Association of periodontal disease, oral procedures, and other clinical findings with bacterial endocarditis in dogs

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Objective—To identify risk factors potentially associated with the development of bacterial endocarditis in dogs and determine whether periodontal disease and surgical procedures (oral and nonoral) were associated with bacterial endocarditis.

Design—Retrospective case-control study.

Animals—76 dogs with (cases) and 80 dogs without (controls) bacterial endocarditis.

Procedures—Medical records were reviewed for information on signalment, physical examination findings, recent medical history, and results of echocardiography, clinicopathologic testing, and necropsy.

Results—None of the dogs with endocarditis had a history of undergoing any dental or oral procedure in the 3 months prior to the diagnosis of endocarditis, and no significant difference was found between groups with regard to the prevalence of oral infection. Dogs with endocarditis were significantly more likely to have undergone a nonoral surgical procedure that required general anesthesia in the preceding 3 months or to have developed a new heart murmur or a change in intensity of an existing heart murmur. Preexisting cardiac disease (congenital or acquired) was not found to be a risk factor.

Conclusions and Clinical Relevance—Results did not provide any evidence of an association between bacterial endocarditis in dogs and either dental or oral surgical procedures or oral infection. Findings suggested that the routine use of prophylactic antimicrobial administration in dogs undergoing oral procedures needs to be reevaluated. (J Am Vet Med Assoc 2009;234:100–107)

In humans, it has long been postulated that transient bacteremia resulting from dental procedures can lead to bacterial endocarditis. However, prophylactic administration of antimicrobials in human patients undergoing dental procedures has not been shown to decrease the incidence of endocarditis, and the efficacy of prophylactic antimicrobial administration is now questioned. In addition, recent studies have concluded that dental treatment is not a risk factor for bacterial endocarditis in people, and it is now thought that most cases of bacterial endocarditis are not caused by invasive procedures.

Similarly, prophylactic antimicrobial administration has been recommended for dogs undergoing dental procedures, even though the evidence that dental procedures may be a cause of bacterial endocarditis in dogs has been sparse and anecdotal. In addition, although there is proof that dental procedures can cause bacteremia in dogs, there is no evidence to suggest that bacteremia associated with dental procedures is a common cause of bacterial endocarditis. In a previous study, the percentage of positive blood culture results was not significantly different between dogs undergoing dental scaling and tooth extraction and control dogs that were not undergoing dental procedures. Furthermore, in a separate study, the incidence of bacteremia was not significantly different between dogs that did or did not receive penicillin prior to dental scaling and extraction. On the other hand, bacterial species commonly found in the oral cavity have been isolated from the heart valves of dogs with endocarditis, and periodontal disease has been associated with histologic abnormalities in a variety of organs, including the kidney, myocardium, and liver. Thus, periodontal disease may be a more important risk factor for development of bacterial endocarditis in dogs than dental procedures.

The study reported here was undertaken in an effort to clarify possible associations between bacterial endocarditis and periodontal disease in dogs and between bacterial endocarditis and dental or other oral surgical procedures. Specifically, the purposes of the study reported here were to identify risk factors potentially associated with the development of bacterial endocarditis in dogs and to document infective foci in dogs with bacterial endocarditis. A secondary purpose of the study was to document historical, physical examination, clinicopathologic, and necropsy abnormalities in a large group of dogs with bacterial endocarditis.

Abbreviations

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<tr>
<th>OR</th>
<th>Odds ratio</th>
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<td>CI</td>
<td>Confidence interval</td>
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Materials and Methods

The study was designed as a retrospective case-control study.

Case selection—Medical records of the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania were searched to identify dogs examined between January 1, 1990, and July 1, 2004, in which bacterial endocarditis had been diagnosed. Dogs were included as cases in the study only if a definitive diagnosis of bacterial endocarditis had been made on the basis of necropsy findings or on the basis of positive blood culture results in conjunction with compatible clinical signs and echocardiographic findings.

Control selection—Control dogs included in the study were randomly selected from the group of all dogs examined at the Matthew J. Ryan Veterinary Hospital during the same period, except that dogs suspected to have bacterial endocarditis were excluded from inclusion in the control group. One control dog was selected for each case dog included in the study, with cases and controls matched solely on the basis of year of examination during the study period. Control dogs were selected on the basis of visit number, rather than patient identification number, to allow for designation of a specific date of examination that corresponded with the date of diagnosis for dogs with endocarditis.

