reported in a family of Boxers, but no controlled prospective studies on L-carnitine supplementation in dogs with DCM have been reported. Because of its important role in fatty acid metabolism and energy production, carnitine supplementation may have benefits for myocardial energy metabolism, even if a primary deficiency is not present. Diagnosis of L-carnitine deficiency can be challenging because total plasma concentrations are often normal, even in the face of myocardial deficiency, and analysis of carnitine fractions at specialized laboratories is required. Therefore we do not often measure levels clinically and typically recommend L-carnitine supplementation empirically for owners of dogs with CHF who "wish to do everything." The optimal dose of L-carnitine is unknown, but 25–75 mg/kg q8–12h PO has been recommended.
  - Coenzyme Q10. Coenzyme Q10, which plays a role in myocardial energy production and has some antioxidant effects, has been recommended as an adjunct treatment for DCM in dogs. While there is some evidence for benefits in people with DCM, there is limited data supporting benefits in dogs with DCM. Some research in dogs with MMVD has been conducted in recent years and there may be some potential benefits in dogs with MMVD and CHF, but more research is needed. The optimal dose of coenzyme Q10 in dogs is unknown and depends, not only on dog size, but also on the form of coenzyme Q10. In humans, ubiquinone (the oxidized form) is better absorbed with meals while ubiquinol (the reduced form) appears to have greater bioavailability and is the active form. Most studies in dogs have used the ubiquinone form.
  - Antioxidants. Dogs with CHF, especially advanced CHF, have an imbalance between the levels of oxidants produced and the endogenous antioxidant protection available. The major antioxidants include enzymatic antioxidants (e.g.,

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superoxide dismutase, catalase, glutathione peroxidase) and oxidant quenchers (e.g., vitamin C, vitamin E, and beta carotene). Supplemental antioxidants are included, provided in many over-the-counter and veterinary diets; however, the benefits of antioxidant supplementation in dogs and cats with CHF require much additional research.

Frequently Asked Questions

WHAT ARE SOME WAYS TO IMPROVE APPETITE IN A DOG OR CAT WITH CHF THAT HAS CHRONIC REDUCED FOOD INTAKE (HYPOREXIA) OR ALTERED APPETITE PATTERNS AND PREFERENCES (DYSREXIA)?

- For animals with chronic CHF, consider an appetite stimulant (capromorelin for dogs; capromorelin or mirtazapine for cats). Capromorelin can be used daily chronically for animals with dysrexia.*
- Provide multiple diet options for animals with hyporexia or dysrexia.
- Change the temperature of the food. Cats often prefer the food to be warmed to body temperature before feeding, although individual dogs may prefer the food to be warmed, at room temperature, or cold.
- Feed several small frequent meals during the day rather than just one or two meals.
- Feed the animal from a different type of dish (e.g., a new food dish or a human dinner plate).
- Feed in a different location in the house.
- Supplement fish oil, which is high in omega-3 fatty acids, reduces inflammatory cytokines and may have modest benefits for appetite (see later).
- Add toppers to help improve appetite. Small amounts of toppers can be rotated to help manage dysrexia while the main pet food is kept the same. Options are listed below but all should be <80 mg/100 kcal sodium. Cats prefer savory options while dogs are more variable and some prefer savory flavors while other prefer sweet toppers.

Sweet toppers

- Sugar (brown or white)
- Vanilla or fruit yogurt; avoid yogurts with artificial sweeteners
- Honey or maple syrup
- Applesauce
- Fresh fruit; examples include apples, oranges, bananas, melon, or berries (avoid grapes and raisins)
- Low-salt breakfast cereal; the label should read "very low-sodium food" and should have less than 80 mg sodium per 100 calories (most breakfast cereals are high in sodium).

Savory toppers

- Low-sodium canned dog foods (see Tufts HeartSmart website).
- Homemade chicken, beef, or fish broth. Avoid store-bought broths because even the low-sodium brands are too high in sodium or contain onions or garlic.
- Cooked chicken, turkey, or fish; not deli/sandwich meats/cold cuts, rotisserie chicken, and any canned fish or meat. Avoid poultry products that say "enhanced," "marinated," or "solution" on the label since this means salt has been added.
- Fresh vegetables (or frozen/canned brands that are "no salt added"). Examples include carrots, green beans, and tomatoes (avoid onions and garlic).

*Dr. Freeman previously served on scientific advisory boards and as a consultant for Aratana Therapeutics and Elanco, the maker of Entyce and Elura.

IF I HAVE JUST DIAGNOSED CHF IN A DOG AND AM STARTING TO MEDICATE THE DOG, SHOULD I TRY TO ALSO CHANGE HIS DIET BEFORE DISCHARGE TO HELP IMPROVE COMPLIANCE WITH NUTRITIONAL RECOMMENDATIONS?

- We typically do not make major dietary changes while the animal is hospitalized or just getting started on new medications. Most animals with acute CHF will not eat but should begin eating when the CHF is better controlled, and normal hydration is established. While hospitalized, avoid diets that are high in sodium, but do not make major dietary changes during hospitalization. At the time of discharge or at the time of initiating CHF medications, owners should be instructed to avoid high-sodium treats, people foods, and high-sodium pill administration techniques. Once the animal is home, feeling better, and stabilized on medications, a gradual change to a new and more optimal diet can be made. Forced dietary changes when the animal is sick can induce food aversions. Most animals started on diuretics or ACE inhibitors need a recheck examination in 7 to 14 days to check kidney values, electrolytes, and response to treatment; this is an ideal time to reevaluate the nutritional status and to optimize the overall nutritional plan, especially if the animal is feeling better and eating well. It is also important to instruct the owner to notify the veterinarian if the patient does not eat adequate amounts of the new recommended foods so that other options can be devised.

WHAT IS THE CAUSE OF THE DIET-ASSOCIATED DILATED CARDIOMYOPATHY BEING IDENTIFIED IN PETS EATING HIGH-PULSE DIETS?

- In 2018 the FDA released an initial alert on its investigation of a potential association between diet and DCM. The associated diets were often labeled as "grain free" but what appears to be more important is that these diets contain pulses (i.e., peas, lentils, chickpeas, dry beans). Diet-associated DCM differs from typical primary (genetic) that occurs in certain breeds, such as Doberman Pinschers and Great Danes, in that diet-associated DCM can occur in any breed (including breeds predisposed to primary DCM) and they can have improvements in cardiac function and longer survival time after a diet change. Taurine deficiency is an uncommon finding in these dogs with diet-associated DCM (except one study of Golden Retrievers).
- High-pulse diets are not only associated with DCM but also appear to cause earlier, subclinical increases in cardiac size, systolic dysfunction, higher cardiac troponin I, and higher likelihood of ventricular arrhythmias. And while the majority of diet-associated DCM cases reported to the FDA have been dogs, cats eating high-pulse diets may also be at risk for this disease.
- The specific cause and mechanism of this current diet-associated DCM problem is not yet known but

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it appears to be specific to diets that include pulses, which can be in both grain-free and grain-inclusive diets. Clinicians should review the diet's full ingredient list; if the diet contains pulses in the top 10 ingredients (or multiple pulses anywhere on the list), some pets might be risk for DCM. The role for potatoes and sweet potatoes in diet-associated DCM remains unclear. If a dog or cat is diagnosed with DCM or earlier changes (e.g., left ventricular enlargement, reduced systolic function, unexplained arrhythmia) and is eating a high-pulse diet, we recommend changing the diet to one without pulses, potatoes, or sweet potatoes that has an appropriate nutritional profile for the individual patient. In addition to diet change, cardiac medications, such as pimobendan and antiarrhythmic drugs, are often indicated.

- Even though taurine deficiency in dogs with diet-associated DCM is uncommon, until more is known, the authors recommend measuring plasma and whole blood taurine concentrations (or supplementing taurine if cost of testing is prohibitive).
- It can take months to years for the heart to improve and most dogs' hearts do not reverse completely unless the disease is detected early. Therefore it is important not to taper or discontinue cardiac medications too quickly. In most cases, we continue pimobendan and antiarrhythmic drugs on an indefinite basis, as sudden death is still possible even after the heart starts to improve.
- Sudden death is common, so careful monitoring and treating arrhythmias aggressively is important.
- Even diets that do not contain pulses can cause a secondary form of DCM due to nutritional deficiencies if they are not nutritionally balanced (e.g., home-prepared diets, diets designed for intermittent/supplemental use). This underscores the importance of getting a complete diet history for every patient at every visit.

SUGGESTED READINGS/RESOURCES

American College of Veterinary Internal Medicine: Online directory of Board-certified veterinary specialists in the six specialties of the American College of Veterinary Internal Medicine (ACVIM), including Nutrition. Retrieved from www.vetspecialists.com.

Consumerlab. Retrieved from www.consumerlab.com.

Cummings School of Veterinary Medicine at Tufts University: Heart Smart website, including low sodium diet and treat lists. Retrieved from https://heartsmart.vet.tufts.edu/.

Cummings School of Veterinary Medicine at Tufts University: Petfoodology website, including posts on giving pills, common pet nutrition myths, and other nutrition-related topics. Retrieved from www.petfoodology.org.

European College of Veterinary and Comparative Nutrition: Online directory of board-certified veterinary specialists in nutrition and other specialties. Retrieved from https://www.ebvs.eu/specialists.

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Appendix 1

Canine and Feline Breed Predilections for Heart Disease

Kathleen E. Cavanagh and Francis W.K. Smith Jr.

