

Report on the Ohio State University study of Boxer Cardiomyopathy

October 21, 1999

Dear Dr. Oliver

I am writing this letter in response to your request for my opinion regarding the interpretation of Bolter monitoring results and the use of the Bolter monitor for screening boxers for inherited arrhythmias. I appreciate your patience. As you and I have discussed, this is a very controversial area and I am not sure I have enough information to clarify this area yet. I have confidence in stating two points. First, ventricular tachyarrhythmias are observed with frequency in the adult boxer population. In fact, of asymptomatic boxers that were presented for screening by Bolter monitoring in this study, 83% had some (between 1 and 50) ventricular premature complexes (VPCs); 66% had greater than 50 over a 24-hour period. (I have included the abstract of this data that we presented at the American College of Veterinary Internal Medicine meeting in June 1999.) This does not mean that ventricular premature complexes are a normal finding in the adult boxer, but rather, that many boxers carry the trait. This work was recently published in the journal of the Veterinary Internal Medicine Association. I have included the paper. Unfortunately, as with most research, the more you look at a problem, the more questions arise. And thus, the number of things that we do not know about this trait is amazing. For instance, just because a dog has the trait does not mean that it will ever show clinical signs from the trait. Why do some dogs die from sudden death, while other severely affected boxers live a long life? It does not appear to be the number of abnormal beats alone that influences clinical signs. We have observed boxers with 1200 VPCs that die of sudden death and others with 60,000 VPCs that come back year after year for a recheck without the development of clinical signs. Why do some boxers eventually progress to congestive heart failure? Can mildly affected individuals be used for breeding programs? Does the use of careful antiarrhythmic therapy make a difference? What is the cutoff point for affected status? If a dog has 150 VPCs in 1999, will it stay that way for the rest of its life? If you breed a dog with 150 VPCs will it only produce dogs with the same mild degree of the disease? (Maybe these would be OK to breed.) Is the dog with 10,000 VPCs necessarily more likely to die suddenly than the one with 330? If you successfully treat a dog with 10,000 VPCs so it lives longer, will the disease affect the heart muscle itself? Does treatment with a ventricular antiarrhythmic actually do anything? At this point there appear to be many

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more questions than answers! ! !

Thus, I urge caution in the interpretation of Bolter monitor screening. If 83% (or at the very least 66%) of the boxer population carries the trait for ventricular arrhythmias and we remove all of those dogs from the breeding population we will have very little left to use for the future of the breed. Reducing the gene pool so significantly will most likely increase the prevalence of other problems like cancer and subvalvular aortic stenosis in the breed. Since there are still so many unanswered questions about this trait, and the impact of removing all carriers of the trait from the population will be great, I beg for the cautious and judicious use of Bolter monitoring.

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