Data collection—Information obtained from the medical records of case and control dogs included in the study consisted of age at the time of diagnosis (case dogs) or examination (control dogs), breed, sex, neuter status, whether the dog had a history of infective oral disease within the previous 3 months, whether the dog had evidence of infective oral disease at the time of the initial physical examination, whether the dog had undergone any dental or surgical procedures in the previous 3 months that had required general anesthesia, whether the dog had undergone general anesthesia at any time in its lifetime (not including routine ovariohysterectomy or neutering at a young age), whether the dog had a history of a heart murmur or preexisting heart disease, whether a new heart murmur or change in the intensity of an existing murmur had been detected, rectal temperature, results of a CBC performed at the time of initial examination, whether there was any evidence of presumptive immunosuppression (eg, neoplasia, hyperadrenocorticism, or corticosteroid administration) prior to or at the time of initial examination, and whether any infective foci (defined as a site of infective disease) had been identified within the 3 months prior to diagnosis or at necropsy. In addition, for case dogs, information was obtained on the method by which endocarditis had been diagnosed (necropsy, aerobic bacterial culture of blood samples, or both), organisms obtained by means of aerobic bacterial culture of blood or endocardium, and whether there were any other necropsy abnormalities (eg, thromboemboli). In dogs with bacterial endocarditis, valvular or mural endocardial involvement was determined on the basis of necropsy or echocardiographic findings.

For all case and control dogs, those portions of the medical record specifically related to oropharyngeal and dental findings, including physical examination forms, daily in-hospital assessment forms, and necropsy reports (in dogs that underwent a necropsy within 3 months after the diagnosis of bacterial endocarditis or 3 months after the initial examination), were evaluated by a board-certified veterinary dentist (CEH) who was blinded to case-control status of individual patients to determine whether dogs had evidence of oral infection. Dogs were considered to have evidence of oral infection if the medical record included specific mention of periodontal disease, oral ulceration, or a tooth root abscess. Dogs were considered to have no evidence of oral infection if the medical record included comments on examination of the oral cavity beyond those associated with mucous membrane color and capillary refill time and there was no mention of periodontal disease, oral ulceration, or a tooth root abscess. Dogs with dental calculus alone and dogs with an oral mass or gingival hyperplasia were also considered to have no evidence of oral infection if the medical record did not include any mention of gingivitis, periodontitis, or oral ulceration.

Dogs with a tooth fracture were considered to have no evidence of oral infection if the medical record did not include any mention of a tooth root abscess. Information in the medical record was considered insufficient to determine whether oral infection was present if the medical record did not include any comments on examination of the oral cavity or only included information on mucous membrane color and capillary refill time.

Statistical analysis—Categoric variables were summarized as percentages. Continuous variables were assessed for normality by means of visual evaluation of the data distribution and the Shapiro-Wilk test. Variables that were normally distributed were summarized as mean and SD; variables that were not normally distributed were summarized as median and range. For categoric variables, the χ² or Fisher exact test was used to compare values for case and control dogs. For continuous variables, the unpaired t test or Wilcoxon rank sum test was used to compare values between case and control dogs.

Univariate ORs and their 95% CIs were calculated to test for associations between putative risk factors and bacterial endocarditis. Because the Wilcoxon rank sum test indicated that age was significantly different between case and control dogs, all univariate ORs were adjusted for age. Confidence intervals were calculated by means of the exact method, except that the CI for the association between bacterial endocarditis and septic arthritis was calculated by means of the Wald procedure because none of the control dogs had septic arthritis.

Multiple logistic regression was used to identify factors significantly associated with bacterial endocarditis. A backward stepwise procedure was used for development of the multiple logistic regression model. All variables with a P value < 0.20 in univariate analyses were included in the initial model, and variables were sequentially removed on the basis of the highest P value until all variables remaining in the model had individual P values < 0.05. Variables with a P value > 0.20 were then sequentially incorporated in the model to determine whether any were significant, and those with P values < 0.05 were retained in the final model.
All analyses were performed with standard software. For all analyses, a value of \( P < 0.05 \) was considered significant.