CANINE BREED	DISEASE
Affenpinscher	PDA (AU)
Afghan Hound	DCM
Airedale	PS
Akita	VSD
Basset Hound	PS VSD
Beagle	PS VSD Bundle branch block (AU) Tetralogy of Fallot (AU)
Bearded Collie	SAS (AU)
Bichon Frisé	PDA Degenerative valve disease
Bloodhound	SAS
Border Terrier	Aortic body tumors (AU) VSD (AU)
Boston Terrier	Degenerative valve disease DCM Chemodectoma (± pericardial effusion) PDA (AU)
Bouvier des Flandres	SAS
Boxer	SAS PS ASD DCM Arrhythmogenic right ventricular cardiomyopathy (Boxer cardiomyopathy) Chemodectoma (± pericardial effusion) Endocardial fibroelastosis (AU) HCM (Cambridge)
Boykin Spaniel	PS Degenerative valve disease
Brittany Spaniel	Persistent right aortic arch
British Bulldog	Arteriovenous fistula (AU) Mitral valve disease (AU) PS (AU)
Bullmastiff	PS DCM
Bull Terrier	Mitral valve dysplasia Mitral valve stenosis SAS

CANINE BREED	DISEASE
Cavalier King Charles Spaniel	Inherited ventricular arrhythmias Right atrial hemangiosarcoma (± pericardial effusion) PDA Degenerative (myxomatous mitral) valve disease Femoral artery occlusion (AU)
Chihuahua	PDA PS Degenerative valve disease
Chow Chow	PS VSD
Cocker Spaniel (American, English)	PDA (American, English) PS Degenerative valve disease DCM (American, English) Sick sinus syndrome (American, English)
Cocker Spaniel, American	Cardiomyopathy (AU) PDA (AU)
Collie (rough and smooth)	PDA
Dachshund	Degenerative valve disease Mitral valve prolapse Sick sinus syndrome PDA
Dalmatian	DCM Mitral valve dysplasia
Doberman Pinscher	ASD DCM Bundle of His degeneration Persistent right aortic arch (AU)
Dogue de Bordeaux	SAS
English Bulldog (Bulldog)	PS Tetralogy of Fallot VSD SAS Chemodectoma (± pericardial effusion) Mitral valve dysplasia Persistent right aortic arch
English Sheepdog	DCM
English Springer Spaniel	PDA VSD Persistent atrial standstill

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CANINE BREED	DISEASE
Estrela Mountain Dog	DCM (Cambridge)
Fox Terrier	Degenerative valve disease PS (wirehaired and smooth) Tetralogy of Fallot (wirehaired) Persistent right aortic arch (AU) (wirehaired and smooth)
French Bulldog	PS (Cambridge)
German Pinscher	Persistent right aortic arch
German Shepherd	SAS Mitral valve dysplasia Tricuspid valve dysplasia Persistent right aortic arch Inherited ventricular arrhythmia (tachycardia) (AU) (Cambridge Veterinary School Database) is Hereditary Ventricular Tachycardia Right atrial hemangiosarcoma (± pericardial effusion)
	Infective endocarditis DCM PDA Cardiomyopathy (AU)
German Shorthair Pointer	SAS
Golden Retriever	SAS Mitral valve dysplasia Tricuspid valve dysplasia Taurine deficient familial DCM Canine X-linked muscular dystrophy Pericardial effusion, idiopathic Right atrial hemangiosarcoma (± pericardial effusion)
Great Dane	Mitral valve dysplasia Tricuspid valve dysplasia SAS PS Persistent right aortic arch DCM Lone atrial fibrillation
Great Pyrenees	Tricuspid valve dysplasia
Greyhound	Persistent right aortic arch
Husky	VSD
Irish Setter	Persistent right aortic arch DCM Right atrial hemangiosarcoma (± pericardial effusion) Tricuspid dysplasia (Cambridge)
Irish Wolfhound	DCM Lone atrial fibrillation
Italian Greyhound	Persistent right aortic arch (AU)
Keeshond (Keeshonden)	Conotruncal defects (CTD) include conal septum, conal VSD, tetralogy of Fallot, and persistent truncus arteriosus PDA PS Mitral valve dysplasia
Kerry Blue Terrier	PDA
Labrador Retriever	Tricuspid valve dysplasia PDA PS DCM Pericardial effusion, idiopathic Right atrial hemangiosarcoma (± pericardial effusion) Supraventricular tachycardia (Cambridge)
Lakeland Terrier	VSD
Lhasa Apso	Degenerative valve disease
Maltese	PDA Mitral dysplasia (Cambridge)
Mastiff	Mitral valve dysplasia PS Tricuspid valve dysplasia (Cambridge)
Miniature Pinscher	Degenerative valve disease
Newfoundland	SAS Mitral valve dysplasia Mitral valve stenosis PDA PS DCM ASD VSD (AU)
New Zealand Huntaway Dog	DCM (Cambridge)
Norfolk Terrier	Mitral valve disease (AU) Syncope (AU)
Old English Sheepdog	Tricuspid valve dysplasia Persistent atrial standstill DCM
Pekingese	Degenerative valve disease
Pembroke Welsh Corgi	PDA (Cambridge)
Poodle	PDA (toy and miniature) Degenerative mitral valve disease (toy and miniature) VSD (toy and miniature) (AU) ASD (standard)
Pomeranian	PDA Degenerative valve disease Sick sinus syndrome
Portuguese Water Dog	Juvenile DCM
Pug	Atrioventricular block (SYN: stenosis of the bundle of His as per Cambridge)
Rottweiler	SAS DCM HCM (Cambridge) Mitral dysplasia (Cambridge)
Saint Bernard	DCM
Saluki	PDA (Cambridge)
Samoyed	PS SAS ASD

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CANINE BREED	DISEASE
Schnauzer, Miniature	PS PDA Degenerative valve disease Sick sinus syndrome
Schnauzer, Standard	PS (AU, Cambridge)
Scottish Deerhound	DCM
Scottish Terrier	PS
Shetland Sheepdog	PDA Degenerative valve disease Conotruncal heart malformation (AU)
Shih Tzu	VSD Degenerative valve disease
Springer Spaniel	DCM
Sussex Spaniel	Cardiomyopathy (AU)
Terriers (e.g., Fox Terrier, Mixed Terriers)	PS Degenerative valve disease
Weimaraner	Tricuspid valve dysplasia Peritoneopericardial diaphragmatic hernia
Welsh Corgi (Pembroke)	PDA (AU)
West Highland White Terrier	PS VSD Tetralogy of Fallot Degenerative valve disease Sick sinus syndrome (Cambridge)
Whippet	Degenerative valve disease
Yorkshire Terrier	PDA Degenerative valve disease

ASD, Atrial septal defect; *AU*, author; *DCM*, dilated cardiomyopathy; *HCM*, hypertrophic cardiomyopathy; *PDA*, patent ductus arteriosus; *PS*, pulmonic stenosis; *SAS*, subaortic stenosis; *VSD*, ventricular septal defect.

Feline

BREED	DISEASE
Abyssinian	Subvalvular pulmonary stenosis
Birman	Systemic hypertension
British Shorthair	Hypertrophic cardiomyopathy Septal defect
Burmese	Septal defect Congenital heart defect
Chartreux	Septal defect Systemic hypertension
Devon Rex	Subvalvular pulmonary stenosis
Maine Coon	Hypertrophic cardiomyopathy Septal defect
Norwegian Forest	Hypertrophic cardiomyopathy
Persian	Systemic hypertension Septal defect
Ragdoll	Hypertrophic cardiomyopathy
Siamese	Systemic hypertension Septal defect Congenital heart defect Mitral valve stenosis PDA Subaortic stenosis Supravalvular aortic stenosis Tetralogy of Fallot Tricuspid stenosis
Siberian	Hypertrophic cardiomyopathy
Sphynx	HCM MVD

HCM, Hypertrophic cardiomyopathy; *MVD*, mitral valve disease; *PDA*, patent ductus arteriosus.

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Appendix 2

Cardiopulmonary Drug Formulary

Francis W.K. Smith Jr., Larry P. Tilley, Mark A. Oyama, and Meg M. Sleeper

DISCLOSURE

Advancing technology has provided clinicians with ever more powerful and effective drugs for treating diseases. As more drugs become available, it becomes progressively more difficult for practitioners to make rational choices between similar drugs. It is also difficult to be aware of the numerous side effects, contraindications, and drug interactions of the many cardiopulmonary drugs available. The following tables and charts have been designed and provided in the hope of facilitating rational drug selections for the treatment of cardiopulmonary disease.

An attempt has been made to make these tables as complete as possible, while focusing on the more common or serious side effects and drug interactions. For a more exhaustive review of individual drugs the reader should refer to the package insert and chapters in this book. The reader should also follow the current veterinary literature because new dosing recommendations may become available as a result of clinical use and scientific research. This advice is especially appropriate for new or infrequently used drugs.

Medicine is a science that is constantly changing. Changes in treatment and drug therapy are required with new research and clinical experiences. The author, editor, and publisher of this book have made every effort to ensure that the drug dosage schedules are accurate. The drug dosages are based on the standards accepted at the time of publication. The product information sheet included in the package of each drug should be checked before the drug is administered to be certain that changes have not been made in the recommended dose of or in the contraindications for administration. Primary responsibility for decisions regarding the treatment of patients remains with the attending clinician. All patients should be carefully monitored for desired efficacious and undesired toxic effects while instituting, titrating, and maintaining therapy.

Drugs are listed in alphabetical order by generic name. The order of presentation in no way reflects the preference for use. General recommendations for therapy may be found in the main body of this book.

