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INTRODUCTION: THE BODY DEFENSE SYSTEMS—THE BODY'S RESPONSE TO DISEASE

Animals, and their humans, live their lives in an unfriendly, hostile environment. They are continually assaulted by hordes of microorganisms such as bacteria, viruses, protozoans, fungi, and parasites. Internally, abnormal cells produced by cellular division must be continually removed from the body. If allowed to survive, they become tumors. Some of these tumors may become malignant and spread throughout the body. Tissues within the body are continually being repaired or replaced as they wear out or become damaged. With all this activity going on in the body, it is a wonder that animals and humans survive in this environment.

IMMUNITY

The animal body has developed an efficient system of defense against disease-producing agents: the *immune system.* Components of the immune system patrol the body 24 hours a day looking for foreign and internal enemies. The activities of this system are called *immunity;* without it, animals could not survive. Immunity can be divided into two large categories: *nonspecific immunity* and *specific immunity.*

Nonspecific Immunity

Nonspecific immunity is composed of several elements: species resistance, mechanical and chemical barriers, the inflammatory response, interferon, and complement. The term *nonspecific* means that the system responds to all antigenic insults in the same manner, not specifically to any one type of pathogenic organism.

Species Resistance

Species resistance refers to the genetic ability of a particular species to provide defense against certain pathogens. For example, canines do not acquire feline leukemia virus, and felines do not contract canine distemper virus. Neither species can contract plant diseases. Knowledge of species resistance can allow a clinician or veterinary technician to focus on the group of diseases seen in that animal species and not spend time ruling out those conditions that do not appear.

Mechanical and Chemical Barriers

The animal's internal body is protected by a mechanical barrier: the skin and the mucous membranes. If unbroken, this barrier prevents the entry of microorganisms, protecting the underlying tissues from injury. The skin also produces substances such as sebum, mucus, and enzymes that act to inhibit or destroy pathogens. Damage to this barrier allows organisms to reach the internal structures of the body and produce disease. Healthy skin is the animal's best defense against the world of microorganisms. It is called the "first line of defense."

Inflammatory Response

If bacteria or other invaders do gain access to the body, a "second line of defense," known as the *inflammatory response,* exists. When a tissue is invaded by microorganisms or injured in any way, the cells that make up that tissue release enzymes called *mediators;* these mediators attract white blood cells to the area *(chemotaxis),* dilate blood vessels, and increase the permeability of the vessels in the area. The characteristic signs of inflammation—heat, redness, swelling, and pain—occur as a result of the release of these chemical substances. Specific types of white blood cells (usually neutrophils) attracted to the area will begin to "gobble up" the invading foreign material in a process known as *phagocytosis.* The increased blood flow to the area will increase the temperature of the tissue, inhibiting the growth of new organisms. It also brings in raw materials for repair of the damaged tissue and clotting factors to assist in hemorrhage control. With time, the body is able to clean up the damage and return the tissue to its normal state.

Interferon and Complement

Chemicals produced by cells invaded by viruses also make up part of nonspecific immunity. Interferon is a

substance that interferes with the ability of viruses to cause disease by preventing their replication within the host cell. Complement, another group of enzymes, is activated during infection. Complement binds to the invading cell wall, producing small holes in the membrane. This results in rupture, or *lysis,* of the foreign cell.

Specific Immunity

Specific immunity, the "third line of defense," is conducted by two types of white blood cells called *lymphocytes.* There are two main categories of lymphocytes, B- and T-cell lymphocytes. *B-cell lymphocytes* produce antibodies in response to specific *antigen* stimulation. This is known as the *humoral response. T-cell lymphocytes* interact more directly with the pathogens by combining directly with the foreign agent and destroying it or rendering it incapable of causing disease. Because this response is more direct than that of the B cell, it is known as *cell-mediated immunity.*

Cell-Mediated Immunity

T cells originate in the bone marrow of the animal. After leaving the bone marrow and entering the circulation, they arrive at the *thymus,* a glandular structure found in the mediastinum just cranial to the heart. The thymus is the primary central gland of the lymphoid system and is quite large in young animals, but decreases in size as the animal matures. Here the T cells "go to college," where they are programmed to recognize the markers that are unique on the cells of that specific animal *(self-recognition).* After "graduation," the T cells move out to the spleen and lymph nodes and circulate through the body, constantly on the lookout for invading substances.

Macrophages, a type of white blood cell, also travel through the tissues looking for foreign substances. When they find one, they attach to it and take the invader to the T cell. The T cell then attaches to the receptor site on the invading cell and divides repeatedly. All the new T cells then migrate to the site of the infection and begin to destroy the invading organisms. T-cell response is rapid and deadly to pathogens.