**Results**

Seventy-six case and 80 control dogs were included in the study. Eighty dogs were initially included in the case group, but 4 were later removed because of inconsistencies in the medical record that left the diagnosis of endocarditis, according to study guidelines, in question. During the study period, 116,092 dogs were examined at the Matthew J. Ryan Veterinary Hospital.

Continuous variables—Case dogs were significantly \((P = 0.002)\) older than control dogs (Table 1), with 71 of the 76 (93%) case dogs being ≥ 4 years old. Median rectal temperature at the time of initial examination was also significantly \((P = 0.044)\) higher in case than in control dogs. Thirty-one of 70 (44%) case dogs had Hct values less than the lower reference limit; however, although 13 (22%) control dogs had rectal temperatures available had temperatures < 37.2°C (99.0°F). Only 11 of 61 (18%) control dogs had rectal temperatures > 39.4°C.

Median RBC count, hemoglobin concentration, and Hct were significantly lower in case than in control dogs (Table 1). Eighteen of 60 (30%) case dogs and 14 of 27 (52%) control dogs had Hct values greater than the upper reference limit; however, although 13 (22%) case dogs had Hct values less than the lower reference limit, only 1 (4%) control dog did.

Absolute WBC and neutrophil counts were also significantly higher in case than in control dogs (Table 1), with 39 of 60 (65%) case dogs but only 9 of 26 (35%) control dogs having leukocytosis. Similarly, 44 of 39 (75%) case dogs but only 9 of 26 (35%) control dogs had neutrophilia. Platelet count was significantly lower in case than in control dogs, with 16 of 26 (62%) case dogs and 4 of 23 (17%) control dogs having thrombocytopenia.

**Categoric variables**—Twenty-three of the 76 (30%) case dogs were of mixed breeding, and 53 (70%) were purebred dogs representing 31 breeds. Forty-six of the 53 (87%) purebred case dogs were medium- or large-breed dogs, and only 7 (13%) were small-breed dogs. The most common breeds among case dogs were Labrador Retriever \((n = 6)\), Golden Retriever \((5)\), Weimaraner \((4)\), and Boxer \((4)\). Breed distribution was not significantly different between the case and control groups. Forty-two (55%) case dogs were male \((24\) neutered), and 34 (45%) were female \((28\) spayed). Neither sex distribution nor distribution of neuter status differed significantly between the case and control groups.

The proportion of dogs in which a new heart murmur or a change in the intensity of an existing murmur had been detected was significantly \((P < 0.001)\) higher for case dogs \((25/76 [33%]\); 21 with a new murmur and 4 with a change in intensity of an existing murmur) than for control dogs \((5/80 [6%]\); all 5 with a new murmur; Table 2). Only 2 of the 25 case dogs with a new murmur or change in intensity of an existing murmur had bacterial endocarditis.