KEY TO FORMULARY ABBREVIATIONS

ACE	angiotensin-converting enzyme
ACT	activated clotting time
ADP	adenosine diphosphate
APTT	activated partial thromboplastin time
BP	blood pressure
Cap	capsule
CHF	congestive heart failure
COPD	chronic obstructive pulmonary disease
CRI	continuous rate infusion
CPR	cardiopulmonary resuscitation
DCM	dilated cardiomyopathy
ECG	electrocardiogram
G	gram
GFR	glomerular filtration rate
GI	gastrointestinal
HCM	hypertrophic cardiomyopathy
IM	intramuscular
Inj	injectable
IO	intraosseous
IT	intratracheal
IV	intravenous
LRS	lactated Ringer solution
µg	micrograms
mg	milligram
mL	milliliter
PO	per os (oral)
PT	prothrombin time
q	every, as in q8h = every 8 hours
SC	subcutaneous
U	units

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Cardiopulmonary Drugs—Formulations, Indications, and Dosages

DRUG TRADE NAME	FORMULATION	INDICATIONS	DOG DOSE (D) CAT DOSE (C)	COMMENTS
Acetylsalicylic acid Aspirin	Tab: 81, 325 mg	Prevention of thromboembolism	D: 5–10 mg/kg PO q24–48h C: 25 mg/kg PO q3 days (81 mg/cat PO q48–72h)	Nonsteroidal antiinflammatory agent Platelet inhibitor Not effective in all animals May decrease efficacy of ACE inhibitors
Albuterol (Proventil, Ventolin, AccuNeb, Vospire, ProAir, generic)	Tab: 2, 4, 8 mg Syrup: 2 mg/5 mL Inhalation aerosol: 90 µg per actuation Nebulizing solution: 0.63, 1.25, and 2.5 mg/3 mL and 5 mg/mL	Bronchodilation in patients with reversible obstructive lung disease and asthma Collapsed trachea	D, C: 20–50 µg/kg PO q8h Emergency: 90 µg by metered dose inhalation every 30 minutes up to 4–6 hours	Beta-2 agonist bronchodilator Reduce dose by 50% first 4 days to prevent anxiety side effect Decrease dose with renal disease Feline aerosol chamber (www.aerokat.com) can facilitate administration
Amiodarone (Cordarone, generic)	Tab: 200 mg Inj: 50 mg/mL	Indicated for severe refractory atrial and ventricular arrhythmias	D: Loading dose of 10 mg/kg PO q12h for 1 week; thereafter 5 mg/kg PO q12–24h C: None	Class III antiarrhythmic with some class I, II, and IV effects Use as last resort for recurrent unstable ventricular tachycardia; takes weeks to achieve therapeutic levels Side effects include anorexia, liver, thyroid, and pulmonary dysfunction
Amlodipine besylate (Norvasc, generic)	Tab: 2.5, 5, 10 mg	Systemic hypertension, refractory heart failure	D: 2.5 mg/dog or 0.1 mg/kg PO q24h C: 0.18 mg/kg PO q24h (0.625–1.25 mg/cat q24h). Can increase to q12h in refractory cases	Dihydropyridine calcium channel blocker Arterial vasodilator; monitor for hypotension Gingival hyperplasia in small percentage of dogs Generally first choice for systemic hypertension in cats
Atenolol (Tenormin, generic)	Tab: 25, 50, 100 mg Oral suspension: 25 mg/mL Inj: 0.5 mg/mL	Atrial and ventricular arrhythmias, hypertrophic cardiomyopathy (outflow obstruction), systemic hypertension, aortic stenosis, cardiac manifestations of hyperthyroidism	D: 0.25–2.0 mg/kg PO q12–24h C: 6.25–12.5 mg PO q12–24h	Beta-1 selective beta-blocker Less bronchoconstriction and vasoconstriction than nonselective beta-blockers Taper dose when discontinuing therapy Avoid in CHF; can be cautiously used once congestion controlled
Atropine sulfate (generic)	Inj: 0.05, 0.1, 0.3, 0.4, 0.5, 0.8, 1.0 mg/mL	Sinus bradycardia, AV block, sick sinus syndrome, cardiac arrest	D: 0.01–0.04 mg/kg IV, IM, IO 0.02–0.04 mg/kg SC q6–8h (IT: double dose) C: Same	Anticholinergic May transiently worsen bradyarrhythmia Preoperatively, use with caution and only with bradycardia in cardiac cases

(*Continued*)

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Cardiopulmonary Drugs—Formulations, Indications, and Dosages—cont'd

DRUG TRADE NAME	FORMULATION	INDICATIONS	DOG DOSE (D) CAT DOSE (C)	COMMENTS
Benazepril (Fortekor, Lotensin, generic)	Tab: 5, 10, 20, 40 mg	Balanced vasodilation in CHF, systemic hypertension Approved for feline renal disease in other countries	D: 0.25–0.5 mg/kg PO q24h C: 0.25–0.5 mg/kg PO q24h (2.5 mg/cat per day)	Angiotensin-converting enzyme inhibitor Monitor electrolytes and renal function Excreted in bile and urine
Buprenorphine (Buprenex)	Inj: 0.324 mg/mL	Pain control following aortic thromboembolism	C: 0.005–0.1 mg/kg IV, IM	Opioid agonist
Butorphanol tartrate (Torbutrol, Stadol, generic)	Inj: 0.5, 1, 2 mg/mL Tab: 1, 5, 10 mg (Torbutrol)	Nonproductive cough (COPD, tracheal collapse, left atrial compression of left mainstem bronchus)	D: 0.055–0.11 mg/kg SC q6–12h; 0.55 mg/kg PO q6–12h C: None	Narcotic cough suppressant More potent than dextromethorphan
Calcium gluconate	Inj: 100 mg/mL (10%), 230 mg/mL (23%)	Acute management of severe cardiac manifestations of hyperkalemia	D, C: 50–20 mg/kg slow IV (5–15 minutes)	Normalizes ionic gradients in myocytes without altering potassium levels Monitor ECG while administering; discontinue if QT shortening, VPCs, or sudden slowing of the heart rate occur
Carnitine (Carnitor, generic)	Tab: 330 mg Can be purchased in bulk as a powder	Canine dilated cardiomyopathy accompanied by carnitine deficiency	D: 50–100 mg/kg PO q8h C: None	Amino acid L isomer is active form Not effective in all cases
Carvedilol (Coreg, generic)	Tab: 3.125, 6.25, 12.5, 25 mg Cap (extended release): 10, 20, 40, 80 mg. Pharmacokinetics of extended-release caps has not been evaluated in dogs	Myocardial failure	D: 0.2–0.4 mg/kg q12h (based on pharmacokinetic studies may be able to titrate to 1.5 mg/kg PO q12h if tolerated) Start slowly at 0.05–0.1 mg/kg PO q12h for 2 weeks; up-titrate the dose every 2–4 weeks C: Not established	Alpha and nonselective beta-blocker Do not use with AV block Monitor closely for worsening of heart failure Absorption highly variable in dogs
Chlorothiazide (Diuril, generic)	Tab: 250, 500 mg Oral suspension: 50 mg/mL	Diseases associated with fluid retention (CHF, hepatic disease, nephrotic syndrome), end-stage heart failure (ascites)	D: 20–40 mg/kg PO q12h C: Same	Thiazide diuretic Less potent than loop diuretics Not effective with low GFR (renal failure) Can use with loop diuretics for increased diuresis, but reduce initial thiazide dose by 50% May precipitate hepatic encephalopathy in patients with severe liver disease

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Clopidogrel (Plavix, generic)	Tab: 75, 300 mg	Prevention of aortic thromboembolism in cats that have already had a thromboembolic event or are at high risk for thromboembolism	D: 0.5–1 mg/kg PO q24h Loading dose of 2–4 mg/kg may be followed with 1 mg/kg q24h C: 18.75 mg PO q24h; loading dose of 37.5 mg immediately following embolism	Platelet inhibitor (ADP receptor blocker) More effective than aspirin in preventing recurrence of aortic thromboembolism Can be used in combination with aspirin or heparin; be aware of possibility of hemorrhage when using combinations Low risk of hemorrhage as sole agent May cause ptyalism because of distaste
Dalteparin (Fragmin)	Inj: 16 mg (2500 U)/0.2 mL; 32 mg (5000 U)/0.02 mL prefilled syringes; 64 mg (10,000 U)/mL multidose vials	Antithrombotic	D: 100–150 U/kg SC q8h C: 180 U/kg SC q6h	Low molecular weight heparin Dose extrapolated from humans and pharmacokinetic studies in animals; optimal dose in dogs and cats is unknown Less likely to cause bleeding complications than warfarin Expensive
Digoxin (Lanoxin, generic)	Tab: 0.0625, 0.125, 0.1875, 0.25 mg Elixir: 0.05 mg/mL	Supraventricular arrhythmias (especially atrial fibrillation), myocardial failure	D: *Maintenance dose*: 0.003–0.005 mg/kg PO q12h. Max. dose for Dobermans 0.25 mg q12h *Oral loading dose*: Twice the maintenance dose for the first 24–48 hours C: 0.01 mg/kg PO q48h (tab preferred)0.007 mg/kg PO q48h (w/furosemide and aspirin); rarely used	Digitalis glycoside Toxicity potentiated by hypokalemia, hyponatremia, hypercalcemia, hypothyroidism, hypoxia Dose on lean body weight, reduce dose 10%–15% with elixirsTherapeutic range 0.5–1 ng/mL 8 hours after a dose Often used in combination with diltiazem or a beta-blocker for rate control of atrial fibrillation
Diltiazem (Cardizem, Dilacor, generic)	Tab: 30, 60, 90, 120 mg Inj: 0.45, 0.83, 1.0 mg/mL Cardizem CD: 120, 180, 240, 300, 360 mg Dilacor XR: 120, 180, 240 mg	Supraventricular arrhythmias, hypertrophic cardiomyopathy, systemic hypertension	D: 0.5–2 mg/kg PO q8h (consider higher dose of 5 mg/kg based on recent studies) 0.1–0.2 mg/kg IV bolus, then 2–6 µg/kg per minute IV CRI Dilacor XR: 3–4 mg/kg PO q24h C: 1.0–2.5 mg/kg PO q8h 0.1–0.2 mg/kg IV bolus, then 2–6 µg/kg per minute IV CRI Dilacor XR: 30–60 mg PO q24h For Atrial fibrillation, to start at 3 mg/kg Dilacor XR and titrate up based on ventricular response rate up to 5 mg/kg	Calcium channel blocker Less myocardial depression than verapamil Dilacor XR capsules contain 60-mg tablets that are used for dosing cats Extended-release formulations have unpredictable bioavailability and are often associated with anorexia, vomiting, weight loss, lethargy, and hepatopathy in cats Not as effective as beta-blockers for treatment of left ventricular outflow tract obstruction

