

Dilated Cardiomyopathy: a daunting disease of the heart

by D. Caroline Coile, Ph.D.

Baron, a fine male Doberman Pinscher in peak condition, jumped into the back seat of the car after a walk in the park and fell down. He was dead. Sasha, an apparently healthy Boxer, appeared to be sleeping in the back yard. She never woke up. Buffo, a young Cocker Spaniel, started coughing one day and only got worse. His owners suspected kennel cough; his veterinarian knew better. Buffo like Baron and Sasha, had cardiomyopathy.

Dilated cardiomyopathy (DCM) is a progressive disease in which the muscles of the heart lose their contractility. In many cases, dogs do not display symptoms until the condition has advanced significantly. It is one of the most common of the serious acquired cardiac diseases in dogs, third only to degenerative valve disease and heartworm as a cause of heart-related death in canines.

The following will examine exactly how DCM stresses the dog's heart, the breeds that are most at risk, the diagnostic procedures and treatment options and some of the current research working to prevent this disorder and improve the lives of affected dogs.

Hard on the Heart

Canine DCM is characterized by weakened and stretched heart muscles that cause the heart to lose its ability to pump blood efficiently, in turn causing blood pressure to drop. The body's self-regulating mechanisms act to raise the blood pressure by constricting small blood vessels in a process called vasoconstriction. The body also releases chemicals into the bloodstream that stimulate the heart to increase both its rate and contractility in order to increase output. This combination of compensatory mechanisms allows the body's blood pressure to remain within normal range. Unfortunately, this is only a temporary fix.

Within one to three days of this compensation, the heart undergoes changes that make it less susceptible to the circulating chemicals, and output again decreases, although usually not to the initial precompensatory level. The body then seeks additional ways to increase blood pressure. One of them is by increasing the total amount of blood by retaining sodium via the kidneys and stimulating the dog to drink more water and eat more salt. These factors increase blood volume by increasing the body's retention of fluids. Again, this is a temporary fix that eventually becomes problematic.

With the increased blood volume filling the chambers of the heart, the walls of the heart stretch to accommodate it. Initially this helps the heart to eject more blood with each contraction, but as the heart muscles continue to weaken and the body continues to compensate, the chamber walls are

stretched more and more. At some point the heart reaches its limit and can expand no more. Although all four chambers of the heart may be affected, chambers on the left side (which primarily are responsible for pumping blood throughout the body) often are affected more dramatically.

At this point, the heart muscles cannot eject the blood with sufficient force, and some of the fluid is pushed back into the heart, and eventually the lungs, with each beat. This ultimately leads to fluid in the lungs, or pulmonary edema. Pulmonary edema causes the dog to cough and have difficulty catching its breath, both classic signs of leftsided heart failure. The nervous system's constant stimulation of the heart to increase output eventually leads to an increased oxygen demand by the heart and ultimately to erratic heartbeats and the destruction of the heart muscle.

These changes take a long time (probably months to years), and during most of this time the dog will be symptomfree. Only when the compensatory mechanisms no longer can function adequately does the dog exhibit symptoms of heart failure. At this point the disease actually is quite progressed, and survival time may not be long. So, although cardiomyopathy may appear to have a sudden onset, in reality it has a slow, but insidious, course.

Not all dogs with cardiomyopathy follow this course, however. For some dogs, and particularly for some breeds, the most salient feature of DCM is an erratic heartbeat, or cardiac arrhythmia. The most prominent arrhythmia of cardiomyopathy is a premature ventricular contraction. When

these PVCs occur only sporadically the dog probably will not have any symptoms of a heart problem. However, if a series of PVCs occur together, the blood is not pumped through the circulatory system properly, and the dog may be weak and even may collapse or faint.

If the heart is able to regain its normal rhythm, the dog will recover within a few seconds or minutes, but if the run of PVCs continues, the condition is termed ventricular tachycardia and can lead to ventricular fibrillation. When a dog is in Vfib no blood is being pumped, and the animal will die unless a regular rhythm can be reestablished.

The Symptoms

Cardiomyopathy has a range of symptoms, some of which are more prevalent in some breeds than others. Respiratory problems, including coughing and labored or rapid breathing, are typical symptoms in dogs with leftsided heart failure resulting from DCM. Some dogs will experience weight loss, general debilitation, abdominal distention or cold extremities. In breeds such as the Doberman Pinscher or Boxer, in which ventricular arrhythmias are a common manifestation of DCM, dogs may have a history of fainting or episodic weakness. Often symptoms appear to develop quite suddenly. This especially is true in dogs that normally are not very active. But, in fact, the condition has been developing slowly (probably for at least six months to 18 months), but only near its final stages is it severe enough to affect a normally

inactive dog noticeably.