Table 1—Comparison of continuous clinical and clinicopathologic findings in dogs with (case dogs) and without (control dogs) bacterial endocarditis.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Case dogs</th>
<th>Control dogs</th>
<th>( P ) value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>NA</td>
<td>76 (8.6 (4.4–15)</td>
<td>6.7 (0.2–19)</td>
</tr>
<tr>
<td>Rectal temperature (°C)</td>
<td>70 (39.3 (34.5–40.9)</td>
<td>31 (23 (18.8–28.8))</td>
<td>27 (36.6–40.5)</td>
</tr>
<tr>
<td>RBCs (\times 10^6) cells/µL</td>
<td>5.0–9.9</td>
<td>59 (5.8 (1.3–9.3))</td>
<td>0 (22 (37.5–46.8))</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>8–15</td>
<td>60 (13.7 (28.2–21.2))</td>
<td>12 (20 (37–71))</td>
</tr>
<tr>
<td>Hct (%)</td>
<td>27–45</td>
<td>60 (40 (11–18))</td>
<td>13 (22 (27–31))</td>
</tr>
<tr>
<td>MCV (fL)</td>
<td>62.7–75.5</td>
<td>59 (70 (59.2–94))</td>
<td>27 (69.61–75.2)</td>
</tr>
<tr>
<td>MCH (pg)</td>
<td>22.5–29.9</td>
<td>59 (23.5 (18.8–28.8))</td>
<td>11 (23.5)</td>
</tr>
<tr>
<td>MCHC (g/dL)</td>
<td>32.2–36.8</td>
<td>60 (33.8 (26.1–40.7))</td>
<td>2 (3)</td>
</tr>
<tr>
<td>RDW (%)</td>
<td>13.2–17.4</td>
<td>36 (15.1 (12.9–20.3))</td>
<td>1 (3)</td>
</tr>
<tr>
<td>Protein (g/dL)</td>
<td>6.0–8.6</td>
<td>41.7 (6.8–8.8)</td>
<td>8 (20)</td>
</tr>
<tr>
<td>WBCs (\times 10^6) cells/µL</td>
<td>5.5–19.5</td>
<td>60 (24.1 (6.5–76.2))</td>
<td>26 (12.5 (3.8–51.6))</td>
</tr>
<tr>
<td>Neutrophils (\times 10^6) cells/µL</td>
<td>2.5–12.5</td>
<td>59 (20.7 (4.1–64.8))</td>
<td>26 (8.2 (3.0–50.6))</td>
</tr>
<tr>
<td>Lymphocytes (\times 10^6) cells/µL</td>
<td>0.0–2.0</td>
<td>51.0 (3.0–14.3)</td>
<td>22 (0)</td>
</tr>
<tr>
<td>Monocytes (\times 10^6) cells/µL</td>
<td>1.5–7.0</td>
<td>59 (10 (0–12.9))</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Eosinophils (\times 10^6) cells/µL</td>
<td>0–0.9</td>
<td>3 (1.2 (0–8.8))</td>
<td>21 (0.7 (0–4.7))</td>
</tr>
<tr>
<td>Basophils (\times 10^6) cells/µL</td>
<td>0–1.5</td>
<td>30 (0.1 (0–2.7))</td>
<td>23 (0.1 (0–2.3))</td>
</tr>
<tr>
<td>Platelets (\times 10^11) platelets/µL</td>
<td>0–0.2</td>
<td>18 (0 (0–4.9))</td>
<td>17 (0)</td>
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MCV = Mean corpuscular volume. MCH = Mean corpuscular hemoglobin. MCHC = Mean corpuscular hemoglobin concentration. RDW = RBC distribution width.
had a diastolic murmur; none of the control dogs had a diastolic murmur. No significant differences were identified between groups with respect to proportion of dogs with a history of preexisting heart disease or proportion of dogs with a preexisting heart murmur that had not changed in intensity. Of the 5 case dogs with a history of preexisting heart disease, 2 had dilated cardiomyopathy, and 3 had chronic degenerative valvular disease. The 1 control dog with preexisting heart disease had cardiomegaly of unknown cause (a cardiac workup was not performed in this dog).

Eleven (15%) case dogs but only 1 (1%) control dog had undergone a surgical procedure in the previous 3 months that had required general anesthesia (P = 0.018; Table 2); however, for dogs in both groups, none of the procedures were dental or involved the oral cavity. Ten (13%) case dogs and 9 (11%) control dogs had undergone a dental or surgical procedure that required general anesthesia > 3 months before the time of initial examination. No significant difference was identified between the case and control groups with regard to the types of surgical procedures performed.

Overall, case dogs were significantly (P = 0.036) more likely to have evidence of presumptive immunosuppression (high plasma corticosteroid concentration or neoplasia) than were control dogs (Table 2). When this category was further subdivided, case dogs were significantly (P = 0.030) more likely to have high plasma corticosteroid concentrations (ie, hyperadrenocorticism [n = 3] or exogenous corticosteroid administration [7]) than were control dogs (ie, hyperadrenocorticism [1]), but were not significantly (P = 0.270) more likely to have neoplasia alone than were control dogs. Four case dogs had both neoplasia and high plasma corticosteroid concentrations, compared with none of the control dogs. Thirteen of the 19 case dogs with neoplasia had > 1 type of neoplasm (primary or metastatic), compared with 4 of the 10 control dogs with neoplasia (OR, 3.8; 95% CI, 0.6 to 24; P = 0.088).

The proportion of dogs with an infective focus involving any part of the body other than the oral cavity was significantly (P < 0.001) higher among case dogs (36/76 [47%]) than among control dogs (12/80 [15%]). The most common infective foci in case dogs included pneumonia (n = 10), septic arthritis involving ≥ 1 joint (9), septic peritonitis (4), prostatitis (4), skin or wound infection (3), pyelonephritis (3), and lower urinary tract infection (3). Other less common infective foci included pyometra, renal leptospirosis, splenic abscess, diskospondylitis, supplicative meningomyelitis and encephalitis, suppurative myocarditis, and gastrointestinal tract disease. Significant differences were found between the case and control groups with regard to proportions of dogs with pneumonia and septic arthritis.

