Letters to the Editor

Questions validity of study on periodontal disease and cardiovascular disease in dogs

This letter is in response to the article “Evaluation of the risk of endocarditis and other cardiovascular events on the basis of the severity of periodontal disease in dogs” published in the February 15, 2009, issue of the JAVMA. As veterinary cardiologists, we have substantial concerns about the methods, data, and conclusions in this report. Our biggest concern is the lack of description of how cardiac disease diagnoses were made in these dogs, other than via “…the judgment of each veterinarian as part of primary-care practice.” Accurate diagnosis of cardiac disease was crucial to this study, and it is imperative that the methods by which these cardiac diagnoses were made be clearly described to establish validity. Infective endocarditis can only be diagnosed on the basis of echocardiographic findings in conjunction with compatible clinical signs or results of bacterial culture of blood samples or on the basis of diagnostic necropsy findings. Thus, we would like to know how many of the 39 dogs in which endocarditis was diagnosed underwent echocardiography or diagnostic necropsy.

In addition, we question the accuracy of the diagnoses of cardiovascular disease made on other dogs in this study. More cases of hypertrophic cardiomyopathy (HCM) were reported than cases of endocarditis, and frequency of HCM was reported to be almost 38% of the frequency of dilated cardiomyopathy. However, HCM is reportedly rare in dogs and, in our experience, observed with far less frequency than bacterial endocarditis. In addition, there were only 208 cases of mitral insufficiency diagnosed in this population of 59,296 dogs (0.4%), which we would consider quite low, especially when compared with the 39 cases of endocarditis.

The Kaplan-Meier curves presented in the manuscript are also inconsistent with previous data on cardiac disease in dogs. According to Figure 1, > 99% of all dogs with endocarditis were alive after 1,200 days. By contrast, endocarditis reportedly is difficult to treat in dogs and is typically associated with a poor prognosis. Similarly, > 99% of dogs with mitral valve insufficiency were reportedly alive after 1,100 days. This is in stark opposition to our clinical experience and a recent large study in dogs indicating median survival time for dogs with mitral valve disease of approximately 585 days.

Although an extremely large amount of data was analyzed for this study, we are concerned about the accuracy of that data and the apparent lack of correlation with established knowledge of cardiac disease in dogs. Although the statistical analysis was robust, we are concerned that the general public will believe the findings of this study to be conclusive, despite use of the word “suggested” in the conclusions. Given the results of another recent study and the recent turnabout in recommendations for humans, we believe there is no clinically important association between periodontal disease and infective endocarditis and that this study cannot be used to make such a suggestion.

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Instructions for Writing a Letter to the Editor

Readers are invited to submit letters to the editor. Letters may not exceed 500 words and 6 references. Not all letters are published; all letters accepted for publication are subject to editing. Those pertaining to anything published in the JAVMA should be received within one month of the date of publication. Submission via e-mail (Journal.Letters@avma.org) or fax (947-925-9329) is encouraged; authors should give their full contact information, including address, daytime telephone number, fax number, and e-mail address.

Letters containing defamatory, libelous, or malicious statements will not be published, nor will letters representing attacks on or attempts to demean veterinary societies or their committees or agencies. Viewpoints expressed in published letters are those of the letter writers and do not necessarily represent the opinions or policies of the AVMA.
We wish to comment on the recent article by Glickman et al titled “Evaluation of the risk of endocarditis and other cardiovascular events on the basis of the severity of periodontal disease in dogs.” While we would agree this article addresses a potentially important issue—that poor periodontal disease may be associated with cardiovascular disease—we are unable to accept at face value the relationships put forth by the authors, primarily because of insufficient diagnostic stringency.

As the authors mention, a serious limitation to the study involved the lack of criteria for diagnosis of cardiovascular diseases. We agree and therefore cannot accept the conclusions made. This is most important with regards to the assertion that endocarditis is significantly associated with severe periodontal disease. It may well be, but the lack of identifiable criteria for the diagnosis of endocarditis renders the conclusion questionable and thus unacceptable. Endocarditis can be difficult to diagnose, generally requiring fulfillment of a number of major and minor criteria and is frequently diagnosed definitively only on postmortem examination. Without knowing how endocarditis was diagnosed in the patients in this study, it is difficult to draw any conclusion regarding potential associations with periodontal disease.

The same is true for other cardiovascular conditions discussed in this study. The validity of the diagnoses is even more concerning when one evaluates the reported prevalences of these other cardiovascular conditions, which differ greatly from prevalences in previous large-scale studies. For example, mitral valve insufficiency was diagnosed in only 0.17% of patients. Given the median age of the patients, a higher prevalence would be expected in this study population. At the other extreme, hypertrophic cardiomyopathy, a reportedly rare condition in dogs, was diagnosed at an extremely high frequency (56 dogs in the study, compared with only 207 dogs with mitral valve insufficiency and only 131 dogs with dilated cardiomyopathy). These inconsistencies support our suggestion that the criteria for diagnosis of cardiac diseases were not stringent enough, rendering any subsequent associations or conclusions meaningless.

It is undoubtedly desirable to provide quality dental care to our patients, and it may be true that periodontal disease is a contributory factor in cardiac disease. Unfortunately, the information provided by this study did not help to answer that question. Without firm evidence of confirmation of cardiovascular disease, the authors cannot expect the veterinary community to accept their conclusions.