Humoral Immunity

B-cell response (humoral) is a slower type of immune response. Like T cells, B cells originate within the animal's bone marrow or in the bursa of Fabricius in some species. Young, inactive B cells produce *antigen-combining receptor sites* over the surface of their cell membranes. On contact with a specific antigen, the cell divides repeatedly, producing a *clone* of identical B cells. Some of these B cells become *plasma cells* and are stimulated to produce large protein molecules called *antibodies;* others remain as *memory cells,* which have the ability to recognize the antigen if it is ever again presented to them. Each clone of B cells, and hence each antibody, is specific for only one antigen. The antibody produced is a large protein molecule *(immunoglobulin)* whose chemical structure contains an area that is able to lock onto the antigen (Fig. I.1). Combining with the antigen may result in rendering the antigen harmless to the body, may cause antigens to clump together *(agglutinate)* and be removed from solution, or may result in the destruction of the antigenic cell. This humoral response is not immediate. It takes time for the B cells to clone and begin to produce antibodies. Within 7 to 10 days after the initial infection, antibodies can be found in the body. However, if the animal has been exposed to the antigen previously and memory cells are present, this period is shorter.

B- and T-cell immunity can be further classified according to the manner in which they develop. *Inherited immunity* occurs as a result of genetic factors that influence the developing animal before birth. *Acquired immunity* is resistance that develops after the animal is born. Acquired immunity may be either natural or artificial. *Natural immunity* occurs every time the animal is exposed to a pathogen. It is a continual process in the animal world. *Artificial immunity* is usually the result of deliberate exposure to a pathogen such as with vaccinations. Both natural and artificial immunity can be further divided into either passive or active immunity. In *passive immunity,* antibodies formed in one infected animal are transferred to another animal that is not infected. This transfer provides the uninfected animal with protection against the pathogen. *Active immunity* occurs when the animal's own immune system encounters a pathogen and responds by producing an immune response.

The ultimate result of both specific and nonspecific immunity is that the body eliminates foreign substances, whether they are bacteria, viruses, protozoa, parasites, or the body's own cells that have become harmful. If this

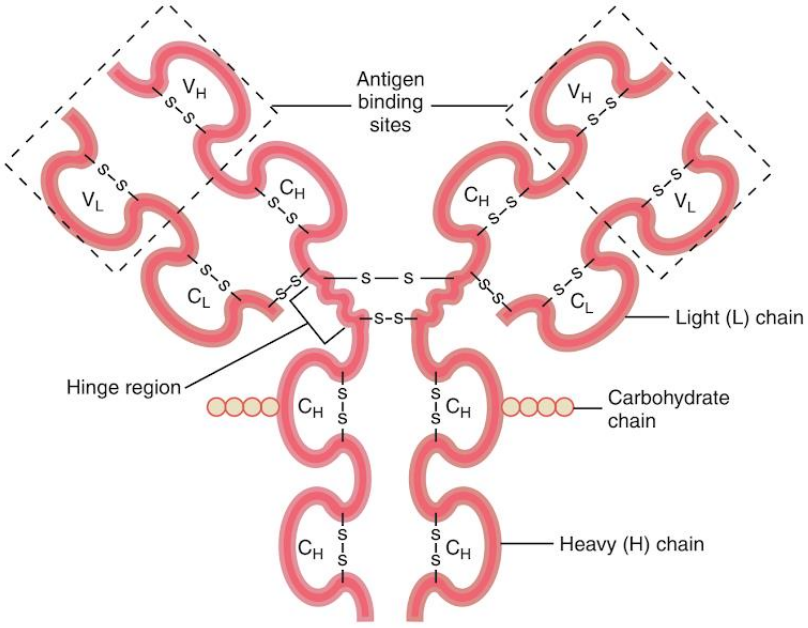

Fig. I.1 Chemical structure of the immunoglobulin G class of antibody. Each molecule is composed of four polypeptide chains (two heavy and two light) plus a short carbohydrate chain attached to each heavy chain. The variable chain gives the immunoglobulin its specificity. *C,* Constant region; C_H, constant region of heavy chain; C_L, constant region of light chain; *s-s,* sulfur-sulfur bonds; *V,* variable region; V_H, variable region of heavy chain; V_L, variable region of light chain.

system fails or is overwhelmed, disease occurs. Many factors affect the proper functioning of the immune system, such as nutrition, stress, sanitation, and age. Concurrent disease can also weaken the immune system, allowing other organisms to gain access to the body. Veterinary technicians must be familiar with the effects these elements have on the health of the animals in their care and be able to educate pet owners in the areas essential for the healthy life of their pets.

WHAT HAPPENS WHEN THE SYSTEM DOES NOT FUNCTION PROPERLY?

This book discusses some of the most commonly seen diseases of domestic animals. The technician should keep the function of the immune system in mind as these diseases are discussed. Disruption of the normal functioning of the immune system results in the clinical illnesses seen in our patients.

Common Diseases *of* Companion Animals

1

Diseases of the Cardiovascular System

LEARNING OBJECTIVES

When you have completed this chapter, you will be able to:

- Demonstrate a working knowledge of the anatomy and physiology of the cardiovascular system.
- Explain to clients how cardiovascular disease affects the patient.
- Explain diagnostic and treatment plans to clients.
- Answer clients' questions concerning the medications needed by the patient.