For 63 of the 76 (87%) case dogs and 66 of the 80 (83%) control dogs, there was sufficient information in the record to determine whether the dog did or did not have evidence of oral infection. The proportion of case dogs with evidence of oral infection (31/63 [49%]) was not significantly (P = 0.39) different from the proportion of control dogs with evidence of oral infection (24/66 [36%]).

**Results of multiple logistic regression analysis—** Only 2 variables were retained in the final multiple logistic regression model: a new heart murmur or change in intensity of an existing heart murmur and undergoing a nonoral surgical procedure in the previous 3 months that required general anesthesia. Case dogs were 7.7 times (95% CI, 2.7 to 21.7; P < 0.001) as likely to have developed a new heart murmur or had a change in intensity of an existing heart murmur than were control dogs. Similarly, case dogs were 14.5 times (95% CI, 1.8 to 119; P = 0.012) as likely to have undergone a nonoral surgical procedure (Table 2).
procedure in the previous 3 months that required general anesthesia than were control dogs.

Other findings in dogs with endocarditis—Location of the lesion was recorded in 72 of the 76 (99%) dogs with endocarditis. In 64 of these 72 dogs, location of the lesion was confirmed at necropsy; whereas in the remaining 8 dogs, location of the lesion was determined echocardiographically. Overall, 63 of the 72 (87%) dogs for which lesion location was identified had valvular involvement; the remaining 9 (13%) dogs had only mural endocardial lesions. A small number of dogs had both valvular and mural lesions or multiple mural lesions. A single valve was affected in 54 of 72 (75%) dogs (mitral valve, 47 dogs; aortic valve, 6 dogs; and pulmonic valve, 1 dog). Six (8%) dogs had concurrent mitral and aortic valve lesions, and 3 (4%) dogs had tricuspid valve lesions in conjunction with mitral valve lesions (n = 2) or an aortic valve lesion (1). Overall, 55 (76%) dogs had lesions involving the mitral valve, 13 (18%) had lesions involving the aortic valve, 8 (11%) had lesions involving the left ventricular mural endocardium, 3 (4%) had lesions involving the right ventricular mural endocardium, 3 (4%) had lesions involving the tricuspid valve, 2 (3%) had lesions involving the wall of the left atrium or left atrial appendage, and 1 (1%) had a lesion involving the pulmonic valve.

Blood or endocardial tissue samples from 26 dogs were submitted for aerobic bacterial culture. In 21 dogs, a single organism was isolated; in 3 dogs, 2 organisms were isolated; and in the remaining 3 dogs, 3 organisms were isolated. Organisms that were isolated included β-hemolytic Streptococcus spp (5 dogs), Enterococcus spp (4), Staphylococcus spp (3), Enterobacter spp (3), and Escherichia coli (3). In some dogs, isolates were not identified to the species level but were characterized as gram-positive cocci (n = 4), gram-negative rods (1), or gram-positive rods (1). Serum antibody titers against Bartonella spp were not measured in any of the dogs with endocarditis.

Thromboemboli were identified in 22 of 68 (32%) dogs that underwent necropsy; with 8 of these 22 (36%) dogs having emboli in > 1 organ. The most common site for thromboemboli was the lungs (9/22 [41%]), followed by the kidneys (7/22 [32%]) and the distal portion of the aorta (6/22 [27%]). Other sites included the spleen, myocardium, iliac artery, liver, prostate, and adrenal glands. None of the dogs with pulmonary thromboemboli had endocarditis lesions involving the right side of the heart. In addition, none of the 7 dogs with endocarditis lesions involving the right side of the heart had evidence of thromboemboli on necropsy.