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Cardiopulmonary Drugs—Formulations, Indications, and Dosages—cont'd

DRUG TRADE NAME	FORMULATION	INDICATIONS	DOG DOSE (D) CAT DOSE (C)	COMMENTS
Dobutamine HCl (generic)	Inj: 12.5 mg/mL	Short-term management of severe myocardial failure	D: 2.5–20 μg/kg per minute (titrate up to effect); administer in D5W C: 2–10 μg/kg per minute (titrate up to effect); administer in D5W	Beta-adrenergic agonist Monitor ECG, BP, and pulse quality Preferable to dopamine in CHF but more expensive Inotropic effect is dose dependent Less arrhythmogenic than most other catecholamines Use with caution in cats; may cause seizures
Dopamine HCl (generic)	Inj: 40, 80, or 160 mg/mL	Shock, short-term management of severe myocardial failure Anuric or oliguric renal failure	D: 2–10 μg/kg per minute (titrate up to effect); administer in D5W, saline, or LRS C: Same	Dopaminergic agonistMonitor ECG, blood pressure, and pulse quality; adjust dose based on response May cause tissue necrosis if extravasation occurs Can administer intraosseously
Doxycycline (Vibramycin and generic)	Tab: 25, 75, 100, 150 mg Cap: 40, 50, 75, 100, 150 mg Oral suspension: 10 mg/mL Inj: 100, 200 mg vial	Adjunctive treatment for heartworm disease	D: Heartworm treatment: 10 mg/kg PO q12h for 4 weeks before administering melarsomine	Tetracycline antibiotic effective against *Wolbachia* coinfection in heartworm disease Effect against *Wolbachia* enhanced by macrocyclic lactones Reduces disease associated with dead heartworms Disrupts heartworm transmission Enhances adulticide activity of macrocyclic lactones
Enalapril maleate (Enacard, Vasotec, generic)	Tab: 1, 2.5, 5, 10, 20 mg	Balanced vasodilation in CHF, systemic hypertension	D: 0.5 mg/kg PO q12–24h (titrate up to effect) C: 0.25–0.5 mg/kg PO q24–48h (titrate up to effect)	Angiotensin-converting enzyme inhibitor Monitor renal function and electrolytes Increased survival in heart failure patients Decrease dose with renal disease
Enoxaparin (Lovenox, generic)	Inj: 30 mg/0.3 mL	Antithrombotic	D: 0.8 mg/kg SC q12h C: 1.25 mg/kg SC q12h	Low molecular weight heparin Dose extrapolated from humans and pharmacokinetic studies in animals; optimal dose in dogs and cats is unknown Less likely to cause bleeding complications than warfarin

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Epinephrine (Adrenalin, generic)	Inj: 1:1000 conc (1 mg/mL) 1:10,000 conc (0.1 mg/mL)	Cardiac arrest	D: 0.1–0.2 mg/kg IV, IO q3–5 min Double dose for IT administration C: Same	Beta-adrenergic agonist Previously recommended dose of 0.01–0.02 mg/kg may be a safer starting dose if a defibrillator is not available Vasopressin may be more effective
Esmolol (Brevibloc, generic)	Inj: 10 mg/mL	Short-term management of supraventricular tachyarrhythmias, ventricular tachycardia, and systemic hypertension	D, C: 50–100 µg/kg IV bolus every 5 minutes (up to 500 µg/kg max) 25–200 µg/kg per minute CRI	Ultra-short-acting beta-1 selective beta-adrenergic blocker
Fluticasone (Flovent)	Metered dose inhaler: 44, 110, 220 µg/actuation	Feline asthma, chronic bronchitis	D: <20 kg, 110 µg via metered dose (activation) q12h; >20 kg, 220 µg via metered dose q12C: C: 110–220 µg via metered dose (activation) q12h (higher dose reserved for severe disease); or 220 µg via metered dose q24	Corticosteroid Feline aerosol chamber (www.aerokat.com) can facilitate administration
Furosemide (Lasix, generic)	Tab: 12.5, 20, 40, 50, 80 mg Inj: 10 and 50 mg/mL Oral solution: 10 mg/mL	Diseases associated with fluid retention (CHF, hepatic disease, nephrotic syndrome), systemic hypertension	D: 2–6 mg/kg PO, IM, IV q8–48h 2–8 mg/kg IV q1–2h for severe pulmonary edema CRI: 0.66 mg/kg IV bolus followed by 0.66 mg/kg/h IV C: 1–4 mg/kg PO, IM, IV q12–48h Titrate to lowest effective dose for maintenance	Loop diuretic Decreased oral absorption in decompensated CHF Monitor hydration and electrolytes; hypokalemia uncommon in dogs unless anorexic or high dose Bioavailability reduced with food Cats usually dislike taste of oral solution Can use in combination with other classes of diuretics in refractory CHF
Glycopyrrolate (Robinul, Cuvposa, Glycate, generic)	Inj: 0.2 mg/mL	Sinus bradycardia, AV block, sick sinus syndrome	D: 0.005–0.01 mg/kg IV, IM, 0.01–0.02 mg/kg SC C: Same	Anticholinergic Longer duration of action Preoperatively, use with caution and only with bradycardia in cardiac cases

(*Continued*)

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Cardiopulmonary Drugs—Formulations, Indications, and Dosages—cont'd

DRUG TRADE NAME	FORMULATION	INDICATIONS	DOG DOSE (D) CAT DOSE (C)	COMMENTS
Heparin (Calciparine, Liquaemin, generic)	Inj: 1000, 5000, 10,000 U/mL	Short-term prevention of thromboembolism	D: Loading dose: 100–500 U/kg SC q8h Chronic dose: 10–50 U/kg q6–8h C: Loading dose: 100–300 U/kg SC q8h Chronic dose: 10–50 U/kg q6–8h	Anticoagulant Antidote: Protamine sulfate Maintain APTT or ACT at 2–2.5 times the pretreatment values
Hydralazine HCl (Apresoline, generic)	Tab: 10, 25, 50, 100 mg Inj: 20 mg/mL	Arterial dilation in CHF, systemic hypertension	D: 1–3 mg/kg PO q12h (titrate up to effect) C: 0.5–0.8 mg/kg PO q12h	Direct-acting arterial vasodilator Causes sodium retention, requiring increased diuretic doses Reflex tachycardia can be controlled with digitalis Decreasing dose 50%–75% for 1–2 weeks and then titrating upward may reduce risk of vomiting Can use injectable formulation orally for more accurate dosing in small patients Use largely replaced by amlodipine because of superior efficacy and tolerability
Hydrochlorothiazide (Microzide, generic)	Tab: 12.5, 25, 50, 100 mg Cap: 12.5	Diseases associated with fluid retention (CHF, hepatic disease, nephrotic syndrome), systemic hypertension, end-stage heart failure (ascites)	D: 2–4 mg/kg PO q12h C: Same	Thiazide diuretic Less potent than loop diuretics Not effective with low GFR (renal failure) Can use with loop diuretics for increased diuresis, but reduce initial thiazide dose by 50%
Hydrocodone bitartrate (Tussigon, generic)	Tab: 5 mg Syrup: 1 mg/mL	Nonproductive cough (COPD, tracheal collapse, left atrial compression of the mainstem bronchus)	D: 0.22 mg/kg PO q6–12h C: Do not use	Narcotic antagonist More potent than dextromethorphan
Hyoscyamine (Anaspaz, Cystospaz, Donnamar, Levsin, generic)	Tab: 0.125 mg	Rate control in sick sinus syndrome and AV block	D: 0.003–0.006 mg/kg PO q8h	Anticholinergic

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Imidapril (Prilium [Europe, Canada])	Oral liquid: 5, 10 mg/mL	Balanced vasodilation in CHF, systemic hypertension	D: 0.25–0.5 mg/kg q24h C: 0.5 mg/kg PO q24h	Angiotensin-converting enzyme inhibitor Monitor renal function and electrolytes Decrease dose with renal disease
Isoproterenol (Isuprel, generic)	Inj: 1:5000 (0.2 mg/mL)	Short-term management of severe bradycardia unresponsive to atropine (sinus bradycardia, AV block, sick sinus syndrome)	D: 0.04–0.09 µg/kg per minute IV (titrate up to effect) 10 µg/kg per minute IM, SC q6h C: Same	Sympathomimetic agent Used as an emergency treatment until artificial pacing can be accomplished
Isosorbide dinitrate (Isordil Titradose, Isochron, generic)	Tab: 5, 10, 20, 30, 40 mg	End-stage CHF	D: 0.2–1.0 mg/kg PO q12h C: Same	Nitrate venodilator Can combine with hydralazine for balanced vasodilation Schedule 12-hour drug-free period to try and avoid tolerance Efficacy uncertain
Lidocaine (Xylocaine, generic)	Inj: 5, 10, 15, 20 mg/mL (without epinephrine)	Ventricular arrhythmias	D: 2 mg/kg slowly IV or IO (double the dose IT) in 2 mg/kg boluses followed by IV drip at 30–80 (occasionally up to 100) µg/kg per minute CRI C: 0.25–0.75 mg/kg IV over 5 minutes	Class I antiarrhythmic Use with caution in cats as may cause seizures Drug of choice for initial control of ventricular tachycardia Effects increased by high potassium and decreased by low potassium
Lisinopril (Prinivil, Zestril, generic)	Tab: 2.5, 5, 10, 20, 30, 40 mg	Balanced vasodilation in CHF, systemic hypertension	D: 0.25–0.5 mg/kg PO q12–24h	Angiotensin-converting enzyme inhibitor Monitor renal function and electrolytes Decrease dose with renal disease
Magnesium (generic)	20% $MgCl_2$ solution for injection (contains 1.97 mEq of Mg++ per mL)	Refractory ventricular arrhythmias; torsade de pointes	D: 0.75–1 mEq/kg/24 h IV infusion (50% of total dose can be given in 2–4 hours if necessary); for ventricular fibrillation: 0.15–0.30 mEq/kg IV over 5–10 minutes	Electrolyte Hypomagnesemia may potentiate ventricular tachycardia