In active working or hunting dogs, the disease usually is noticed earlier because the dog gradually loses its ability to exercise. In some cases the dog dies suddenly without any overt warning, presumably as a result of ventricular arrhythmia. Sudden death is the first and only sign of DCM in almost one fifth of Doberman Pinschers with the condition. Dilated cardiomyopathy mostly is a disease of large and giant breeds, but it also strikes certain breeds at a much higher rate than other breeds of the same size. The Scottish Deerhound has the highest percentage of affected dogs seen at veterinary hospitals, followed by the Doberman Pinscher, Irish Wolfhound, Great Dane, Boxer, Saint Bernard, Afghan Hound, Newfoundland, Old English Sheepdog, English Cocker Spaniel, English Springer Spaniel, American Cocker Spaniel, Labrador Retriever and Golden Retriever.¹ Because of the popularity of the Doberman Pinscher, the Great Dane and the Boxer, however, veterinarians treat more dogs of these breeds for DCM than the other at-risk breeds.

DCM (or at least congestive heart failure resulting from DCM) is seen roughly twice as often in males than females. Most dogs presented with symptoms are between 4 and 10 years old, and the incidence increases with age. A few dogs are diagnosed with DCM before reaching 1 year of age, however. Some Portuguese Water Dog puppies develop and die from DCM at only a few weeks of age.²

The Diagnostic Procedure

Given the life and death possibilities of cardiomyopathy, timely diagnosis is important so the dog's quality and length of life can be improved as much as possible. Preliminary tests can be performed by a general veterinarian, whereas some tests require more specialized equipment and interpretation and ideally should be performed by a specialist in veterinary cardiology.

Blood and urine tests usually are in the normal range, although some values may reflect the effects of low cardiac output. A radiograph (Xray) of the heart usually will show some degree of cardiac enlargement (called cardiomegaly), but this is less evident in Boxers and Dobermans than it is in giant breeds and Cocker Spaniels.

In about half the dogs with DCM, listening to the heart (auscultation) will reveal soft to moderate heart murmurs. The heart rate usually is rapid, often more than 200 beats per minute, in dogs with heart failure. In dogs with PVCs, the erratic heartbeat sometimes can be detected with auscultation in conjunction with feeling the pulse. Normally, for every beat that is heard, a pulse should be felt. When a PVC occurs, however, it sounds like there is a missed or extra beat that is not accompanied by a pulse.

PVCs are best detected with electrocardiography, a painless procedure performed on an awake dog. Most veterinarians can run an electrocardiogram and instantly transmit the data to a specialist for analysis. Most dogs with DCM have some

abnormalities evident in their ECGs. The majority of giant breed dogs with DCM will have atrial fibrillation, a disturbance in the normal rhythm of the ECG. Many dogs, especially Dobermans and Boxers, with DCM will have PVCs and ventricular tachycardia.

Note that although PVCs are suggestive of DCM, the presence of a few PVCs also could be due to other factors and is cause for a thorough check. The problem with using an ECG to detect PVCs is that unless the PVCs are frequent, they easily can be missed during the relatively short duration of a typical office visit ECG, which often runs for only about 30 seconds. For breeds in which PVCs are the major symptom of DCM, 24hour Holter monitoring is a vital diagnostic tool. The Holter monitor basically is a portable ECG that the dog wears as a harness. It records the ECG as the dog goes about its normal routine at home over a 24hour period, covering 90,000 to 110,000 heartbeats. Not only can it more reliably detect whether a dog has any PVCs, but also their frequency and whether they tend to cluster together.

In the Boxer, the Holter monitor is considered absolutely essential for diagnosis,³ and many people advocate using the instrument as a means of screening potential breeding stock. Most Boxers that eventually die of DCM have hundreds to thousands of PVCs in a 24hour period, as well as clusters of ventricular tachycardia. Interestingly, the incidence of arrhythmias in Boxers seems to have a circadian pattern, peaking in early morning and late afternoon,⁴ a pattern also seen in human DCM patients.