OUTLINE

KEY TERMS

Bradycardia
Cardiomyopathy
Congenital
Echogenicity
Embolism
Endocarditis
Myocarditis
Holosystolic
Hypertrophic
Hypervolemia
Hypovolemia
Precordial thrill
Tachycardia
Taurine
Thrombus

The cardiovascular system plays an important role in maintaining homeostasis throughout the body. It performs this function by regulating the flow of blood through miles of vessels and capillaries. It is in capillaries that vital nutrients are transported into the body cells and removal of waste materials from the cells occurs.

To understand cardiovascular disease, one must first study the anatomy and physiology of the cardiovascular system (refer to an anatomy and physiology text for a detailed description). Simply stated, the cardiovascular system is composed of a pump (the heart) and pipes (vessels). The pump circulates fluid (blood) through vessels, where it delivers its content to the cells and removes waste products. This system is a "closed" system—that is, change in one portion of the system affects other portions of the system.

ANATOMY AND PHYSIOLOGY

The Pump

At the center of the cardiovascular system is the heart, a four-chambered pump designed to contract, pumping blood to all parts of the body. Two atria (right and left) sit on top of two ventricles (also right and left). The right atrium is separated from the right ventricle by the right atrioventricular valve, also called the *tricuspid valve* because it has three leaflets. The left atrium is separated from the left ventricle by the left atrioventricular valve, or the *mitral valve*. The atrioventricular (AV) septum divides the entire right side of the heart from the left side. Lining tissue of the heart, the endocardium, also covers these valves. Specialized cardiac muscle cells, located in the sinoatrial (SA) node just inside the right atrium, generate an electrical impulse that spreads across both atria and then down the septum to the AV node, where it is slowed down. From there, the impulse travels into the Bundle of His (the AV bundle) and then out to the ventricles along the Purkinje fibers. The arrival of this electrical impulse results in the contraction of the atria and ventricles simultaneously (systole). Blood from the right atrium fills the right ventricle by gravity (80%) and by contraction (20%). Blood from the left atrium fills the left ventricle. The closing of the AV valves produces the first heart sound. Contraction of the ventricles pushes blood *into* the pulmonary artery through the pulmonic valve on the right side of the heart and into the aorta through the aortic valve on the left side and returns blood to the right heart from veins. Closing of the pulmonic and aortic valves creates the second heart sound. This electrical activity can be measured as it moves across the surface of the body by using an *electrocardiograph* (Fig. 1.1). The electrocardiographic instrument measures the electrical activity generated by the heart by the placement of electrodes at specific points on the body surface. Each mechanical contraction of the heart is preceded by an electrical wave front that stimulates heart muscle contraction. This

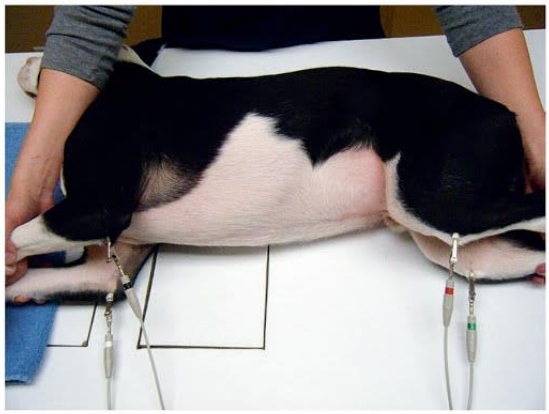

Fig. 1.1 Example of correct positioning and lead placement for performing electrocardiography (ECG). Note that the dog is in right lateral recumbency, the limbs are perpendicular to the body, and the white electrode is on the right forelimb, the black electrode on the left forelimb, the green electrodes on the right hindlimb, and the red electrode on the left hindlimb. (From Bassert J, Thomas J. *McCurnin's clinical textbook for veterinary technicians*. 8th ed. St Louis, MO: Saunders; 2014.)

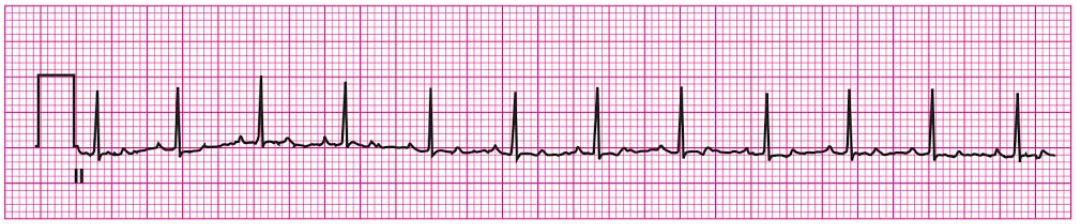

Fig. 1.2 Six-lead electrocardiogram documenting normal sinus rhythm with a heart rate of approximately 150 beats/min. (Modified from August JR. *Consultations in feline internal medicine.* Vol 6. St. Louis, MO: Saunders; 2010.)

electrical wave front begins at the SA node and travels to the muscle cells of the ventricle through the cardiac conduction system. These wave fronts are recorded as the electrocardiogram (ECG). Fig. 1.2 shows a normal ECG of a dog. Fig. 1.3 represents the normal pathway for electrical conduction through the heart.