Discussion

Results of the present study did not provide any evidence of an association between bacterial endocarditis in dogs and either dental or oral surgical procedures or oral infection. The only 2 factors found by means of multiple logistic regression analysis to be significantly associated with bacterial endocarditis were detection of a new heart murmur or change in intensity of an existing heart murmur in dogs with clinical signs of sepsis is not surprising, as most dogs with endocarditis have some degree of valvular involvement, and vegetative lesions on valve leaflets are likely to result in either poor leaflet coaptation and turbulent regurgitant flow or valvular stenosis and turbulent outflow (aortic and pulmonic valves). It is noteworthy that less than half of all dogs with endocarditis had new or changed murmurs, however, indicating that the absence of a heart murmur in dogs with clinical signs of sepsis is not reliable in ruling out the presence of endocarditis.

The significant association in the present study between bacterial endocarditis and undergoing a nonoral surgical procedure in the previous 3 months that required general anesthesia is more difficult to explain. Any predisposition to development of bacterial endocarditis secondary to surgical procedures would likely represent a multifactorial process involving the location and type of procedure, underlying disease, IV catheterization, type and duration of anesthesia, surgical contamination, and perioperative care. It can also be postulated that patients who have recently undergone a surgical procedure were more likely to have substantial illness (such that surgical intervention was required) and thus more likely to be immunosuppressed, hypercoagulable, or in a proinflammatory state, conditions that might favor development of endocarditis. Importantly, only 11 of the 76 (15%) case dogs and 1 of the 80 (1%) control dogs had undergone a surgical procedure within the 3 months prior to diagnosis of bacterial endocarditis or initial examination, and none of these procedures were dental or involved the oral cavity. In addition, the proportion of dogs that had undergone a surgical procedure that required general anesthesia > 3 months before the time of initial examination did not differ significantly between the case and control groups. The small proportion of cases having undergone recent surgical procedures prior to development of endocarditis emphasizes that although this was a significant risk factor, most dogs with endocarditis do not have a history of a recent surgical procedure. The most recent recommendations from the American Heart Association regarding prophylactic administration of antimicrobials for prevention of bacterial endocarditis in human patients do not support use of prophylactic antibiotics in patients undergoing respiratory, gastrointestinal, or genitourinary manipulation solely for the purpose of prevention of endocarditis unless the patient is considered high risk (defined as patients with underlying cardiac conditions associated with the highest risk of adverse outcome from infective endocarditis, not necessarily those with increased lifetime risk of acquisition of the disease).

The finding that none of the dogs with bacterial endocarditis in the present study had undergone a dental or other oral surgical procedure in the 3 months prior to diagnosis lends support to the argument that these types of procedures are not a significant risk factor for development of endocarditis in dogs. Similarly, recent studies in humans have not identified an association between dental procedures and endocarditis. Moreover, most of the earlier studies linking the 2 were based on cases in which dental treatment had been performed months prior to the onset of clinical signs, and it is now generally accepted that the incubation period for streptococcal endocarditis is < 2 weeks. Recommendations from the American Heart Association regarding prophylactic
administration of antimicrobials for prevention of bacterial endocarditis in humans also state that only a small number of cases of infective endocarditis would be prevented by such prophylaxis, even if prophylaxis were 100% effective, and that prophylactic antimicrobial administration prior to dental procedures is reasonable only for patients with underlying cardiac conditions associated with the highest risk of adverse outcomes from infective endocarditis.12 When considered in conjunction with the fact that structural heart disease, with the possible exception of subaortic stenosis, does not appear to be a risk factor for endocarditis in dogs, prophylactic antimicrobial administration in dogs undergoing routine dental or oral procedures is difficult to justify, regardless of whether they are healthy or have congenital or acquired heart disease (other than subaortic stenosis). A randomized, controlled clinical trial with large case numbers would be needed to truly resolve this issue.

We did not identify any significant difference between case and control groups in regard to the proportion of dogs in each group with evidence of oral infection. Detection of oral infection in dogs in the present study was limited by a variety of factors, of which the most important were the retrospective nature of the study and the fact that examinations of the oral cavity for signs of infection were not performed in a standardized manner and results of examinations that were performed were not recorded in a systematic way in the medical records. For the present study, we attempted to overcome some of these limitations by having medical records examined by a single board-certified veterinary dentist blinded to the case-control status of individual dogs. However, a true assessment for periodontal disease should ideally include systematic procedures for examining the oral cavity and a detailed scoring system to record the nature and severity of disease.20–22

It has been estimated that 70% to 75% of human patients with endocarditis have preexisting cardiac abnormalities.23 In contrast, the prevalence of preexisting heart disease did not differ significantly between case and control groups in the present study and, unlike findings in previous studies,24–26 none of the dogs with endocarditis had subaortic stenosis. However, the authors have seen cases of presumptive endocarditis involving dogs with subaortic stenosis, and it is possible that the criteria for case selection (requiring necropsy or positive results of blood culture) in the present study may have skewed the population. Nevertheless, our results did suggest that there is not a strong association between subaortic stenosis and endocarditis in dogs.