In most other breeds the diagnostic tool of choice is the echocardiogram, in which the heart is examined with ultrasound. This, too, is a painless procedure performed on an awake dog. The examiner places a handheld microphonelike instrument called a transducer on the dog's chest. The transducer emits high-frequency sound waves that are reflected back to it from the heart. These sound waves then are translated into a moving image, showing the internal structures of the heart, the heart valves, the size of the heart chambers, the thickness of the heart muscle and how well the heart is contracting. Dogs with DCM characteristically have dilated heart chambers (especially the left atrium and left ventricle), a thin heart muscle and weak heart contractions with low cardiac contractility. The echocardiogram also can exclude other possible heart defects, although a dog with DCM also may have mitral valve insufficiency because the dilated chambers result in the mitral valve leaflets becoming separated and misaligned. The echocardiogram provides the cardiologist with a twodimensional picture of the heart and can be an invaluable aid in estimating how the blood might be flowing through the heart. Doppler ultrasound enables the cardiologist to actually envision the direction and speed of blood flowing through the heart, aiding in pinpointing areas of blood turbidity and sources of heart murmurs.

Uncovering Etiologies

Although science can help us determine if DCM is present in a certain animal, in most cases the cause of the condition is unknown. Examination of the components of the heart muscle (myocardium) of affected animals has not yet identified any consistent primary abnormalities, but researchers continue to look at promising new aspects.^{5,6,7}

It is possible that DCM is not one disease with a single cause but rather a group of diseases that ultimately causes similar cardiac abnormalities. Most cases of DCM are termed idiopathic (unknown cause) or primary DCM. The myocardium can be affected adversely by a variety of problems, however, some of which can give rise to DCM. These "secondary" cardiomyopathies may result from nutritional deficiencies, metabolic disorders, cardiac toxins, inflammations, trauma, tumors or infections.⁸

Nutritional Deficiencies

Taurine, an essential amino acid found in meat, is important in many bodily processes, including normal heart function. Some dogs with DCM have abnormally low amounts of taurine in their blood plasma. This seems to be especially prevalent in Cocker Spaniels, Golden Retrievers and breeds not typically considered at risk for DCM. Taurine deficiency also has been associated with DCM in dogs fed an exclusively vegetarian diet and in some Dalmatians fed a prescription diet that has since added higher levels of taurine.⁹

Taurine deficiency was found to be a common cause of DCM in cats in the late 1980s. Increasing the amount of taurine in commercial cat food enormously decreased the frequency of this disorder. Unlike cats, dogs are not obligate carnivores, meaning they usually can maintain a normal level of taurine in their blood plasma without having to include it in their diet. In most dogs with DCM, taurine deficiency does not seem to be the culprit.

Lcarnitine is found in foods of animal origin, especially red meat. It is essential for the mitochondrial transport of fatty acids, which are in turn the most important energy sources for the heart muscles. Lcarnitine also helps rid the mitochondria of some potentially toxic metabolites. If these functions are compromised as a result of inadequate levels of Lcarnitine, the end result is poor myocardial performance. Most dogs probably get an adequate amount of Lcarnitine in their diets, but it is possible that some dogs have a metabolic defect that prevents it from being assimilated into the heart muscle normally. Unfortunately, measurements of Lcarnitine in the blood don't accurately reflect the amount of Lcarnitine in the heart muscle, and measuring the Lcarnitine in the heart muscle requires a biopsy. Some dogs (in fact, most Dobermans^o) with DCM have lowered Lcarnitine levels in the heart muscle, leading to speculation that Lcarnitine deficiency might cause DCM. It is just as likely, however, that the decreased myocardial Lcarnitine is not a cause of DCM, but rather a secondary effect of some other abnormality. Low levels of Lcarnitine can result from a condition called

cystinuria, in which dogs have impaired reabsorption of cystine and other amino acids. Cystinuric dogs with DCM may be suffering from L-carnitine deficiency. As with taurine deficiency, though, L-carnitine deficiency more likely is the exception rather than the rule.

Vitamin E and selenium deficiencies have been associated with myocardial disease in some young dogs, but they do not appear to be common causes.

Toxins

Anthracycline antibiotics such as the drug doxorubicin (Adriamycin®) are used in the treatment of some cancers. In some dogs this drug results in arrhythmias, decreased cardiac output and degeneration of parts of the heart muscle. This is more likely to occur in those breeds in which a high incidence of idiopathic DCM is found, in dogs with preexisting cardiac abnormalities and at high peak-level dosages of medication. Other myocardial toxins include (but are not limited to) ethyl alcohol (which is given intravenously to treat dogs that have ingested ethylene glycol antifreeze), cocaine, and plant toxins such as buttercups, lily of the valley and foxglove.