The electrical activity of this pump is automatic but can be adjusted by input from the *neuroendocrine system* to meet the demands of the animal's body. Both the sympathetic and the parasympathetic nervous systems augment the rhythmic contraction of the heart.

Many cardiac diseases involve a failure of this pump to function properly. *Congestive heart failure (CHF), cardiomyopathy, valvular disease,* and *congenital malformations* can all affect the pumping efficiency of the heart and, ultimately, the function of the entire body.

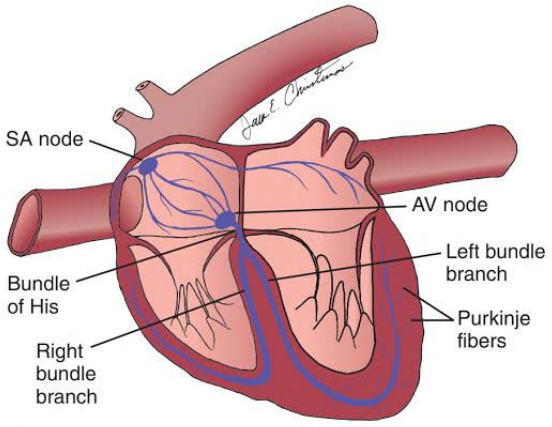

Fig. 1.3 Normal pathway for electrical conduction through the heart. *AV,* Atrioventricular; *SA,* sinoatrial. (From McBride DF. *Learning veterinary terminology.* 2nd ed. St. Louis, MO: Mosby; 2002, by permission.)

The Vessels

Connected to the pump are a series of vessels. Arteries carry oxygenated blood at high pressure (the systolic blood pressure) to arterioles and onto capillaries, where exchange of nutrients and gases occurs. Blood then moves into venules, through veins, and is returned to the right side of the heart via the vena cava. Excessive fluid remaining in the tissue surrounding capillaries is returned to the vascular system via the lymph vessels. Arteries, whose walls contain a large amount of smooth muscle, are capable of dilation and constriction, routing blood to areas where it is needed and away from those areas not in need. Constriction serves to increase blood pressure, and dilation serves to decrease it.

TECH ALERT

The pulmonary artery is the only artery in the body carrying unoxygenated blood, and pulmonary veins are the only veins carrying oxygenated blood!

Vascular diseases affect the flow of blood through the body and, ultimately, its return to the heart. If the volume of blood returning to the heart is abnormal, the heart will compensate by altering the rate of contraction, the strength of contraction, or both to return homeostasis to the circulatory system.

Heart Failure

When the blood returning to the heart cannot be pumped out at a rate matching the body's need, *heart failure* occurs. Many causes for heart failure exist, and the disease is often difficult to explain. The clinical signs of the disease and treatment regimens depend on the diagnosis and evaluation of the *individual* animal. The veterinarian must determine whether the failure is the result of *myocardial dysfunction* (pump failure) or *circulatory failure* (lack of circulating fluid volume).

Myocardial dysfunction is seen in diseases such as the following:

- Cardiomyopathy
- Myocarditis
- Taurine deficiency in cats

Circulatory failure results from the following conditions:

- Hypovolemia (shock, hemorrhage, dehydration)
- Anemia
- Valvular dysfunction
- Congenital shunts or defects

TECH ALERT

Technicians should train themselves to always listen to the heart, not just for heart rate, but also for any arrhythmias or abnormal sounds so they can alert the doctor. You should always listen for 1 full minute for rate and arrhythmias.

Heart failure is termed CHF when the failing heart allows fluid congestion and edema to accumulate in the body. Most heart failure will become "congestive" as the pump progressively fails.

Today, it is possible for researchers to look into the myocardial cells themselves, even to the level of the deoxyribonucleic acid (DNA) within the nucleus to explain the physiological changes seen in patients with heart failure. To understand these diseases, the technician needs an understanding of the workings of the myocardial cell in general.

The myocardial cell is striated and involuntary. Each cell contains parallel sarcomeres containing myosin and actin fibers just like skeletal muscle. Movement of these fibers over one another results in a shortening of the cell and contributes to muscle shortening or contraction. Unlike skeletal muscle, myocardial cells have a very small sarcoplasmic reticulum for calcium storage and hence they are dependent on blood calcium for contraction. The myocardial cells are linked to other myocardial cells through strong electrical intercalated discs. This network of myocardial cells is able to react as one electrically coupled unit. Cardiac muscle cells have a longer refractory period than skeletal muscle cells to allow for filling of the chambers of the heart during diastole. Researchers have found that disarray of these sarcomeres within the cardiac muscle is often responsible for problems seen in patients with heart failure.

