Several underlying diseases/conditions (e.g., neoplasia; tachyarrhythmia; viral, bacterial, or fungal infection; electric shock; heartworm infection; abnormal thyroid function) and deficiencies (e.g., taurine and/or L-carnitine deficiency) can cause dilated cardiomyopathy (DCM) in dogs1-3; this article focuses on the idiopathic form. Idiopathic DCM—a disease of the heart muscle—is characterized by poor contractility and dilation of heart chambers that is not precipitated by systemic or other cardiac diseases.1,2 Although idiopathic DCM is one of the more common cardiac causes of morbidity and mortality in dogs, the mechanism of damage to the myocardium is poorly understood.1,2 Diagnosis and treatment with appropriate medications and nutritional support often improve quality of life for patients with idiopathic DCM; unfortunately, this disease is progressive and terminal, often ending in sudden death.1-3

Etiology and Pathophysiology

Idiopathic DCM is most commonly diagnosed in middle-aged, large- and giant-breed dogs such as Doberman pinschers, boxers, German shepherds, Newfoundlands, Dalmatians, and Labrador retrievers, although some small breeds have also been affected.1,2 Idiopathic DCM is prevalent in Doberman pinschers and boxers and has been recognized in some lines (via a familial link) of certain spaniel breeds (i.e., springer, American, and English cocker spaniels) and in bulldogs, which suggests a possible genetic basis in certain breeds.2,3 An autosomal dominance inheritance pattern has been found in Doberman pinschers; an X-linked pattern in Great Danes and an autosomal recessive pattern in Irish wolfhounds are suspected.4 A particularly lethal form of DCM has been documented in young Portuguese water dogs that have inherited an autosomal recessive trait linked to chromosome 8.2,4

Two distinct types of cellular damage to myocardial cells are found in patients with idiopathic DCM:5

- Fatty infiltrative-degenerative cellular damage is most common in boxers and Doberman pinschers.
- Attenuated, wavy fiber cellular damage is most common in giant, large, and medium-sized canine breeds.

As the initial damage to myocardial cells begins and progresses, the heart muscle becomes less effective because of poor contractility, causing a decrease in cardiac output and eventual enlargement of the heart chambers (i.e., remodeling).1,2,6 The major functional problem in DCM is systolic dysfunction, particularly of the left atrium and left ventricle; this is often followed by poor contractility in the right atrium and right ventricle.1,3 The muscles of the atria and ventricles become thin and more flaccid as the number of healthy myocardial cells decreases, resulting in a decrease of stroke volume and cardiac output during contractions.1,3 Cellular damage to the myocardium also affects the electrical signals in the heart and may cause ventricular and supraventricular arrhythmias.2,5,6

A decrease in cardiac output and subsequent dilation of the heart chambers activate the sympathetic nervous system and the neurohormonal compensatory mechanisms, which results in the release of cytokines and increases in heart rate and peripheral vascular resistance.1,4 This continuous systemic response to poor cardiac output contributes, in part, to the development of congestive heart failure (CHF) in affected patients, and cytokines damage the remaining healthy myocardial cells and may also adversely affect other body cells.2-4 Poor contractility, dilated heart chambers, and thin heart walls characterize the end stage of a number of pathologic processes involving myocardial cells.7 Most dogs do not show clinical signs during early stages of the disease; clinical signs are exhibited only when cardiac output becomes significantly reduced, at the onset of CHF, and/or when an arrhythmia develops.8,9 In some affected dogs that appear healthy, a diagnosis is not made until necropsy following sudden death.1

Key Points

- Dogs with dilated cardiomyopathy (DCM) may show subtle clinical signs in the early stages of the disease.
- Prompt diagnosis and treatment of DCM may prolong life and/or improve the quality of life for a patient with this progressive myocardial disease.
- Ongoing communication between the client and the veterinary staff increases the likelihood that health issues secondary to DCM will be promptly recognized and treated.