(*Continued*)

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Cardiopulmonary Drugs—Formulations, Indications, and Dosages—cont'd

DRUG TRADE NAME	FORMULATION	INDICATIONS	DOG DOSE (D) CAT DOSE (C)	COMMENTS
Melarsomine (Immiticide)	Inj: 25 mg/mL	Heartworm disease	D: 2.5 mg/kg IM give a single injection of 2.5 mg/kg and then in one month give two additional doses 24 hours apart C: None	Heartworm adulticide Administer via deep IM injection Many heartworm experts recommend split dose (as for class III patients) also for class I and II Doxycycline may be administered for 4 weeks before melarsomine treatment to reduce pulmonary reaction
Methoxamine (Vasoxyl)	Inj: 20 mg/mL injection	Used primarily in critical care patients or during anesthesia to increase peripheral resistance and increase blood pressure	D, C: 200–250 µg/kg IM, or 40–80 µg/kg IV; repeat dose as needed	Sympathomimetic. Alpha-1 adrenergic agonist Short onset and duration of action Adverse effects related to excessive stimulation of alpha-1 receptor (prolonged peripheral vasoconstriction) Reflex bradycardia may occur
Metoprolol (Lopressor, generic)	Tab: 25, 50, 100 mg Inj: 1 mg/mL	Atrial and ventricular arrhythmias, hypertrophic cardiomyopathy, systemic hypertension, myocardial failure	D: 0.25–1.0 mg/kg PO q8h C: Same	Beta-1 selective beta-blocker Less bronchoconstriction and interference with insulin therapy than with nonselective beta-blockers Taper dose when stopping Dose should be slowly titrated over several weeks in dogs with myocardial failure
Mexiletine (generic)	Cap: 150, 200, 250 mg	Ventricular arrhythmias	D: 4–8 mg/kg PO q8–12h C: None	Class I antiarrhythmic Reduce dose with liver disease Take with food to reduce GI side effects
Nitroglycerin (Nitro-BID, Nitroquick, Nitrostat, Nitrolingual, Nitro-Dur, Minitran, generic)	2% ointment (1 inch = 15 mg) Transdermal patch: 0.1, 0.2, 0.3, 0.4, 0.6, 0.8 mg/h	Usually used for short-term management of acute CHF End-stage CHF	D: 0.25 inch/5 kg cutaneously q6–8h; Patch: 2.5–10 mg (small-giant dog) C: 1/8–1/4 inch cutaneously q6–8h	Nitrate venodilator Can combine with hydralazine for balanced vasodilation Apply to ears if warm to touch, otherwise use shaved area in inguinal or axillary region (use gloves when applying) Schedule 12-hour drug-free period to try and avoid tolerance Efficacy uncertain

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Nitroprusside sodium (Nitropress, generic)	Inj: 50 mg/vial	Short-term balanced vasodilation in severe CHF	D: 1–10 µg/kg per minute in D5W; start with 2 µg/kg per minute and increase gradually until desired blood pressure is achieved C: Unknown	Nitrate vasodilatorProtect solution from light Adjust drip rate to maintain mean arterial pressure of approximately 70 mm Hg Discontinue if metabolic acidosis develops Large dose or prolonged use may cause cyanide toxicity
Omega-3 fatty acids (ALA, EPA, and DHA)	Cap: concentrations of EPA and DHA vary with product	Heart failure (to counter cachexia and reduce inflammatory mediators); arrhythmias	D: 40 mg/kg EPA and 25 mg/kg DHA PO q24h C: Same	Fatty acid Side effects rare
Phenoxybenzamine (Dibenzyline)	Cap: 10 mg	Systemic hypertension, especially secondary to a pheochromocytoma	D: 0.25–2.5 mg/kg PO q12h; start at 0.25 mg/kg and titrate upward to effect C: Same	Alpha-1 adrenergic blocker Direct-acting vasodilator
Phenylephrine hydrochloride (generic)	Inj: 10 mg/mL	Acute hypotension	D: 1–3 µg/kg per minute IV CRI; start low and titrate to effect or 0.01 mg/kg IV q15 minutes C: Same	Alpha-1 adrenergic agonist Vasopressor
Pimobendan (Vetmedin)	Chewable tab: 1.25,5.0 mg Cap: 1.25, 2.5, 5.0 mg (Canada, Australia, and Europe)	Licensed for treating signs of mild, moderate, or severe CHF (class II, III, and IV) from dilated cardiomyopathy or valvular insufficiency in dogs Occult DCM in Doberman Pinschers	D: 0.25–0.30 mg/kg PO q12h C: 1.25 mg/cat q12h PO (off-label use for DCM, HCM with heart failure, or restrictive/unclassified cardiomyopathy refractory to standard therapy)	Phosphodiesterase III inhibitor and a calcium sensitizer that acts as an inotropic vasodilator Do not use in aortic stenosis, HCM (dogs), HCM cats without heart failure, or when augmentation of cardiac output is inappropriate
Prazosin HCl (Minipress, generic)	Cap: 1, 2, 5 mg	Balanced vasodilation in CHF, systemic hypertension	D: 1 mg/15 kg PO q8h Titrate to effect C: 0.25–0.5 mg/cat PO q24h	Alpha-1 adrenergic blocker Direct-acting vasodilator Tolerance develops
Procainamide (generic)	Cap: 250, 375, 500 mg Tab: 250, 375, 500 mg Tab: CR, SR: 250, 500, 750, 1000 mg Inj: 100, 500 mg/mL Some oral formulations may be unavailable	Ventricular and supraventricular arrhythmias, WPW	D: 10–30 mg/kg IM, PO q6h 2 mg/kg IV over 3–5 minutes up to total dose of 15 mg/kg 25–50 µg/kg per minute CRI C: 2–5 mg/kg PO q6–8h	Class 1 antiarrhythmic agent Beware hypotension with IV administration Effects increased by high potassium and decreased by low potassium Monitor ECG; 25% prolongation of QRS is sign of toxicity May be difficult to obtain Use with caution in cats Reduce dose with severe renal and liver disease

(*Continued*)

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Cardiopulmonary Drugs—Formulations, Indications, and Dosages—cont'd

DRUG TRADE NAME	FORMULATION	INDICATIONS	DOG DOSE (D) CAT DOSE (C)	COMMENTS
Propantheline bromide (Pro-Banthine, generic)	Tab: 7.5, 15 mg	Rate control in sick sinus syndrome and AV block	D, C: 0.25–0.5 mg/kg PO q8–12h	Anticholinergic
Propranolol (Inderal, generic)	Tab: 10, 20, 40, 60, 80 mg Cap: 60, 80, 120, 160 mg Inj: 1 mg/mL Solution: 4 mg/mL	Atrial and ventricular arrhythmias, hypertrophic cardiomyopathy, systemic hypertension, thyrotoxicosis	D: 0.2–1.0 mg/kg PO q8h 0.02–0.06 mg/kg IV over 5–10 minutes C: <4.5 kg: 2.5–5 mg PO q8–12h >4.5 kg: 5 mg PO q8–12h 0.02–0.06 mg/kg IV over 5–10 minutes	Nonselective beta-blocker Start with low dose and titrate to effect Taper dose when discontinuing therapy Reduce dose with liver disease Beware of possible bronchoconstriction
Quinidine gluconate (generic) Quinidine sulfate (generic)	Tab SR: 324 mg Inj: 80 mg/mL Tab SR: 300 mg	Ventricular and supraventricular arrhythmias, WPW, conversion of atrial fibrillation	D: 6–20 mg/kg PO, IM q6h 6–20 mg/kg PO q8h with sustained-release products 5–10 mg/kg IV (very slowly) C: None Note: Dose calculated for quinidine base equivalent; see comment section	Rarely used; replaced by mexiletine and sotalol for managing ventricular arrhythmias Class 1 antiarrhythmic Reduce dose in CHF, hepatic disease, and hypoalbuminemia Quinidine base in each quinidine salt; quinidine gluconate: 324 mg tab = 200 mg quinidine; quinidine sulfate: 300 mg tab = 250 mg quinidine
Ramipril (Vasotop. Altace, generic)	Tab: 0.625, 1.25, 2.5, 5, 10 mg Cap: 0.625, 1.25, 2.5, 5, 10 mg	Balanced vasodilation in CHF; systemic hypertension	D: 0.125 mg/kg PO q12h C: 0.5 mg/kg PO q24h	Angiotensin-converting enzyme inhibitor Long duration of effect in cats No consistent effect on blood pressure in cats
Rivaroxaban (Xarelto)	Tab: 2.5, 10, 15 mg	Prevention of aortic thromboembolism in cats that have already had a thromboembolic event or are at high risk for thromboembolism	C: 0.6 ± 0.1 mg/kg 2.5 mg PO q24 h for 7 days	Platelet inhibitor (ADP receptor blocker) More effective than aspirin in preventing recurrence of aortic thromboembolism Used in combination with clopidogrel; more effective than monotherapy Low risk of hemorrhage as sole agent
Rapamycin (Rapamune) (Sirolimus)	Tab: 0.5, 1, 2 mg tablets	Treatment of subclinical feline HCM to prevent or delay progression of LV hypertrophy	C: Once-weekly low dose (0.3 mg/kg)	Rapamycin is a promising novel drug for continued study of the treatment of subclinical feline hypertrophic cardiomyopathy
Sildenafil (Viagra, Revatio)	Tab: 25, 50, 100 mg Tab (Revatio): 20 mg	Pulmonary hypertension	D: 2 mg/kg PO q12h but dose interval may range from 8 to 24h C: 1 mg/kg PO q8h	Phosphodiesterase-5 inhibitor Arterial vasodilator that preferentially dilates the pulmonary vasculature Expensive Can cause hypotension