Viruses

When parvovirus came upon the dog world in the late 1970s, survivors of puppyhood infection often developed DCM by the time they reached a year of age. Distemper in young puppies also can leave survivors with residual damage to the heart

muscle, but neither disease currently is a common cause of DCM. Other viruses can damage the heart in both humans and dogs, leading to DCM, although the incidence in dogs is unknown.

Parasites

Although heartworms are the most widespread parasites that can inhabit the heart, in the southern United States the protozoan parasite *Trypanosoma cruzi* causes Chagas' disease, the symptoms of which can include cardiac enlargement, arrhythmia and sudden death. Affected dogs usually are so ill with other diffuse symptoms that they would not be considered typical cases of DCM, however. Dogs that do survive may develop chronic symptoms of DCM.

Heredity

The greatly increased incidence in some breeds, and in some lines within breeds, is highly suggestive of hereditary influences in at least some cases of DCM. Some preliminary evidence consistent with an autosomal dominant mode of inheritance (in which the presence of only one gene for DCM is needed for the disease to develop) has been reported in a family of Boxers.²

Although most cases of DCM do not have an obvious genetic component, some human families have been found with a pattern that matches sexlinked inheritance,³ and some other families with DCM have been found to have mutations that

cause various types of abnormalities in structural proteins. At least in humans, hereditary DCM can result from aberrant genes at more than one location on the chromosomes.⁴ Similarly, DCM in dogs could reflect different genetic etiologies among breeds and perhaps even within single breeds.

Treatment & Therapies

Unfortunately, except in the very few cases that are caused by nutritional deficiencies, DCM cannot be prevented or cured. Most dogs live only six to 12 months after the onset of symptoms, although some live several years. Some others, however, live only a few weeks after diagnosis. The prognosis for longterm survival particularly is poor for Dobermans, which often die within three to six months.

Because DCM differs in severity and symptoms among dogs and because dogs respond differently to medications, each therapy must be tailored to the individual patient. Current therapies can improve and prolong a dog's quality of life somewhat, but they have their limits. They center on alleviating the congestive state, reducing cardiac stress by lowering the blood pressure, improving the heart's contractility and reducing arrhythmias.

Supplemental therapies center on replacing substances that might be deficient, such as L-carnitine or taurine. Dogs with advanced symptoms of congestive heart failure should be placed on a low-sodium diet to reduce the need for higher

dosages of diuretics. Emergency therapy is beyond the scope of this article; the drugs presented here are the ones most commonly used for the chronic treatment of DCM.

Drugs

Diuretics, most notably furosemide (Lasix®), help combat the accumulation of fluid in or around the lungs that is symptomatic of congestive heart failure. They do this by decreasing the excessive blood volume that can be characteristic of patients with congestive heart failure. Furosemide generally is safe, but it can cause dehydration along with excessive thirst and urination and occasionally can have other undesirable side effects. Thus, it is important to use the lowest effective dosage. Despite side effects, furosemide probably is the single most important drug for treating symptoms of congestive heart failure.

Digitalis glycosides, such as digoxin (Cardoxin®, Lanoxin®) and digitoxin, may help the heart contract better and may help slow the heart rate in dogs with atrial fibrillation. Digoxin more often is used in veterinary medicine, although digitoxin may be a better choice in dogs with compromised kidney function (because digoxin is excreted mostly by way of the kidneys, whereas digitoxin is excreted by the liver). Many dogs do not seem to respond to treatment with these drugs. In a study of 22 dogs with DCM, only five responded favorably to digoxin; these five, however, did live significantly longer.¹⁵

Digoxin has a narrow range over which it exerts therapeutic effects before causing toxicity. Common signs of toxicity include appetite loss, vomiting, diarrhea and lethargy. Blood tests can be used to monitor circulating levels of digoxin and determine the appropriate dosage.

Vasodilators increase the diameter of small blood vessels and help prevent the damage longterm vasoconstriction ultimately inflicts on the heart. By reducing resistance to the outflow of blood from the heart, they increase the heart's output and decrease edema. Care must be taken that the blood pressure does not become too low, however. Nitroglycerin cream or transdermal patches (both vasodilators) sometimes are prescribed for dogs with DCM, although their effectiveness is not considered profound.