Clinical Signs and Diagnostics

The clinical signs of DCM vary, are often vague, and can be associated with a
number of other conditions. All the clinical signs result from left- or right-sided cardiac dysfunction and/or low cardiac output, but they vary according to the side of the heart that is most affected and the presence of arrhythmias or HF. A dog with early idiopathic DCM may present with subjective clinical signs, such as exercise intolerance, lethargy, weakness, or restless behaviors, but a physical examination may reveal no abnormalities, and the results of a blood chemistry profile may be within normal limits. As the disease advances, the clinical signs may include decreased appetite, weight loss, various arrhythmias, heart murmurs, prerenal azotemia, an increase in pulse rate, jugular vein distention (right-sided heart failure), syncope, clinical signs of HF, or sudden death. Syncope is characterized by temporary loss of consciousness, which is usually caused by inadequate blood supply to the brain but may also be due to a sudden drop in blood pressure; sustained ventricular arrhythmias; low blood levels of glucose, calcium, or sodium; seizure activity; metabolic disorders; or hypovolemia. Syncope may be associated with a coughing fit in an affected dog with marked left atrial enlargement; this is known as cough drop. A detailed history can help differentiate between collapse related to a cardiac condition versus a seizure episode. Research suggests that breeds associated with an increased risk for developing idiopathic DCM may benefit from annual Holter monitoring and annual echocardiography to diagnose occult DCM or other cardiac diseases in the early stages.

A clinician who suspects a cardiac cause of any clinical sign or intends to evaluate the heart in a predisposed breed should perform survey radiography, a blood chemistry profile, vertebral heart scoring, and electrocardiography (ECG). Dogs with abnormal results suggesting cardiomegaly require echocardiography for a diagnosis to be made. A diagnosis of idiopathic DCM can be made when the clinical signs have been evaluated and/or an echocardiogram shows evidence of dilated heart chambers, and there are no other abnormalities that can contribute to remodeling (e.g., heartworm infection, valve disorders, pericardial effusion, hyper- or hypothyroidism, tumor, congenital heart defect). Accurate diagnosis requires that the technician use proper technique and patient care during radiography, ECG, and/or echocardiography.

Radiography
Lateral and ventrodorsal or dorsoventral radiographs should include the entire thorax and surrounding structures in proper alignment at peak inspiration. Because the heart shadow appears differently in the ventrodorsal and dorsoventral views, the same view should be used consistently according to the clinician's preference. The dorsoventral view is less stressful for cardiac patients with a deep chest, respiratory distress, or a serious arrhythmia. For best results, the radiographic settings should include a short exposure time, a high kilovoltage peak, and low milliamperes. Patients with anxiety, serious arrhythmia, or respiratory distress may require medication before radiography to prevent exacerbation of conduction disturbances or a decrease in oxygen saturation.

Electrocardiography
ECG provides information about heart rhythm and conduction patterns and is usually used with other diagnostic tools to make a diagnosis (FIGURE 1, FIGURE 2). The paper speed, leads used, calibration of equipment, and patient's body position should be consistent during ECG. A change in the patient's body position can affect waveform amplitudes, and improper lead placement may affect interpretation of the results. Artifacts may result from (1) electrical interference from other equipment or (2) movement, rapid respiration, or trembling of the patient. Gentle patient restraint is ideal; using calming techniques or holding the patient's mouth closed to reduce panting should produce an ECG tracing without distorted conduction patterns.

Figure 1. Electrocardiography of an 8-year-old, female mixed-breed dog with dilated cardiomyopathy.
Idiopathic Dilated Cardiomyopathy in Dogs

**Holter Monitoring**
A Holter monitor is a vest with modified chest leads that is usually worn for at least 24 hours to produce a digitized recording of cardiac electrical activity. A trained Holter technician must evaluate the data; therefore, Holter monitoring is available only at a limited number of veterinary centers. According to subjective observations by pet owners, some dogs are reluctant to move when wearing a Holter monitor. This may be problematic because a lack of physical activity may decrease the occurrence of arrhythmias that would otherwise be detected.