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Sotalol(Betapace, generic)	Tab: 80, 120, 160 mg	Atrial and ventricular arrhythmias	D: 1–2 mg/kg PO q12h (medium-size dogs: start with 40 mg per dog, then increase to 80 mg, if needed) C: 2 mg/kg PO q12h	Antiarrhythmic agent with class II (beta blocking) and class III effects Monitor closely with CHF because of negative inotropic properties
Spironolactone (Aldactone, generic)	Tab: 25, 50, 100 mg	Diseases associated with fluid retention (CHF, hepatic disease, nephrotic syndrome), systemic hypertension, hypokalemia	D: 1–2 mg/kg PO q12h 2 mg/kg/day (cardiac antiremodeling) (European labeled dose: 2 mg/kg PO q24h) C: 1–2 mg/kg PO q12h	Aldosterone antagonist Potassium-sparing diuretic 2–3 days to achieve peak effect Weak diuretic so usually combined with a loop diuretic No effect on cardiac remodeling in Maine Coon cats with HCM Ulcerative facial dermatitis developed in one-third of Maine Coon cats treated with 2 mg/kg q12h dose
Tadalafil (Cialis)	Tab: 2.5, 5, 10, 20 mg	Pulmonary hypertension	D: 1 mg/kg PO q24h	Phosphodiesterase-5 inhibitor Arterial vasodilator that preferentially dilates the pulmonary vasculature Expensive Can cause hypotension
Taurine (generic)	Tab: 250 mg Cap: 500	Dilated cardiomyopathy: cats and selective cases in dogs (especially American Cocker Spaniel)	D: 500 mg PO q12h C: 250 mg PO q12h	Amino acid Clinical improvements noted in 4–10 days Echo improvement usually by 6 weeks Continue supplement for 12–16 weeks while correcting diet
Terbutaline (generic)	Tab: 2.5, 5 mg Inj: 1 mg/mL	Asthma, COPD, symptomatic bradycardia (AV block, sick sinus syndrome)	D: 1.25–5 mg/dog PO q8–12h C: 0.1 mg/kg PO q8h or 0.625–1.25 mg/cat q12h; 0.05 mg/kg SC, IM, IV	Beta-2 agonist bronchodilator Reduce dose by 50% in first 4 days to prevent restless behavior Decrease dose with renal disease
Theophylline (extended-release generic)	Tab: 100, 200, 300, 450 mg Cap: 50, 75, 125, 200 mg (availability of extended-release formulations may be variable)	Asthma, COPD, sick sinus syndrome	D: 9 mg/kg PO q8–12h Extended release: 10 mg/kg PO q12h C: 4 mg/kg PO q12h Extended release: 19 mg/kg PO q24h at night	Methylxanthine bronchodilator Reduce dose with CHF, liver disease, cimetidine, orbifloxacin, enrofloxacin Reduce dose by 50% in first 4 days to prevent anxiety side effect Therapeutic range: 10–20 µg/mL Dose on lean body weight

(*Continued*)

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Cardiopulmonary Drugs—Formulations, Indications, and Dosages—cont'd

DRUG TRADE NAME	FORMULATION	INDICATIONS	DOG DOSE (D) CAT DOSE (C)	COMMENTS
Torsemide (Demadex, generic)	Tab: 5, 10, 20 mg Cap: 100 mg Inj: 10 mg/mL	Refractory CHF with blunted response to furosemide	D: 0.1-0.3 mg/kg PO q12h C: 0.1–0.3 mg/kg PO q24h Titrate to effect	Loop diuretic Longer duration of action, decreased susceptibility to diuretic resistance, and adjunctive aldosterone antagonist properties compared with furosemide Monitor for electrolyte disturbances, azotemia, and dehydration
Triamterene (Dyrenium, generic)	Cap: 50, 100 mg	Diseases associated with fluid retention (CHF, hepatic disease, nephrotic syndrome), systemic hypertension, hypokalemia	D: 1–2 mg/kg PO q12h C: Same	Potassium-sparing diuretic Weak diuretic Usually combined with loop diuretic Does not block aldosterone
Vasopressin (generic)	Inj: 20 U/mL	Treatment of hypotension and cardiac arrest (CPR)	D: Vasopressor 0.01–0.04 U/kg per minute CRI; CPR: 0.2–0.8 U//kg IV (can repeat at 3- to 5 minute intervals and alternate with epinephrine) C: Same	Noncatecholamine vasopressor peptide Potential advantages over epinephrine: Longer half-life Effective in acidosis No harmful beta-adrenergic effects
Verapamil (Calan, Isoptin, generic)	Tab: 40, 80, 120 mg Inj: 2.5 mg/mL	Supraventricular arrhythmias	D: 0.05 mg/kg slow IV (1–2 minutes) boluses given at 10–30 minute intervals (to effect) to a maximum cumulative dose of 0.2 mg/kg C: 0.05 mg/kg slow IV, may repeat twice as described for dog	Calcium channel blocker Diltiazem is a safer alternative in heart failure Potent vasodilator and negative inotrope
Warfarin (Coumadin, generic)	Tab: 1, 2, 2.5, 3, 4, 5, 6, 7.5, 10 mg	Prevention of thromboembolism (does not lyse existing thrombi)	D: 0.1–0.2 mg/kg PO q24h C: Same	Anticoagulant Initiate therapy with 4 days of heparin to prevent initial hypercoagulable state Control animal's lifestyle and environment to minimize risk of trauma Adjust dose to maintain PT at 1.5–2 times baseline value or INR of 2–3

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CALCULATING A CONTINUOUS RATE INFUSION FOR LIDOCAINE

A CRI dosage of 25 to 75 µg/kg per minute of lidocaine can be used in dogs with an intravenous loading dose of 1 to 2 mg/kg. In cats, various sources support a CRI dosage of 10 to 40 µg/kg per minute of lidocaine with an intravenous loading dose of 0.25 to 0.75 mg/kg.

Lidocaine solution is made up by replacing 75 mL of fluid from a liter bag with 75 mL of 2% lidocaine (half the amount if 500-mL bag). Using the appropriate body weight and corresponding fluid rate in the table below will achieve a CRI dosage of 50 µg/kg per minute.

BW (KG)	ML/HOUR
5	10
10	20
20	40
30	60
40	80
50	100
60	120
70	140

SOURCES OF DRUG INFORMATION IN ADDITION TO CHAPTERS IN THIS BOOK

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Appendix

3

Echocardiographic Normal Values

Virginia Luis Fuentes

RATIO INDICES

Normalization Based on Left Ventricular or Left Atrial Diameter Divided by Aortic Diameter

RATIO	VIEW	MEDIAN	LOWER 2.5% RI	UPPER 2.5% RI
LVDd/Ao	RPLax 4ch and 5ch	2.10	1.76	2.43
LA/Ao	RPLax 4ch and 5ch	2.09	1.81	2.44
LA/LVDd	RPLax 5ch and 4ch	0.99	0.89	1.09

Ratio values are independent of body weight. In 95% of normal dogs the aortic-normalized left ventricular diameter in diastole from the above table will be between 1.76 and 2.43. Values above 2.43 suggest the end-diastolic left ventricular diameter is greater than normal.
Ao, Systolic aortic diameter from long-axis view; *LA*, left atrial diameter at end-systole from long-axis four-chamber view; *LVDd*, left ventricular diameter at end diastole; *RPLax 4ch*, right parasternal long-axis four-chamber view optimized for LV inlet; *RPLax 5ch*, right parasternal long-axis four-chamber view optimized for left ventricular outflow tract.
From Strohm LE, Visser LC, Chapel EH et al: Two-dimensional, long-axis echocardiographic ratios for assessment of left atrial and ventricular size in dogs, *J Vet Cardiol* 20:330–342, 2018.