CARDIOMYOPATHIES

Canine Dilated Cardiomyopathy

Dilated cardiomyopathy (DCM) is one of the most common acquired cardiovascular diseases of dogs. It is primarily a disease of older, male, large- and giant-breed dogs such as Scottish Deerhounds, Dobermans, Boxers, Irish Wolfhounds, St. Bernards, Newfoundlands, Afghans, and Old English Sheepdogs. The disease has also been seen in English and American Cocker Spaniels. It is rare in dogs weighing less than 12 kg.

The pathology of the disease involves dilation of all chambers of the heart. This dilation (caused by weak, thin, and flabby cardiac muscle) results in a decrease in cardiac output and an increase in cardiac afterload (blood left in the heart in diastole). The cause of this disease is unknown, although its onset often follows myocardial insult from viral, bacterial, nutritional, or immune-mediated diseases. DCM results in impaired systolic function of the ventricles and, therefore, decreased stroke volume (the volume of blood ejected from the heart with each contraction). The effect on the animal is one of low-output circulatory failure, exhibited by weakness, exercise intolerance, syncope, or shock.

Dogs with DCM frequently experience development of atrial fibrillation (AF), which further contributes to a decrease in cardiac output. Signs of AF include rapid, irregular heart rhythms or sudden death. Patients may remain normal until the atria dilate excessively. The enlarged atria are unable to contract normally, and clinical signs of heart disease become evident. The cause of this dilation appears to be breed related. In Dobermans, the disease appears to be familial, related to an autosomal dominant gene. Great Danes and Irish Wolfhounds also demonstrate a genetic predisposition for this disease. In Cocker Spaniels, a taurine deficiency results in DCM. The disease in Cocker Spaniels appears to be related to diets high in lamb meat and rice and low in taurine. Although DCM is primarily a disease of older dogs, Portuguese Water Dogs exhibit a juvenile onset of the disease, which is also genetic. Puppies anywhere from 2 to 32 weeks of age can be affected.

Clinical Signs

- Giant- or large-breed male dogs; 4 to 10 years of age
- Right-sided heart failure: ascites, hepatomegaly, weight loss, abdominal distension
- Left-sided heart failure: coughing, pulmonary edema, syncope

- Exercise intolerance
- Murmur of mitral regurgitation heard best on left chest
- +/– gallop rhythm
- +/– tachyrhythm

Diagnosis

- Radiographs: may be normal early in the disease. May show enlarged heart later in the disease time line; left ventricular enlargement, enlargement of both atria may be visible
- Echocardiology: test of choice for examination of the heart; will demonstrate left and right atrial wall thinning along with left ventricular dilation
- ECG: may show widened QRS and P waves, rhythm disturbances but is fairly insensitive to changes seen in DCM

Laboratory Tests

- The use of cardiac biomarkers is gaining in popularity for diagnosis of DCM. These tests look for myocardial cell injury seen in DCM.
- Atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP), and pro-BNP blood tests are commercially available. In DCM, these values will be significantly increased.
- Troponin 1 (cTn1) will also be increased. (Whole blood is recommended over plasma for this test, but technicians should check with local laboratory before sample collection.)

Treatment

- No cure exists for DCM; treatment is aimed at keeping the dog comfortable
- Diuretics: furosemide to decrease fluid load and reduce work of the heart
- Enalapril: angiotensin-converting enzyme (ACE) inhibitor prevents the formation of angiotensin II, a potent vasoconstrictor; helps decrease vascular resistance and improve cardiac output
- +/– beta-blockers (β-blockers): metoprolol, propranolol, esmolol are examples
- Pimobendan: a calcium sensitizer with inhibitory properties. It increases the calcium binding capability at cTn1 sites. The result is a more forceful contraction of the myocardial cell. The drug also has an antithrombotic effect and is a positive inotrope. Its use has been shown to slow the progression of the disease and to improve survival times

Information for Clients

- DCM is a progressive disease that is almost always fatal.
- Most dogs will die within 6 months to 2 years.
- Dogs may die suddenly of malignant cardiac arrhythmias. Avoid excessive exercise in these animals.
- The disease does appear to be more prevalent in certain breeds of dogs and has been proven to run in families of many dog breeds. Biomarkers may be of use to diagnose the disease early on.

Canine Hypertrophic Cardiomyopathy

In the rare canine disease hypertrophic cardiomyopathy (HCM), the left ventricular muscle hypertrophies or thickens, decreasing the filling capacity of the ventricle and often blocking the outflow of blood during systole. The cause appears to be heritable.

Clinical Signs

- Fatigue
- Cough
- Tachypnea
- Syncope
- Presence or absence of cardiac murmurs
- Sudden death
- Some animals may be asymptomatic

Diagnosis

- Echocardiology: indicates concentric thickening and hypertrophy of the left ventricle

Treatment

- None routinely used

Information for Clients

- Sudden death and CHF may occur in dogs with HCM.
- The disease may run in families of certain breeds: German Shepherds, Rottweilers, Dalmations, Cocker Spaniels, Boston Terriers, Shih Tzus.