**Echocardiography**
To evaluate heart function using echocardiography, the following modes are used:

- **M mode** is a one-dimensional view that produces wavy lines to represent various phases of the cardiac cycle.
- **Two-dimensional mode** displays the depth and width of tissue from images obtained from various locations on the chest wall. This mode is used to document anatomic changes resulting from a disease or defect.
- **Doppler mode** records the direction and velocity of blood flow and the presence of turbulence. This mode requires expert technique and a clear understanding of cardiac function. Pulsed-wave, continuous wave, and color-flow mapping can provide more precise information about stroke volume.

For echocardiography, the patient is shaved on both sides of the thorax over the heart region and placed in lateral recumbency on a table with a hole (FIGURE 3) that allows the operator to place a transducer on the chest wall (FIGURE 4). The procedure is usually well tolerated by patients, but it may help to have the client present to relax the patient during the procedure (FIGURE 5).

**Associated Pathology**
Myocardial cell damage may affect impulse conduction, which may result in arrhythmias, abnormalities in the sinoatrial and/or atrioventricular node, or complete heart block or bundle branch block. Doberman pinschers and boxers may have conduction disturbances consistent with dilation of the atrium or ventricles before these changes are evident on an echocardiogram. Prolonged ventricular arrhythmias can cause syncope and may be seen in advanced cases of DCM. A veterinary cardiologist should be consulted before initiating therapy for conduction disturbances in dogs with DCM.

The development of secondary heart valve insufficiency, hypertension, or CHF is common in the later stages of idiopathic DCM, and the presenting signs depend on the side of the heart that is most affected. In advanced cases of DCM, patients with...
Glossary

\( \alpha_2 \)-Agonists—a sedative hypnotic class of drugs that includes clonidine, rauwolfia, detomidine, dexametomidine, xylazine, and medetomidine

Artifact—in ECG, an electrical impulse that originates from somewhere other than the heart and causes spikes on the monitor

Cardiac cachexia—gradual loss of lean body mass (muscle); a number of factors contribute to this condition in dogs

Cardiac output—the volume of blood pumped by the left ventricle into the aorta each minute; it is affected by heart rate, myocardial contractility, metabolic conditions, and other factors; assessing the heart rate, blood pressure, and capillary refill time can help detect low or high cardiac output

Cardiomyopathy—a general term for heart disease in which the heart chambers are dilated, thickened, and/or stiffened

Cytokines—regulatory proteins produced by the immune system that trigger inflammation and respond to infection, stress, hormones, inflammation, other chemical mediators, etc.

Hemolysis—free hemoglobin in the plasma, usually the result of ruptured red blood cells during collection; however, pathologic processes can destroy red blood cells, leading to hemolysis

Icterus—the presence of bilirubin in the plasma/serum, indicated by a yellow color, the intensity of which is compared with a standard solution and graded

Inotropy—the force of a muscle contraction; positive inotropic drugs improve the strength of a contraction

Lipemia—a milky appearance indicating the presence of lipids in the blood; this is normal after eating

Myocardial contractility—the quality that enables heart muscle to contract involuntarily and continue beating in response to sympathetic (stimulated) and parasympathetic (inhibited) systems

Neurohormones—hormones that are secreted by neural cells in response to stimuli from the nervous system; in cardiomyopathy, these hormones are secreted in response to poor cardiac output

Opioids—synthetic narcotics such as fentanyl, hydromorphone, morphine, butorphanol, and buprenorphine

Remodeling—normal heart muscle stretching (dilating) to compensate for damaged myocardium

Right axis shift—seen on an ECG when more electrical forces are moving to the right than is normal (deviation to the right >+103 in dogs) due to right ventricular enlargement; the depths of Q and S waves are considered along with the height and axis of the R wave and the ST segment when ventricular enlargement is evaluated

Stroke volume—the amount of blood that the heart’s ventricles pump out in one beat, measured in milliliters per minute

Syncope—brief loss of consciousness leading to collapse; a clinical sign that may be due to a number of underlying disorders or diseases; clients may mistake syncope for a seizure

Vertebral heart score—a system that indexes heart size to body size using midthoracic vertebra