ALLOMETRIC SCALING

Mean (95% Prediction Intervals) for Left Ventricular M-mode Values in Healthy Dogs of Varying Weights

BW (KG)	LVDD (CM)	LVDS (CM)	IVSD (CM)	LVFWD (CM)
3	**2.1** (1.8–2.6)	**1.3** (1.0–1.8)	**0.5** (0.4–0.8)	**0.5** (0.4–0.8)
4	**2.3** (1.9–2.8)	**1.5** (1.1–1.9)	**0.6** (0.4–0.8)	**0.6** (0.4–0.8)
6	**2.6** (2.2–3.1)	**1.7** (1.2–2.2)	**0.6** (0.4–0.9)	**0.6** (0.4–0.9)
9	**2.9** (2.4–3.4)	**1.9** (1.4–2.5)	**0.7** (0.5–1.0)	**0.7** (0.5–1.0)
11	**3.1** (2.6–3.7)	**2.0** (1.5–2.7)	**0.7** (0.5–1.1)	**0.7** (0.5–1.0)
15	**3.4** (2.8–4.1)	**2.2** (1.7–3.0)	**0.8** (0.6–1.1)	**0.8** (0.5–1.1)
20	**3.7** (3.1–4.5)	**2.4** (1.8–3.2)	**0.8** (0.6–1.2)	**0.8** (0.6–1.2)
25	**3.9** (3.3–4.8)	**2.6** (2.0–3.5)	**0.9** (0.6–1.3)	**0.9** (0.6–1.3)
30	**4.2** (3.5–5.0)	**2.8** (2.1–3.7)	**0.9** (0.7–1.3)	**0.9** (0.6–1.3)
35	**4.4** (3.6–5.3)	**2.9** (2.2–3.9)	**1.0** (0.7–1.4)	**1.0** (0.7–1.4)
40	**4.5** (3.8–5.5)	**3.0** (2.3–4.0)	**1.0** (0.7–1.4)	**1.0** (0.7–1.4)
50	**4.8** (4.0–5.8)	**3.3** (2.4–4.3)	**1.1** (0.7–1.5)	**1.0** (0.7–1.5)
60	**5.1** (4.2–6.2)	**3.5** (2.6–4.6)	**1.1** (0.8–1.6)	**1.1** (0.7–1.6)
70	**5.3** (4.4–6.5)	**3.6** (2.7–4.8)	**1.1** (0.8–1.6)	**1.1** (0.8–1.6)

BW, Body weight; *CM*, centimeter; *LVDd*, left ventricular end-diastolic diameter; *LVIDs*, left ventricular end-systolic diameter; *IVSTd*, interventricular septal thickness in diastole; *LVFWd*, left ventricular free wall thickness in diastole.
From Cornell CC, Kittleson MD, Torre PD, et al: Allometric scaling of M-mode cardiac measurements in normal adult dogs, *J Vet Intern Med* 18:311, 2008.

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Mean (95% Prediction Intervals) for Left Ventricular 2D and M-mode Echocardiographic Values in Healthy Dogs of Varying Weights

MEASUREMENT	MODE	RIGHT PARASTERNAL VIEW	97.5 PERCENT.	95 PERCENT.	75 PERCENT.	50 PERCENT.	25 PERCENT.	5 PERCENT.	2.5 PERCENT.	SCALING EXPONENT (B)
LVDd	2D	LAx	1.59	1.55	1.43	1.36	1.29	1.19	1.16	0.316
LVDd	2D	SAx	1.62	1.57	1.45	1.37	1.30	1.20	1.16	0.316
LVDd	M-mode	SAx	1.64	1.60	1.48	1.40	1.33	1.23	1.20	0.299
LVDs	2D	LAx	1.11	1.07	0.96	0.89	0.82	0.73	0.71	0.351
LVDs	2D	SAx	0.97	0.92	0.80	0.73	0.66	0.58	0.55	0.392
LVDs	MM	SAx	0.96	0.92	0.80	0.73	0.66	0.57	0.55	0.387
LAD	2D	LAx4ch	1.57	1.53	1.43	1.37	1.30	1.22	1.19	0.309

Allometric scaling is based on the principle that linear measurements of the heart (in cm) will be proportional to (approximately) the cube root of body weight in kg (= body weight$^{0.3}$).
For example, normalized left ventricular diameter in diastole (LVDdN) = LVDd (cm)/(body weight (kg))$^{0.3}$
The exact value for the scaling exponent depends on whether the echocardiographic measurements are made from M-mode or 2D, or long-axis views versus short-axis views. In the above table, if the left ventricular end-diastolic diameter is measured from a right parasternal long-axis view, the LVDdN (i.e., LVDd/body weight$^{0.316}$) will measure >1.59 in fewer than 2.5% of dogs of a similar weight.
2D, Two-dimensional echocardiography; *LAD*, left atrial diameter; *LAx*, long axis; *LAx4ch*, long-axis four-chamber view; *LVDd,* left ventricular diameter in diastole; *LVDs*, left ventricular diameter in systole; *MM*, M-mode echocardiography; *percent.,* percentile; *SAx*, short axis.
From Visser L.C., Ciccozzi M.M., Sintov D.J., Sharpe A. N. Echocardiographic quantitation of left heart size and function in 122 healthy dogs: a prospective study proposing reference intervals and assessing repeatability. *J. Vet. Intern. Med*. 33: 1909–1920, 2019.

95% Prediction Intervals for Left Ventricular End-diastolic and End-systolic Volumes in Nonsighthounds

BODY WEIGHT	LVEDV LOWER PI	LVEDV UPPER PI	LVESV LOWER PI	LVESV UPPER PI
1	1.4	4.7	0.4	1.5
2	2.8	7.2	0.9	2.3
3	4.0	10.1	1.4	3.5
4	5.4	13.3	1.9	4.8
5	6.8	16.8	2.5	6.2
6	8.1	20.1	3.1	7.6
7	9.3	23.1	3.6	9.0
8	10.4	25.8	4.1	10.1
9	11.4	28.2	4.4	11.0
10	12.4	30.8	4.8	11.8
11	13.6	33.8	5.2	13.0
12	15.0	37.3	5.8	14.4
13	16.6	41.2	6.5	16.2
14	18.4	45.5	7.3	18.2
15	20.2	50.1	8.2	20.3
16	22.0	54.6	9.1	22.5
17	23.8	59.0	9.9	24.7
18	25.6	63.4	10.8	26.9
19	27.3	67.6	11.7	29.1
20	29.0	71.8	12.5	31.2
22.5	32.9	81.4	14.4	35.8
25	36.2	89.6	16.0	39.7
27.5	38.8	96.1	17.3	42.9
30	41.1	101.6	18.4	45.6

95% Prediction Intervals for Left Ventricular End-diastolic and End-systolic Volumes in Nonsighthounds—cont'd

BODY WEIGHT	LVEDV LOWER PI	LVEDV UPPER PI	LVESV LOWER PI	LVESV UPPER PI
32.5	43.3	107.1	19.5	48.2
35	45.5	112.6	20.5	50.8
37.5	47.8	118.3	21.5	53.3
40	50.1	124.0	22.5	56.0
42.5	52.4	129.9	23.6	58.7
45	54.8	135.8	24.8	61.5
47.5	57.3	142.0	26.0	64.5
50	59.9	148.5	27.2	67.8
52.5	62.7	155.4	28.6	71.3
55	65.6	162.8	30.1	75.0
57.5	68.7	170.7	31.6	79.0
60	72.1	179.0	33.3	83.3
62.5	75.6	187.9	35.1	87.8
65	79.3	197.4	37.0	92.7
67.5	83.3	207.4	39.0	98.0
70	87.4	218.1	41.1	103.6
72.5	91.7	229.4	43.2	109.7
75	96.1	241.6	45.4	116.3
77.5	100.6	254.6	47.6	123.6
80	105.2	268.6	49.8	131.6

LVEDV, Left ventricular end-diastolic volume (mL); *LVESV*, left ventricular end-systolic volume (mL); *PI*, 95% prediction intervals.
From Wess G, Bauer A, Kopp A: Echocardiographic reference intervals for volumetric measurements of the left ventricle using the Simpson's method of discs in 1331 dogs, *J Vet Intern Med* 35:724, 2021.

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95% Prediction Intervals for Left Ventricular End-diastolic and End-systolic Volumes in Sighthounds

BODY WEIGHT	LVEDV LOWER PI	UPPER PI	LVESV LOWER PI	UPPER PI
1	2.4	12.9	0.9	5.1
2	3.4	14.7	1.3	5.9
3	4.9	16.9	1.9	6.9
4	6.7	19.7	2.7	8.3
5	9.1	23.7	3.8	10.1
6	11.8	29.0	5.0	12.5
7	14.7	35.3	6.3	15.4
8	17.5	41.6	7.6	18.3
9	20.1	47.3	8.9	21.0
10	22.4	52.5	10.0	23.5
12.5	28.3	65.4	12.8	29.8
15	33.8	78.0	15.5	35.9
17.5	38.8	89.5	18.0	41.6
20	43.3	99.9	20.2	46.8
22.5	47.4	109.1	22.3	51.5
25	50.9	117.2	24.2	55.7
27.5	54.1	124.3	25.8	59.5
30	57.2	131.3	27.5	63.1
32.5	60.6	139.2	29.2	67.1
35	64.3	147.7	30.9	71.2
37.5	68.1	156.5	32.7	75.4

95% Prediction Intervals for Left Ventricular End-diastolic and End-systolic Volumes in Sighthounds—cont'd

BODY WEIGHT	LVEDV LOWER PI	UPPER PI	LVESV LOWER PI	UPPER PI
40	71.6	165.0	34.4	79.4
42.5	74.7	172.4	35.8	82.9
45	77.4	178.8	37.1	85.9
47.5	79.8	184.5	38.2	88.6
50	82.1	189.9	39.3	91.1
52.5	84.4	195.4	40.3	93.8
55	86.7	201.0	41.4	96.5
57.5	89.0	206.7	42.6	99.3
60	91.4	212.6	43.7	102.3
62.5	93.7	218.7	44.9	105.4
65	96.1	224.9	46.0	108.6
67.5	98.4	231.4	47.2	112.0
70	100.7	238.2	48.3	115.6
72.5	102.9	245.3	49.5	119.4
75	105.1	252.8	50.6	123.5
77.5	107.1	260.7	51.7	127.9
80	109.0	269.2	52.7	132.6

LVEDV, Left ventricular end-diastolic volume (mL); *LVESV*, left ventricular end-systolic volume (mL); *PI*, 95% prediction intervals.
LVEDV-I, Left ventricular end-diastolic volume (ml) indexed to body weight (kg); PI, prediction intervals; LVESV-I, left ventricular end-systolic volume (ml) indexed to body weight (kg).
From Wess G, Bauer A, Kopp A: Echocardiographic reference intervals for volumetric measurements of the left ventricle using the Simpson's method of discs in 1331 dogs, *J Vet Intern Med* 35:724, 2021.