Boxer Right Ventricular Cardiomyopathy

This cardiomyopathy occurs in adult Boxer dogs that present with ventricular arrhythmias, syncope, and sudden death. This is a genetic disease seen within families of Boxers and appears to be an autosomal dominant trait with variable penetration. Some dogs may show no signs of the disease, whereas others may have varying signs.

Clinical Signs

- Syncope—may be associated with exercise
- Sudden death
- Some dogs will present with left or biventricular heart failure

Diagnosis

Physical Examination

- Many dogs will have a normal physical examination
- Tachyarrhythmias, ascites, and murmurs may be present

Laboratory Findings

- Biomarkers may be of value in diagnosing this disease
- Cardiac cTn1 levels will be elevated
- Clinical serum chemistries may be within normal limits

Imaging

- ECG: a short recording may be normal. However, these dogs will have ventricular premature contractions on an ECG if the recording is long enough to see them
- Holter monitor: will allow the veterinarian to more accurately diagnose this disease. Increased numbers of ventricular premature complexes (VPCs) should indicate a problem
- Radiographs: usually normal but may show left ventricular enlargement
- Echocardiology: will show left ventricular dilation and systolic dysfunction. Some dogs will have right ventricular enlargement

Treatment

- Mexiletine: to decrease the VPCs
- Pimobendan, ACE inhibitors, if ventricular dilation is present
- Owners should be warned that sudden death of these dogs can occur usually with exercise or excitement

TECH ALERT

When monitoring anesthesia on Boxer dogs, be alert for the presence of VPCs on the ECG monitor. They should not occur in normal dogs. If they are present, this may indicate the dog needs a further cardiac workup.

Feline Dilated Cardiomyopathy

Before the late 1980s, feline DCM was one of the most frequent cardiac diseases reported in cats. After the association of the disease with taurine deficiency, additional taurine was added to commercial diets, and the incidence of the disease significantly decreased. The pathological condition is similar to DCM in dogs. Evidence has been found of a genetic predisposition to DCM in cats fed taurine-deficient diets.

Clinical Signs

- Older, mixed-breed cats
- Dyspnea
- Inactivity
- Anorexia
- Acute lameness or paralysis, usually in the rear limbs
- Pain and lack of circulation in the affected limbs
- Hypothermia

Diagnosis

- Clinical signs
- ECG: increased QRS voltages, wide P waves, ventricular arrhythmia
- Echocardiology: dilated heart chambers

Treatment

- Oral taurine supplementation: 250 to 500 milligrams twice per day (mg/day)
- Furosemide: to reduce fluid load on the heart
- Oxygen: to increase oxygen levels to the cells
- Digoxin: to increase cardiac contractility and improve cardiac output
- Enalapril: ACE inhibitor to prevent the formation of angiotensin II and decrease vascular resistance; improves cardiac output
- Pimobendan
- Hydralazine: relaxes vascular smooth muscle and decreases peripheral resistance; improves cardiac output
- Anticoagulants can be used to dissolve or prevent further blood clots

TECH ALERT

Avoid intravenous (IV) fluid replacement in cats until pulmonary edema or pulmonary effusion is under control.

Information for Clients

- The most dangerous time during treatment of feline DCM is the first 2 weeks.
- Cats that survive the first 2 weeks and respond well to taurine supplementation have a favorable prognosis.

- Cats that do not respond to taurine supplementation have a poor long-term prognosis.

TECH ALERT

- Be extremely careful when handling these cats. The cat may die suddenly while you are attempting to collect laboratory samples or obtain radiographs.

Feline Hypertrophic Cardiomyopathy

HCM in cats is similar to the disease in dogs, with left ventricular hypertrophy being the predominant pathology. This disease is the most common cardiomyopathy seen in cats. Of the feline cardiac cases, up to 35% involve HCM. Neutered male cats between 1 and 16 years of age have been found to be most at risk. This disease is more common in Main Coon and Ragdoll breeds. The cause of the disease may be related to abnormal myocardial myosin or calcium transport within the myocardial cells. The left ventricle becomes thickened and stiff. Mitral regurgitation and aortic embolization occur frequently.

As the atria dilate, the endothelium lining the chambers is damaged, resulting in the release of clotting enzymes, which can result in clot formation. The cats that form thrombi also show evidence of hypercoagulability of their platelets. Thromboembolism occurs in about 16% to 18% of feline HCM cases. Although the thrombus can lodge in any artery, it appears that the trifurcation of the aorta is a frequent spot resulting in a decrease in circulation to both rear legs.