CHF present with clinical signs of left-sided failure (pulmonary edema and congestion in the lungs), right-sided failure (ascites, pleural effusion, and, possibly, edema in the limbs), or biventricular failure (clinical signs of left- and right-sided failure).1,3,6

Special Concerns—Anesthesia and Analgesia

It is important for veterinary technicians to understand the cardiovascular effects not only of pain but also of pain medications and/or anesthesia in dogs with heart disease. Veterinary technicians may be required to administer pain medications to dogs with DCM. In each patient, the disease state varies (e.g., early or late DCM, concurrent arrhythmias, CHF), and some patients may have coexisting systemic conditions, which increase the risk for morbidity and mortality when considerable pain is present or surgery is required.2,6–8 Pain has a significant effect on the cardiovascular system, lung function, and the gastrointestinal system; therefore, adequate analgesia should never be withheld from a cardiac patient.8 Opioids alone or in combination with benzodiazepines as well as local anesthetics are well tolerated in most dogs with DCM; the veterinarian will likely choose the opioid and the route of administration according to the intensity and duration of pain.8 Acceptable anesthetic induction agents include etomidate or ketamine combined with diazepam (to minimize the cardiovascular effects of ketamine); maintenance agents include isoflurane or sevoflurane.6 Drugs that are not recommended for use in affected dogs include \( \alpha_2 \)-agonists, thiobarbiturates, acepromazine, and halothane; anticholinergics should also be avoided, except when vagally mediated bradycardia is contributing to hypotension.5,6 For anesthesia, consider the following recommendations:

- Intravenous fluids (crystalloids or colloids) may be necessary; volume overload should be avoided by using lower-than-normal infusion rates6,8
- Current cardiac function should be evaluated using ECG, echocardiography, blood pressure evaluation, and a blood chemistry profile before induction of anesthesia2,8
- Preoxygenation via a mask is recommended only if it does not add to the patient’s stress or excitement6,8
- Mask induction may be unacceptable in cardiac patients because it can add to a patient’s stress or excitement and can require a large volume of gas8
- For adjusting dosages, the veterinarian should use a balanced technique by combining a low dosage of a premedication drug with an induction agent and titrating to effect7
- Intraoperative electronic monitoring should include ECG, blood pressure monitoring, end-tidal carbon dioxide monitoring, and pulse oximetry2,8
The cardiovascular system may be directly affected by anesthetics and analgesics, and dogs with DCM have a reduced cardiac output, which affects absorption of medications given by any route.\textsuperscript{2,8} 

**Treatment**

Treatment of dogs with idiopathic DCM involves the use of medications to increase cardiac output, normalize blood pressure, treat arrhythmias and/or CHF, and lessen the effect of neurohormones.\textsuperscript{1,6,7} Treatment is based on the clinical signs and the diagnostic test results of individual patients, but all dogs with a diagnosis of idiopathic DCM should be treated with positive inotropic agents.\textsuperscript{2,8} Positive inotropic drugs improve the strength of heart contractions, thereby improving cardiac output.\textsuperscript{1,3} Whether to administer an angiotensin-converting enzyme inhibitor or other medication tailored to a patient’s needs is determined by a clinician after evaluation of test results, breed influence, age, related conditions, and underlying organ dysfunction.\textsuperscript{1,6,8} Nutritional therapy, including L-carnitine and taurine supplementation, is used as adjunct therapy in some dogs with idiopathic DCM.\textsuperscript{2,9} 

L-carnitine is important for fat utilization and energy metabolism in myocardial cells, and carnitine-responsive DCM has been identified in Doberman pinschers and boxers.\textsuperscript{1,2,9} L-carnitine supplementation is recommended at a rate of 50 to 100 mg/kg q8h, but it can be cost prohibitive and patients may benefit from a lower dose.\textsuperscript{2,9} Veterinary-prescribed diets are formulated to meet the needs of dogs with cardiovascular disease. These diets are low in sodium and may contain L-carnitine, taurine, omega-3 fatty acids, magnesium, and potassium. Potassium and magnesium supplementation may be considered in patients receiving diuretic therapy for CHF if the diet is not adequate.\textsuperscript{9} Cardiac cachexia may be seen in patients with CHF,\textsuperscript{2,4} so these patients should receive a diet that provides the nutrients required to compensate for an altered metabolism. In patients with DCM, cardiac cells undergo oxidative stress and the energy requirements of myocardial cells are increased.\textsuperscript{1,9} Omega-3 fatty acid supplementation may reduce oxidative stress.\textsuperscript{1,9} 