In the present study, rectal temperature at the time of initial examination was significantly higher in case than in control dogs. However, 56% (39/70) of the dogs with endocarditis were afebrile when initially examined, suggesting that the lack of a fever was a poor criterion for eliminating bacterial endocarditis from the list of potential differential diagnoses in dogs with other clinical signs suggestive of the disease.

Similarly, although significantly different when compared with controls, only 33% (25/76) of the dogs with endocarditis in the present study had a new murmur or change in the intensity of a preexisting murmur at the time of initial examination, suggesting that the lack of a heart murmur should not be used to rule out bacterial endocarditis. The relatively low percentage of dogs with new or changed heart murmurs in the present study may be partially explained by the fact that 13% (9/72) of case dogs had mural endocarditis without any evidence of valvular involvement, which would be unlikely to be associated with a murmur. Findings for rectal temperature and heart murmurs in dogs with endocarditis in the present study were consistent with results of a previous study,27 which reported that fever and a heart murmur may not be present initially in some dogs with endocarditis, but may be noted later.

Anemia is reportedly common in dogs with endocarditis,23,28 and RBC count, Hct, and hemoglobin concentration were significantly lower in dogs with endocarditis in the present study, compared with control dogs. Leukocytosis and neutrophilia have also been described as common findings in dogs with endocarditis,23,26,27 and results of the present study supported these findings. Thrombocytopenia has previously been associated with bacterial endocarditis, and in a recent study,23 thrombocytopenia was suggested to be associated with prognosis.

Some previous studies21–26,29 of bacterial endocarditis in dogs have revealed a higher incidence of mitral valve involvement, whereas others have revealed a higher incidence of aortic valve involvement.27,28 In the present study, most dogs in which a single valve was affected had mitral valve involvement (47/54 [87%]), whereas only 6 of the 34 (11%) dogs with only a single valve involved had aortic valve involvement. Overall, 55 of the 72 (76%) dogs in which lesion location could be determined had mitral valve involvement, and only 13 (18%) had aortic valve involvement. The rare occurrence of tricuspid and pulmonic valve involvement was consistent with findings of previous studies.23,26–29 The reason for the preferential involvement of the mitral valve versus the aortic valve in the present study was unknown. A recent study30 found preferential aortic valve infection in dogs with Bartonella endocarditis, but anti-Bartonella antibody titer was not evaluated in dogs in the present study. Importantly, 9 (13%) dogs in the present study did not have valvular involvement but had lesions involving the ventricular or atrial endocardium. Mural endocarditis is uncommon in humans31–34 and rarely reported in dogs,35–37 and other animals.38–40 However, mural endocarditis may be underreported in dogs, as this condition would often not be identified antemortem. Nevertheless, this represents an important subset of dogs with endocarditis that likely have fewer identifiable cardiac abnormalities, such as a murmur or echocardiographic lesion.

Organisms most commonly cultured from blood and postmortem tissue samples in the present study were similar to those described in previous reports and included Streptococcus spp, Enterococcus spp, Staphylococcus spp, Enterobacter spp, and E coli. Thromboembolism was identified in 22 of 68 (32%) dogs that underwent necropsy, which was slightly lower than the percentage in a previous study.23 Pulmonary thromboembolism (9/22 [41%]) was most common, but there was no association between right-sided heart involvement and pulmonary thromboembolism.

