Boxer Cardiomyopathy

Boxer cardiomyopathy, as we know it, consists primarily of an electrical conduction disorder that causes the heart to beat erratically (to have an arrhythmia) some of the time. If the erratic beats occur infrequently and singly the dog will probably not have symptoms of heart disease. If the erratic beats occur in sequence, then weakness, collapse or sudden death may result. These arrhythmias may or may not be detected by listening to the heart with a stethoscope. Whether or not they are detected depends on the frequency of the abnormal rhythm. If they occur frequently they can easily be heard with a stethoscope.

The arrhythmia usually consists of VPCs (ventricular premature contractions) that are heard as an extra beat or a skipped beat that does not have a corresponding pulse. To identify these the listener must therefore have one hand on the stethoscope, holding it to the chest, and one hand feeling for a pulse (usually at the femoral artery on the inside of the hind leg). In the normally functioning heart there is a pulse for every beat that is heard.

When a VPC occurs a beat is heard through the stethoscope (and it sounds like a stutter as it is not in the normal rhythm sequence of the sinus beats) but there may not be a pulse to go with it. These VPCs have a characteristic pattern on an ECG and this is the way they are confirmed.

Often this is the first abnormality noted in a Boxer with cardiomyopathy. Usually the dog is having no symptoms of heart disease when a veterinarian notices these VPCs during a routine exam. If the frequency of these irregular beats increases the animal may suffer "fainting spells" called syncopal episodes. This happens because the abnormal beats do not pump blood effectively (no corresponding pulse) to the vital organs like a normal beat would and the brain becomes oxygen deprived while the abnormal beats are occurring. Usually when an animal faints they are having what is know as a run (several in a row) of VPCs. If the heart corrects itself the animal regains consciousness in a matter of seconds to minutes.

If the run of VPCs continues this is termed ventricular tachycardia and can lead to the development of ventricular fibrillation. Ventricular fibrillation is fatal if the rhythm is not converted. This ventricular fibrillation (V-fib) is the cause of sudden death in most Boxers with cardiomyopathy. There is no blood being effectively pumped through the body when the animal is in V-fib. Cardiomyopathy can also be responsible for sudden death associated with anesthesia. Now, just because a boxer has VPCs does not absolutely mean it has cardiomyopathy IF there is another disease process at work. I have seen animals with severe infection or cancer have VPCs that resolved completely once the infection was cleared or the malignancy removed. If, however, VPCs were seen in an otherwise healthy Boxer, one would have a high index of suspicion for cardiomyopathy because of the prevalence of the disease in the Boxer breed.

Some Boxers with cardiomyopathy will enter another phase of disease where the ventricles of the heart start to dilate. At this time it is unclear whether this is a progression of the electrical conduction disorder, a separate disease more like that seen in other large-breed dogs such as the Doberman pinscher, or a subset of Boxer CM that is not necessarily a progression of the previously arrhythmic dogs. With this condition the walls of the heart become thin, the heart muscle weakens and these animals show symptoms of heart failure such as coughing (from lung congestion) and/or fluid retention in the abdomen (ascites) depending on which side of the heart is most affected. In time, as the heart becomes very enlarged, it begins to be an inefficient pump and dogs so affected may require numerous medications to keep the heart functioning well enough to sustain life. Still, most Boxers affected with cardiomyopathy will ultimately die of their arrhythmia, not of congestive heart failure. The only way to definitively make the diagnosis of cardiomyopathy is to have a veterinary pathologist evaluate tissue samples from the heart muscle after death.

Since Boxer CM is a disease characterized primarily by arrhythmia, echocardiography (ultrasound of the heart) is not the method of choice to make a diagnosis of CM in this breed. An echocardiogram is useful to determine if the heart is functioning properly. It will also help detect and identify the source of any murmurs that may have been heard on auscultation with a stethoscope by allowing visualization of the heart valves and blood flow patterns through those valves. It can be used to rule out the inherited condition of sub-aortic stenosis (SAS), which is known to affect the Boxer and can also lead to sudden death. It can also show whether or not there is any enlargement of the heart chambers or any thinning (as seen in dilated cardiomyopathy) or thickening (as seen in hypertrophic cardiomyopathy) of the heart muscle walls. It is not a good tool for detecting an arrhythmia unless the arrhythmia is very frequent and is noticed during the echo exam. Most Boxers with CM will have normal echocardiograms unless they also have SAS or have the type of CM (progression, different disease or subset?) that causes dilation of the ventricles.

It has been shown that dogs on commercial diets have adequate amounts of L-Carnitine in their plasma and that 80% of dogs with cardiomyopathy that have a deficiency of L-Carnitine in the heart muscle have normal to increased L-Carnitine levels in their blood. Although there has been a correlation between two Boxer siblings with dilated cardiomyopathy and a response to supplementation with L-Carnitine, many more boxers have shown no improvement with supplemental L-Carnitine. The two sibling Boxers were found not to have a deficiency of Carnitine in their diet, but most likely had an inability to utilize the Carnitine present in their blood and to transport it into the heart cells where it must be actively concentrated so that it can be used for fatty-acid metabolism, generation of energy and detoxification of certain metabolic compounds. These dogs most likely had an inherited defect of the membrane transport of L-Carnitine.

While supplementation with L-Carnitine improved the heart contractility (strength of beating) of these dogs and caused a temporary improvement, it did not decrease their arrhythmias. One of these dogs eventually died due to ventricular arrhythmia, the other due to an apparent sudden onset of hypoadrenocorticism (Addison's disease). Both parents were also affected with CM but died before treatment with L-Carnitine could be evaluated. (Keene, 1991). We don't have any real evidence that L-carnitine will help dogs with cardiomyopathy. On the other hand, we don't have any evidence that supplementation with L-carnitine is harmful.