**Conclusion**

Dogs with vague clinical signs associated with cardiac disease should undergo a physical examination and baseline ECG and/or survey radiography of the thorax. For all patients with DCM, the patient record should be clearly flagged and a return visit scheduled to allow a detailed discussion about nutrition, current or new clinical signs, and medications and their administration. A protocol should be developed and readily available in case a patient with DCM requires analgesia and/or anesthesia. Veterinary technicians must be (1) diligent in understanding the effects of pain and the systemic effects of medications for treating cardiac patients and (2) mindful of these effects during diagnostic procedures. This applies to caring for any patient with an underlying health condition, but errors in judgment can lead to more serious consequences in patients with an unhealthy cardiovascular system. Client education is vital because DCM progresses despite treatment. Clients must understand that the available medications only manage the clinical signs; there is no cure or prevention for idiopathic DCM. Nutritional support may also have a role, but the pathologic process in the myocardium continues despite the best efforts of the veterinary team.\textsuperscript{1,2,6} 

**Recommended Reading**


**References**

Idiopathic Dilated Cardiomyopathy in Dogs

1. The major functional deficit in early DCM is a result of
   a. diastolic dysfunction of the right and left ventricles.
   b. systolic dysfunction of the right and left ventricles.
   c. a decrease in stroke volume.
   d. systolic dysfunction of the left ventricle and left atrium.

2. In DCM, a systemic neurohormonal response occurs primarily because of
   a. a decrease in cardiac output.
   b. the onset of CHF.
   c. ventricular arrhythmias.
   d. biventricular failure.

3. ________ are common clinical signs in dogs in the early stages of DCM.
   a. Pulmonary edema and ascites
   b. Syncope and bradycardia
   c. Exercise intolerance and weakness
   d. Pleural effusion and peripheral edema

4. Positive inotropic drugs are used to treat DCM because they
   a. inhibit the release of cytokines.
   b. improve contractility of the myocardium.
   c. improve venous return.
   d. treat hypertension.

5. Syncope in dogs with DCM can be associated with
   a. significant left atrial enlargement.
   b. sustained ventricular arrhythmias.
   c. sudden decrease in blood pressure.
   d. all of the above

6. Which statement about idiopathic DCM is false?
   a. Conduction disturbances may occur before dilation of heart chambers is evident on an echocardiogram.
   b. A decrease in cardiac output causes enlargement of the heart chambers.
   c. Dilation of the heart chambers decreases the heart rate.
   d. Most dogs remain asymptomatic in the early stages.

7. Which medications tend to be well tolerated in most patients with DCM?
   a. opioids, etomidate, ketamine, and benzodiazepines
   b. etomidate, ketamine, halothane, and α₂-agonists
   c. opioids, α₂-agonists, ketamine, and local anesthetics
   d. acepromazine, ketamine, opioids, and local anesthetics

8. The neurohormonal compensatory mechanisms that accompany poor cardiac output
   a. do not contribute to the onset of CHF.
   b. are not linked to syncope associated with a coughing fit.
   c. do not lead to damage of healthy myocardial cells.
   d. do not affect heart rate and peripheral vascular resistance.

9. Reduced cardiac output in a patient with DCM
   a. affects the absorption of medications given by any route.
   b. may not cause significant clinical signs.
   c. contributes to remodeling of the heart chambers and the release of cytokines.
   d. all of the above

10. Supplementation using l-carnitine
    a. may slow the progression of myocardial cell damage.
    b. may improve energy metabolism in myocardial cells.
    c. provides no benefit to Doberman pinschers and boxers.
    d. may help to prevent cardiac cachexia.