Angiotensinconverting enzyme (ACE) inhibitors, such as enalapril (EnacardTM, Vasotec[®]), also increase vasodilation. When used in conjunction with furosemide and digoxin, ACE inhibitors gradually can improve exercise tolerance and reduce the symptoms of heart failure and even may help prolong life. In a comparison of dogs with DCM given enalapril with those given a placebo, the enalapril-treated dogs survived an average of 143 days before treatment failure compared with 57 days in the placebo-treated dogs.¹⁶

Because enalapril occasionally can interact with the kidneys, kidney function should be monitored regularly with a blood test during the course of treatment. Another ACE inhibitor, benazepril (Lotensin[®]), is not officially approved for use in dogs but may be a better choice for dogs with concurrent

kidney problems.

Sympathomimetic amines, such as dobutamine, bind to certain receptors of the heart to increase its contractility and cardiac output. Dobutamine is not used that often in veterinary medicine because it must be infused over several hours under conditions of careful monitoring, and it may increase ventricular arrhythmias. Its effects, however, often referred to as a "dobutamine holiday," may last a week or more. Human patients who receive a threeday dobutamine infusion every few weeks have been reported to have improved cardiac function, but this has not been studied in dogs and would be impractical for most chronic situations. Antiarrhythmics also may be beneficial in reducing the ventricular rate. Digoxin can reduce the rate to some extent, but other drugs, such as propranolol, are more effective. Other antiarrhythmics, such as procainamide, can reduce the number of PVCs in affected dogs, but dogs still can die suddenly when taking these drugs. Some preliminary studies suggest that sotalol (Betapace®), a powerful antiarrhythmic, has had favorable results in some Boxers with DCM, but because of possible side effects, dogs must be monitored carefully. Another antiarrhythmic drug with encouraging results is mexiletine.¹⁷

Supplements

Lcarnitine will help the very small group of dogs that have DCM as a result of L-carnitine deficiency. Unfortunately,

these dogs cannot be identified easily. Some dogs treated with large doses of oral L-carnitine supplements have increased their levels of circulating L-carnitine and have seemed to improve subjectively. Some of these have shown improvement based upon echocardiograms, but L-carnitine supplementation does not seem to improve cardiac arrhythmias, nor does it seem to prevent sudden death.

L-carnitine supplementation does not do any harm, but it is expensive. It is more economical (approximately 25 cents per gram) to buy it in bulk.¹⁸

Taurine may be beneficial to most Cocker Spaniels and possibly Golden Retrievers with DCM. In one study, a direct relationship was found between DCM and low plasma taurine levels in 11 American Cocker Spaniels that were in congestive heart failure due to DCM. After several months of supplementation with taurine (or taurine and L-carnitine), most of the dogs improved and eventually were able to be taken off their heart medications.¹⁹ Taurine also can be bought in bulk at a reduced cost (approximately 2.5 cents per gram).²⁰

Coenzyme Q10 supplementation has produced significant improvements in humans with DCM by generating energy in the mitochondria of the heart muscles.²¹ It also may be beneficial in dogs. Price may vary, but 100 50mg pills cost about \$20.

Fish oil has been helpful in reducing body wasting in some Dobermans with DCM.

Consult your veterinarian about which supplements and at what dosages might benefit your dog.

Surgery

Humans with DCM routinely undergo heart transplants. Although this procedure also is performed routinely in laboratory dogs for experimental purposes, it is not available for pet dogs.

An experimental technique for human DCM patients is called dynamic cardioplasty, in which a part of the latissimus dorsi muscle is wrapped around the heart and induced to contract so that it acts somewhat like the heart muscle. Results so far in dogs have been somewhat disappointing. Other surgical techniques are being tried in humans and no doubt also will be attempted with dogs in the future.

Lifestyle

Besides instituting appropriate therapy and monitoring, owners of dogs with cardiomyopathy should observe certain precautions. Dogs with DCM should be kept relatively quiet and not allowed to exercise strenuously. Excessive exercise will do nothing to strengthen the heart muscles and, in fact, because it requires the heart to compensate, actually can hasten the damage to the heart muscles. In addition, exercise may bring on episodes of arrhythmia and fainting.

Once a dog shows symptoms of congestive heart failure, it should be placed on a low-sodium diet. Most commercial dog foods, unless formulated specifically for low-sodium content, have unacceptably high sodium levels for dogs with congestive heart failure. Some prescription dog foods, such as Hill's® k/

d® (which has 0.20 percent salt on a drymatter basis), Hill's h/d® (0.10 percent), Hill's Heathblend™ Canine Geriatric (0.15 percent), Hill's Canine Senior® (0.17 percent) and Purina® CNMVCV® (0.12 percent) have appropriate levels of sodium. A variety of homemade diets appropriate for dogs with heart disease also are available.²²

Dogs with cardiomyopathy should undergo anesthesia only when absolutely necessary and with extensive precautions. A recent study of Dobermans with occult (asymptomatic) DCM showed that anesthesia and surgery seemed to worsen arrhythmias and contractility. Because of the prevalence of occult cardiomyopathy in Dobermans, these researchers advocate that any Doberman scheduled for elective surgery first should be screened with echocardiography.