NORMAL FELINE ECHOCARDIOGRAPHIC VALUES

Predicted Cardiac Dimensions and 95% Prediction Intervals by Weight for 18460 Cats With Normal Echocardiograms

BODY WEIGHT	IVSD	LVDD	LVFWD	LVDS	FS	LA	AO	LA/AO
(KG)	(MM)	(MM)	(MM)	(MM)	(%)	(MM)	(MM)	
2.0	3.3 (2.5–4.3)	**12.8** (10.2–16.0)	**3.1** (2.4–4.1)	**6.9** (4.6–10.5)	**45** (28–62)	**8.5** (6.3–11.2)	**7.5** (6.0–9.5)	**1.13** (0.85–1.40)
2.5	3.4 (2.6–4.5)	**13.6** (10.9–17.0)	**3.2** (2.5–4.4)	**7.4** (4.8–11.2)	**45** (28–62)	**9.1** (6.8–12.0)	**8.0** (6.3–10.1)	**1.14** (0.86–1.41)
3.0	**3.5** (2.7–4.7)	**14.2** (11.4–17.8)	**3.4** (2.6–4.5)	**7.7** (5.1–11.7)	**45** (28–62)	**9.6** (7.2–12.7)	**8.4** (6.7–10.7)	**1.14** (0.86–1.42)
3.5	**3.7** (2.8–4.9)	**14.8** (11.9–18.5)	**3.6** (2.7–4.7)	**8.0** (5.3–12.2)	**45** (28–62)	**10.0** (7.6–13.4)	**8.8** (7.0–11.1)	**1.15 (**0.87–1.42)
4.0	**3.8** (2.8–4.9)	**15.4** (12.2–19.2)	**3.7** (2.8–4.8)	**8.3** (5.5–12.6)	**45** (28–62)	**10.5** (7.9–13.9)	**9.1** (7.2–11.6)	**1.15** (0.88–1.43)
4.5	**3.9** (2.9–5.1)	**15.8** (12.7–19.8)	**3.8** (2.9–5.0)	**8.6** (5.7–13.0)	**45** (28–62)	**10.9** (8.2–14.5)	**9.4** (7.5–11.9)	**1.15** (0.88–1.43)
5.0	**3.9** (3.0–5.2)	**16.3** (13.0–20.3)	**3.9** (3.0–5.1)	**8.6** (5.7–13.0)	**45** (28–62)	**11.2** (8.4–14.9)	**9.7** (7.7–12.3)	**1.16** (0.88–1.43)
5.5	**4.0** (3.0–5.3)	**16.7** (13.4–20.9)	**4.0** (3.0–5.3)	**9.0** (6.0–13.7)	**45** (28–62)	**11.6** (8.7–15.4)	**10.0** (7.9–12.6)	**1.16** (0.89–1.44)
6.0	**4.1** (3.1–5.4)	**17.1** (13.7–21.4)	**4.1** (3.1–5.4)	**9.0** (6.0–13.7)	**45** (28–62)	**11.9** (8.9–15.8)	**10.2** (8.1–12.9)	**1.16** (0.89–1.44)
6.5	**4.2** (3.1–5.5)	**17.4** (14.0–21.8)	**4.2** (3.1–5.5)	**9.4** (6.2–14.3)	**45** (28–62)	**12.2** (9.2–16.2)	**10.5** (8.3–13.2)	**1.17** (0.90–1.45)
7.0	**4.2** (3.2–5.6)	**17.8** (14.2–22.2)	**4.3** (3.2–5.6)	**9.6** (6.3–14.6)	**45** (28–62)	**12.5** (9.4–16.6)	**10.7** (8.4–13.5)	**1.18** (0.90–1.46)

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Predicted Cardiac Dimensions and 95% Prediction Intervals by Weight for 18460 Cats With Normal Echocardiograms—cont'd

BODY WEIGHT (KG)	IVSD (MM)	LVDD (MM)	LVFWD (MM)	LVDS (MM)	FS (%)	LA (MM)	AO (MM)	LA/AO
7.5	**4.3** (3.2–5.7)	**18.1** (14.5–22.6)	**4.3** (3.3–5.7)	**9.8** (6.5–14.9)	**45** (28–62)	**12.7** (9.6–16.9)	**10.9** (8.6–13.8)	**1.18** (0.91–1.46)
8.0	**4.3** (3.3–5.8)	**18.4** (14.7–23.0)	**4.4** (3.3–5.8)	**10.0** (6.6–15.1)	**45** (28–62)	**12.7** (9.6–16.9)	**11.1** (8.8–14.0)	**1.19** (0.91–1.47)
8.5	**4.4** (3.3–5.8)	**18.7** (15.0–23.4)	**4.4** (3.4–5.9)	**10.1** (6.7–15.4)	**45** (28–62)	**13.2** (10.0–17.6)	**11.3** (8.9–14.3)	**1.19** (0.92–1.47)
9.0	**4.4** (3.3–5.9)	**19.0** (15.2–23.7)	**4.5** (3.4–5.9)	**10.3** (6.8–15.6)	**45** (28–62)	**13.5** (10.1–17.9)	**11.5** (9.1–14.5)	**1.20** (0.92–1.47)
9.5	**4.5** (3.4–6.0)	**19.3** (15.4–24.0)	**4.6** (3.4–5.9)	**10.4** (6.9–15.8)	**45** (28–63)	**13.7** (10.3–18.2)	**11.6** (9.1–14.7)	**1.20** (0.92–1.48)
10.0	**4.5** (3.4–6.0)	**19.5** (15.6–24.4)	**4.6** (3.5–6.1)	**10.5** (6.9–16.0)	**45** (28–63)	**13.9** (10.5–18.5)	**11.8** (9.3–14.9)	**1.21** (0.92–1.48)

Mean and 95% prediction intervals using allometric scaling. *Ao*, 2D aortic diameter from the same frame as the LA diameter; *FS*, left ventricular fractional shortening; *IVSd*, interventricular septal thickness at end diastole; LA, 2D left atrial diameter from a right parasternal short-axis view at early diastole; *LA/Ao*, left atrial/aortic ratio; *LVDd*, left ventricular diameter at end-diastole; *LVDs*, left ventricular diameter at end-systole; *LVFWd*, left ventricular free wall thickness at end diastole.
From Haggstrom J, Andersson AO, Falk T, et al: Effect of body weight on echocardiographic measurements in 19,866 pure-bred cats with or without heart disease, *J Vet Intern Med* 30:1601–1611, 2016.

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Appendix 4

Available Canine and Feline Genetic Tests for Breed-Specific Cardiac Diseases

Note that there are genotypic tests (gene test where the gene is identified or the less-definitive linkage tests where there is an identified gene close to the gene of interest) and phenotypic tests such as an echocardiogram that can be used to characterize inherited disorders. There are cardiac diseases that are present in multiple breeds with different mutations at their core, and congenital disorders with no proven genetic basis. This appendix covers available gene and linkage testing. This list will change as new tests come to market.

BREED	CONDITION	LABORATORY
Canine		
Boxer	Arrhythmogenic right ventricular cardiomyopathy	NCSU Veterinary Cardiac Genetics Laboratory
Doberman Pinscher	Dilated cardiomyopathy	NCSU Veterinary Cardiac Genetics Laboratory UC Davis Veterinary Genetics Laboratory
Rhodesian Ridgeback	Inherited ventricular arrhythmia	NCSU Veterinary Cardiac Genetics Laboratory
Schnauzer (standard and giant)	Dilated cardiomyopathy	University of Missouri DCM Testing
Newfoundland	Subaortic stenosis	NCSU Veterinary Cardiac Genetics Laboratory
Portuguese Water Dog	Juvenile dilated cardiomyopathy	PennGen
Feline		
Maine Coon	Hypertrophic cardiomyopathy	UC Davis Veterinary Genetics Laboratory
BREED	**CONDITION**	**LABORATORY**
		NCSU Veterinary Cardiac Genetics Laboratory
Ragdoll	Hypertrophic cardiomyopathy	NCSU Veterinary Cardiac Genetics Laboratory
		UC Davis Veterinary Genetics Laboratory

Laboratory contact information:
NCSU Veterinary Genetics Laboratory
NC State University
College of Veterinary Medicine Research
Bldg. 326
1060 William Moore Dr.
Raleigh, NC 27607, United States
919-515-3277
PennGen
3900 Delancey Street, Rm. 4022
Philadelphia, PA 19104, United States
215 898-5703
UC Davis Veterinary Genetics Laboratory
PO Box 1102
Davis, CA 95617-1102, United States
530 752-2211
University of Missouri DCM Testing
Dr. Gary Johnson, DCM Testing
320 Connaway Hall-UMC
1500 Bouchelle Ave
University of Missouri
Columbia, MO 65211, United States
573-884-3712

CARDIAC GENETICS KEY

AD = autosomal dominant
AR = autosomal recessive
ARVC = arrhythmogenic right ventricular cardiomyopathy
ASD = atrial septal defect
DCM = dilated cardiomyopathy
HCM = hypertrophic cardiomyopathy
IP = incomplete penetrance
MMVD = myxomatous mitral valve disease
PDA = patent ductus arteriosus
PS = pulmonic stenosis
SAS = subaortic stenosis
SCD = sudden cardiac death
ToF = tetralogy of Fallot
TVD = tricuspid valve dysplasia
VSD = ventricular septal defect

Courtesy Jessica Ward, DVM, DACVIM (Cardiology).

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