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The authors respond:

We appreciate comments regarding our article published in the February 15, 2009, issue of the JAVMA. This was an observational (retrospective cohort) epidemiologic study based on electronic medical records of 59,296 dogs with periodontal disease and 59,296 dogs without periodontal disease followed prospectively in > 650 primary-care Banfield veterinary hospitals from 2002 through 2006. The hypothesis was tested that an increase in the severity of periodontal disease was associated with an increase in the risk of cardiovascular diseases such as endocarditis and cardiomyopathy, but not common endocrine, urinary tract, and musculoskeletal conditions; cancer; or infectious diseases. Cardiovascular diseases were considered because they have been associated with microbes commonly found in the oral cavity. An observational study does not involve intervention, experimental or otherwise. It uses existing records and systematic data collection to identify predisposing factors for subsequent disease.

Peddle et al question the judgment of primary-care veterinarians to correctly diagnose cardiovascular diseases. Banfield veterinarians typically refer dogs to clinical specialists if the diagnosis is uncertain, although for a variety of reasons, such referrals may not occur, including economic concerns of the owners.

A recent survey of small animal veterinarians found a greater acceptance of veterinary specialists by veterinarians in private practice and a trend towards more frequent referral to specialists. Banfield veterinarians also use a computerized clinical information system (PetWare) that generates potential rule-out diagnoses based on the basis of clinical signs and provides suggested diagnostic and treatment protocols.

A limitation to our study was that diagnostic criteria were not established a priori for cardiovascular events and diagnoses were subject to the judgment of each veterinarian. Missclassification with respect to recording of cardiac events, however, should have been unbiased and should have made it more difficult to identify a significant relationship between periodontal disease and cardiac diseases. Despite this limitation, risks of endocarditis, cardiomyopathy (both hypertrophic and dilated), and mitral valve insufficiency were significantly increased among dogs with stage 3 periodontal disease, but not among dogs with stage 1 periodontal disease, compared with dogs without periodontal disease. In addition, detection of a cardiac murmur, arrhythmia, or coughing was significantly associated with severe periodontal disease, yet none of these conditions require advanced diagnostic techniques. Similar associations were not found with common non-cardiac conditions.

On the basis of their clinical experience, Peddle et al question the high proportion of dogs we found with hypertrophic cardiomy-
opathy and note that the Kaplan-Meier curves in the manuscript are inconsistent with previously published data on cardiac disease. However, their perspective is likely based on dogs referred to veterinary teaching hospitals or specialty practices. Our findings in primary-care practices should not necessarily parallel their experience.

We agree with Ettinger et al that prospective clinical studies with predefined protocols are needed before veterinarians can be expected to accept an association between periodontal and cardiovascular diseases in dogs, even though this has been repeatedly demonstrated in humans. Minimal criteria for epidemiologic evidence needed to establish a causal relationship include a temporal relationship, strength of association, dose-response relationship, plausibility, coherence, specificity, and consistency. A single large study like ours cannot produce sufficient evidence to meet all of these causal criteria. Additional population-based studies are needed to demonstrate consistency, and these will require availability of large, computerized clinical databases similar to that created by Banfield. Experimental studies are critical for characterizing pathophysiologic mechanisms linking periodontal disease with systemic conditions.

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Manufacturer defends organic premix diet

Regarding the article “Diffuse osteopenia and myelopathy in a puppy fed a diet composed of an organic premix and raw ground beef,” we would like the opportunity to defend our reputation, based on 25 years of exemplary results. Our time-tested pet food premixes have been around since 1985 with a track record of consistently good results. We have a loyal network of veterinarians and breeders across the country who use and recommend our foods, and tens of thousands of dogs are currently being fed our foods.

While our hearts go out to the dog and owner alike described in this report, we respectfully disagree that our food was the cause of this ailment. All of our foods are formulated to have a calcium-to-phosphorus ratio of 1.4:1. At the same time, we do not want to minimize the severity of the situation, and as a precautionary measure, we have sent our foods for an unscheduled independent chemical analysis to confirm our values. The only possible explanation for the values in this article indicating a nutritional inadequacy on the part of our food is that only a small sample was tested and not an entire bag. Our foods are not homogenous kibble. Finer ingredients, namely powders (such as calcium carbonate and nutrient-rich kelp), settle toward the bottom of the bag. Thus, unless an entire bag of food was tested, the full spectrum of ingredients would not be represented and results would not be representative. Also, although we are confident in the nutritional profiles of our mixes, we believe that only comparing the chemical profile with published recommendations from the Association of American Feed Control Officials to determine a food’s nutritional adequacy ignores crucial information on the biological availability and digestibility of nutrients. Digestive enzymes found in unprocessed foods, like those in our premixes, enhance biological availability, whereas extreme heat (common to the preparation of commercial pet foods) leads to the depletion of enzymes and therefore depressed levels of digestible energy. Because all of our foods are made from whole, unprocessed, raw ingredients, we believe that all of our foods are more than adequate nutritionally, and we believe this is why we see such positive results in puppies, adult dogs, and senior dogs alike.

Ward Johnson  
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The authors respond:

We appreciate the comments provided by Ward Johnson, President of Sojourner Farms. Our report was not an attempt to denigrate the quality of his product. Rather, our intent was to illustrate the importance of feeding a balanced diet. Our data demonstrating the nutritional imbalances of the Sojos European Style diet refer to the premix as formulated in 2006, when the puppy of our report became ill. We are pleased to now note Mr. Johnson’s assurance and Sojourner Farms’ advertising that the current premix formulation is supplemented to achieve a more appropriate calcium-to-phosphorus ratio. However, we reiterate our concern that when the premix is mixed with meat, as the product...
label instructs, the calcium-to-phosphorus ratio becomes substantially inverted. In addition, vitamin D, another nutrient whose deficiency contributed to the disease in our puppy, was not and still does not appear to be supplemented in the premix.

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