Unfortunately, the ideal anesthetic protocol for these dogs has yet to be determined.²³

Current Research

To improve upon and add to the list of therapy options and increase our knowledge of what causes DCM, studies are being conducted at several research centers. Because of DCM's varying nature among breeds, much of this research is breedspecific, although this does not negate the possibility of findings being applicable to other breeds. Several breed clubs support research for their breeds, and individuals also can support cardiomyopathy research.

Researchers at the University of Guelph in Ontario, Canada, led by Dr. Michael O'Grady, are following healthy Dobermans

throughout their lives with extensive testing to search for clues that will predict which ones will develop DCM. They are investigating the efficacy of various agents administered in the earliest stages of cardlomyopathy in stopping or slowing the progression of the disorder.

Researchers there also are attempting to pinpoint the cause of DCM in Dobermans and specifically are analyzing the abnormal enzymes suspected of having a role in the condition. A new study is being organized that will allow Dobermans from any locale to participate by means of annual Holter monitoring. Researchers ask owners to contact them before making the difficult decision of euthanasia and to consider allowing them to first perform an ultrasound, electrocardiogram, chest Xray and, following euthanasia, a heart biopsy. As difficult as such a decision would be, perhaps some consolation can be found in knowing that a cherished friend will leave a legacy of knowledge.

At Michigan State University, researchers are trying to produce a genetic test for cardiomyopathy in Dobermans. Dr. Fat Venta has led this research team since the tragic death of the former leader, Dr. Tracy Hammer, in the TWA Flight 800 crash. Donations toward this project, the Tracy Hammer Doberman Finscher Research Fund, can be made by sending a check and a note indicating it is for Venta's Doberman Finscher cardiomyopathy research program to the CVM Development Office, Attention: Patty Jacobs, director of development, A135 East Fee Hall, Michigan State University, East Lansing, MiCh. 488241316.

Also at Michigan State University, Dr. John Gerlach is examining possible viral contributions to cardiomyopathy in Salukis. Identification of a viral component could lead to methods of avoiding the spread of cardiomyopathy; if no such component is identified, breeders would need to turn more attention to prevention by selective breeding.

Dr. Kate Meurs of Ohio State University is recruiting Boxers for cardiac evaluation and collection of DNA samples. Samples from Boxers that are affected, unaffected but related to an affected animal, and unaffected and unrelated to an affected animal are being collected to search for genetic markers of DCM in the breed. In conjunction with Dr. Phil Fox of the Animal Medical Center, hearts of deceased Boxers with DCM are being examined with both microscopic and magnetic resonance imaging techniques in the search for clues about DCM's cause.

At Tufts University, Dr. Joseph Alroy is examining a type of cardiomyopathy that occurs in Portuguese Water Dogs in which young puppies are affected and die within six months. It is suspected that defective cytoskeletal proteins (proteins that maintain the structural integrity of the cell) might cause gradual deterioration of the heart muscle cells. By studying hearts of affected dogs at different stages of the disease, it is hoped that the molecular and genetic basis of DCM in these dogs eventually will be identified.

Many research projects dealing with human DCM may one day yield help for dogs with the condition. Among current investigations is the possibility that growth hormone may help

some people with DCM. In humans with the condition of acromegaly, an excessive amount of growth hormone results in abnormal thickening of the heart walls. Using this reasoning, researchers have given human growth hormones to DCM patients with abnormally thin heart walls and have reported that the heart walls indeed have grown thicker in response. This treatment still is far from providing an answer for dogs with DCM, however, because of several potentially severe side effects, high expense (hundreds or thousands of dollars) and the lack of commercially available canine growth hormones. These are only samples of ongoing research. Advances in treatment continue to be made, and perhaps one day DCM will be only a bad memory. Meanwhile owners of affected breeds must be on the lookout for signs of cardiomyopathy, and owners of affected dogs must seek treatment to make their pets as comfortable as possible and, like all dog owners, to make the very best of the far too short time we all have to share with our best friends.

About the Author

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