Not surprisingly, the proportion of dogs with an infective focus involving any part of the body other than the oral cavity was significantly higher among case dogs (36/76 [47%]) than among control dogs (12/80 [15%]) in the present study. Owing to the retrospective nature of the study and the criteria for case selection, most infective foci were identified at necropsy. Thus, we were unable to determine whether these foci represented a primary disease process or had developed secondary to septicemia or sep-
tic embolization, although it seems likely that certain foci (eg, skin wounds and septic peritonitis) were primary. Infective foci reported in dogs with bacterial endocarditis in previous studies have included abscesses, wounds, pneumonia, oral infections, dental disease, pyelonephritis, glomerulonephritis, prostatitis, and perianal fistulas, among others. In the present study, pneumonia and septic arthritis were the most common foci of infection, followed by septic peritonitis, prostatitis, and pyelonephritis, but only pneumonia and septic arthritis were more common in case than in control dogs. The large number of dogs from (9) with endocarditis and septic arthritis was noteworthy because lameness in dogs with endocarditis is often attributed to deposition of immune complexes within the joints, rather than sepsis. Our results suggested that evaluation of the joints in dogs with lameness and signs consistent with endocarditis may be helpful in establishing a definitive diagnosis.

In the present study, we used strict criteria (ie, necropsy evidence or positive blood culture results in conjunction with compatible clinical and echocardiographic findings) to identify cases of bacterial endocarditis. Although use of these strict criteria may have caused us to exclude some dogs with bacterial endocarditis from the study because the diagnosis had not been definitively confirmed, it ensured that all case dogs included in the study did in fact have the condition. Limitations of the present study included the relatively small number of dogs in each group and problems inherent with retrospective case series, such as incomplete medical records, subjective characterization of clinical findings, and variability in treatments administered, prior to or at the time of initial examination that may have altered clinical or clinicopathologic findings. Dogs suspected to have endocarditis may have been examined more extensively to identify infective foci or may have undergone more extensive diagnostic testing than control dogs. Owing to our strict criteria for establishing a definitive diagnosis of bacterial endocarditis, the diagnosis was made at the time of necropsy in most dogs in the present study, which prohibited evaluation of outcome. Results of serum biochemical analyses were not examined in the present study, and exposure to Bartonella spp was not assessed during diagnostic testing in most cases.

There is evidence in humans that bacteremia and systemic inflammation secondary to periodontal disease adversely affect various organ systems and may be linked to a number of diseases, including subacute bacterial endocarditis and atherosclerosis. Until recently, the systemic effects of periodontal disease in dogs had not been investigated, but the negative impact on health appears to be real. However, whether periodontal disease has a role in the development of bacterial endocarditis in humans is still unclear. Findings of the present study do not provide evidence to support the suggestion that oral infection or dental procedures are significant risk factors for development of endocarditis in dogs. Thus, the practice of routine prophylactic antimicrobial administration to prevent endocarditis in dogs undergoing dental procedures should be reevaluated. Prospective studies on the systemic effects of chronic periodontal disease in dogs are needed to better define its role in development of systemic disease.

References

Selected abstract for JAVMA readers from the American Journal of Veterinary Research

Use of dietary cation anion difference for control of urolithiasis risk factors in goats
Meredith L. Jones et al

Objective—To determine correlations between dietary cation anion difference (DCAD) and urine pH, urine specific gravity, and blood pH in goats.

Animals—24 crossbred goat wethers.

Procedures—Goats were randomly assigned to 1 of 4 DCAD groups (–150, –75, 0, or +75 mEq/kg of feed) and fed pelleted feed and ground hay for 7 days. The diet was then supplemented with ammonium chloride to achieve the assigned DCAD of each group for 7 days. Urine was obtained for pH and specific gravity measurements at hours –2 to –1, 1 to 3, 5 to 7, 9 to 11, and 13 to 15 relative to the morning feeding. Blood pH was determined on alternate days of the study period.

Results—Goats in the –150 and –75 mEq/kg groups had a urine pH of 6.0 to 6.5 two days after initiation of administration of ammonium chloride, and urine pH decreased to < 6.0 by day 7. Goats in the 0 mEq/kg group had a urine pH from 6.0 to 6.5 on day 5, whereas urine pH in goats in the +75 mEq/kg group remained > 6.5 throughout the trial. Urine specific gravity differed only between the –150 mEq/kg and the –75 mEq/kg groups. Blood pH in the –150 mEq/kg group was significantly lower than that in the other groups.

Conclusions and Clinical Relevance—Goats in the 0 mEq/kg DCAD group had a urine pH of 6.0 to 6.5 five days after initiation of feeding the diet, and that pH was maintained through day 7, without significant reduction in blood pH. This may serve as a target for diet formulation for the prevention of urolithiasis. (Am J Vet Res 2009;70:149–155)