Clinical Signs

- A soft, systolic murmur (grade 2–3 or 6)
- Gallop rhythms or other arrhythmia
- Acute onset of heart failure or systemic thromboembolism

Diagnosis

- Radiographs: may show a normal-size heart or mild left atrial enlargement. May see the "valentine" heart shape in the dorsoventral view
- ECG: increased P-wave duration, increased QRS width, sinus tachycardia
- Echocardiology: increased left ventricular wall thickness and a dilated left atrium
- Biomarkers: BNP, pro-BNP, and CTn1 will be increased
- Magnetic resonance imaging (MRI): most accurate method of diagnosis

Treatment

- ACE inhibitors
- +/– Propranolol, Atenolol: β-blocker; used to decrease myocardial oxygen demand, decrease sinus heart rate

  or
- Diltiazem: calcium channel blocker; inhibits cardiac and vascular smooth muscle contractility; reduces blood pressure and cardiac afterload
- ACE inhibitors
- Low-dose heparin or low-dose aspirin
- Diuretic: furosemide

TECH ALERT

Monitor ECG, heart rate, and blood pressure; may see bradycardia and hypotension at higher doses.

Information for Clients

- Cats with HCM may experience heart failure, arterial embolism, and sudden death.
- Cats with heart rates less than 200 beats/min have a more favorable prognosis compared with cats whose rates are greater than 200 beats/min.
- The median survival time is about 732 days.

Thromboembolism

Thrombus formation is a common and serious complication of myocardial disease in the cat. It is estimated that between 10% and 20% of cats with HCM will experience development of thrombi on the left side of the heart, which may dislodge and become trapped elsewhere in the arterial system. Cats appear to have inherently high platelet reactivity, making clot formation a more likely sequel to endothelial damage and sluggish blood flow occurring with myocardial disease. Approximately 90% of these emboli lodge as "saddle thrombi" in the distal aortic trifurcation, resulting in hindlimb pain and paresis. Rarely will a thrombus lodge at other arterial sites such as the renal artery, the coronary arteries, the cerebral arteries, or the mesenteric artery.

The goal of treatment is to dissolve the thrombus and restore perfusion to the area. Several drugs have been tried with varying results. Tissue plasminogen activator (tPA) has shown some success, but it is expensive.

Heparin has also been used with some success. Low-dose aspirin therapy can be used prophylactically in cats with myocardial disease.

Clinical Signs

- Acute onset of rear leg pain and paresis accompanied by vocalization
- Cold, bluish foot pads (decreased circulation)
- Lack of palpable pulses in rear limbs
- History or clinical findings of myocardial disease

Diagnosis

- Clinical signs
- Nonselective angiography, if available

Treatment

- TPA (Activase [Genentech]): serves as a fibrolysin resulting in the breakdown of clots already formed in the vasculature
  or
- Heparin: acts on coagulation factors in both the intrinsic and extrinsic coagulation pathways, inhibits the formation of a stable clot
- Prophylaxis: low-dose aspirin

TECH ALERT

Aspirin use in cats can cause toxicities because of their inability to rapidly metabolize and excrete salicylates. Cats must be dosed carefully and monitored carefully when receiving aspirin therapy.

Information for Clients

- Cats experiencing painful, cold, or paralyzed rear legs should be seen at the hospital immediately.
- The prognosis for cats with thromboembolism is guarded to poor.
- Surgical removal of the thrombus is difficult.

CONGENITAL HEART DISEASE

Although malformations of the heart and great vessels represent a small cause of clinical heart disease, it is important to identify them in newly acquired pets or those to be used for breeding. Technicians should be encouraged to use their stethoscopes to routinely listen to the heart. With practice, subtle changes will become noticeable, allowing the technician to note abnormalities in the patient's record.

Many malformations have a genetic basis. Breed predilections for congenital heart disease are listed in Table 1.1. The diagnostic approach for congenital heart disease should include a detailed history, with special

TABLE 1.1 Canine Breed Predilections for Congenital Heart Disease

Breed	Defect(s)
Basset Hound	P
Beagle	PS
Bichon Frise	PDA
Boxer	SAS, PS, ASD
Boykin Spaniel	PS
Bull Terrier	MVD, AS
Chihuahua	PDA, PS
Chow Chow	PS, CTD
Cocker Spaniel	PDA, PS
Collie	PDA
Doberman Pinscher	ASD
English Bulldog	PS, VSD, TOF
English Springer Spaniel	PDA, VSD
German Shepherd	SAS, PDA, TVD, MVD
German Shorthaired Pointer	SAS
Golden Retriever	SAS, TVD, MVD
Great Dane	TVD, MVD, SAS
Keeshond	TOF, PDA
Labrador Retriever	TVD, PDA, PS
Maltese	PDA
Mastiff	PS, MVD
Newfoundland	SAS, MVD, PS
Pomeranian	PDA
Poodle	PDA
Rottweiler	SAS
Samoyed	PS, SAS, ASD
Schnauzer	PS
Shetland Sheepdog	PDA
Terrier breeds	PS
Weimaraner	TVD, PPDH
Welsh Corgi	PDA
West Highland White Terrier	PS, VSD
Yorkshire Terrier	PDA

AS, Aortic stenosis; *ASD,* atrial septal defect; *CTD,* cor triatriatum dexter; *MVD,* mitral valve dysplasia; *PDA,* patent ductus arteriosus; *PPDH,* peritoneopericardial diaphragmatic hernia; *PS,* pulmonic stenosis; SAS, subaortic stenosis; *TOF,* tetralogy of Fallot; *TVD,* tricuspid valve dysplasia; *VSD,* ventricular septal defect.

From Oyama MA, Sisson DD, Thomas WP, Bonagura JD. Congenital heart disease. In Ettinger SJ, Feldman EC, eds. *Textbook of veterinary internal medicine.* 6th ed. Vol 2. St. Louis, MO: Saunders; 2005.

attention paid to the breed, sex, and age of the patient. Clinical signs of CHF include failure to grow, dyspnea, weakness, syncope, cyanosis, seizures, and sudden death; however, many animals with congenital malformations may be asymptomatic.

Most cases of congenital abnormalities are identified during the first visit to the veterinarian after the pet has been purchased. On examination, a loud murmur often accompanied by a *precordial thrill* (a vibration of the chest wall) may be heard. With some defects, the clinician may observe pulse abnormalities, cyanosis, jugular pulses, or abdominal distension. Laboratory test results may all be normal. Radiography may suggest cardiac disease in some animals; however, echocardiography can provide an accurate diagnosis of the defect.

Causes of congenital heart disease include genetic, environmental, infectious, nutritional, and drug-related factors. More is understood of the genetic factors than the other causes. Studies suggest the defects are polygenetic in nature and that they might be difficult to eliminate entirely from a specific breed.

This section discusses the most commonly seen congenital defects. See additional cardiology texts for more detailed descriptions of each defect.

Patent Ductus Arteriosus

Failure of the ductus arteriosus to close after parturition results in blood shunting from the systemic circulation to the pulmonary artery. Normally, the ductus carries blood from the pulmonary artery to the aorta during fetal development. The increase in oxygen tension in the blood at birth results in closure of the path in the first 12 to 14 hours of life. If the ductus remains open, blood will hyperperfuse the lung, and the left side of the heart will become volume overloaded (Fig. 1.4). The resulting cardiac murmur is often referred to as a "machinery murmur"; this type of murmur is heard best over the main pulmonary artery high on the left base.

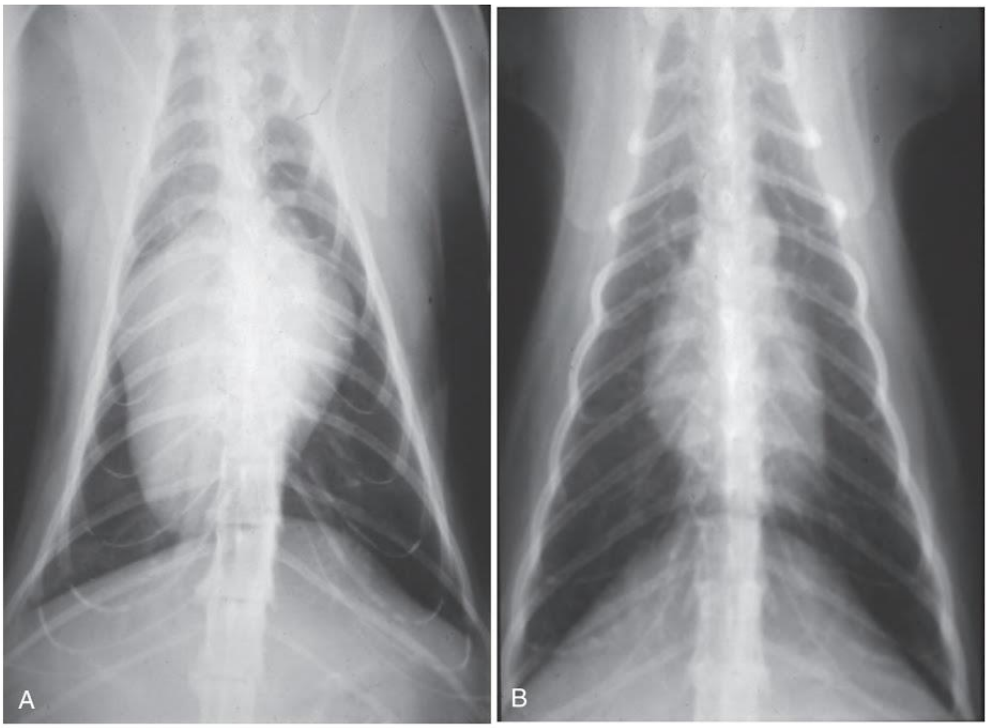

Fig. 1.4 (A) Hypertrophic cardiomyopathy (HCM) in the feline. (B) The apex of the heart is shifted to the right with HCM. (From August J. *Consultations in feline internal medicine.* 5th ed. St. Louis, MO: Saunders; 2005, by permission.)