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Poor oral hygiene as a risk factor for infective endocarditis–related bacteremia

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Abstract

Background—Infective endocarditis (IE) often is caused by bacteria that colonize teeth. The authors conducted a study to determine if poor oral hygiene or dental disease are risk factors for developing bacteremia after toothbrushing or single-tooth extraction.

Methods—One hundred ninety-four participants in a study were in either a toothbrushing group or a single-tooth extraction with placebo group. The authors assessed the participants' oral hygiene, gingivitis and periodontitis statuses. They assayed blood samples obtained before, during and after the toothbrushing or extraction interventions for IE-associated bacteria.

Results—The authors found that oral hygiene and gingival disease indexes were associated significantly with IE-related bacteremia after toothbrushing. Participants with mean plaque and calculus scores of 2 or greater were at a 3.78- and 4.43-fold increased risk of developing bacteremia, respectively. The presence of generalized bleeding after toothbrushing was associated with an almost eightfold increase in risk of developing bacteremia. There was no significant association between any of the measures of periodontal disease and the incidence of bacteremia after toothbrushing. The

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oral hygiene or disease status of a tooth was not significantly associated with bacteremia after its extraction.

Conclusion—Bacteremia after toothbrushing is associated with poor oral hygiene and gingival bleeding after toothbrushing.

Clinical Implications—Improvements in oral hygiene may reduce the risk of developing IE.

Keywords

Bacteremia; bacteria; infective endocarditis; heart valves; risk factors

In 1909, Horder[1] emphasized that "oral sepsis" was an important factor in the genesis of infective endocarditis (IE). Since that time, however, the focus has been on prevention of bacteremia that results from invasive procedures. Traditionally, it has been recommended that people with a spectrum of medical conditions and devices receive antibiotic prophylaxis (AP) before dental, gastrointestinal, genitourinary and other invasive procedures.[2] There is, however, an emerging consensus that bacteremia resulting from invasive procedures is, at most, a rare cause of IE, which is itself a rare disease, and that AP poses a greater risk than benefit to patients.[3,4]

Recently, there have been major changes in recommendations from experts on this issue. Since 2007, American Heart Association (AHA) guidelines focus only on dental procedures for patients with cardiac conditions whom the AHA defines as being at "higher" risk of experiencing morbidity and mortality resulting from IE, largely because most of the case reports and studies of bacteremia relate to dental rather than nondental procedures.[3] In the United Kingdom, the 2008 National Institute for Health and Clinical Excellence guidelines recommended the complete abandonment of AP for all patients with cardiac conditions undergoing any invasive procedures.[4] Although the recommendations for the prevention of IE have a greater focus on improved oral health, there are no data from prospective clinical studies to demonstrate that poor oral health is a risk factor for IE.

Gingivitis and periodontitis are inflammatory diseases of the gingiva and supporting structures of the teeth. They are caused by specific bacterial species. There is evidence that the surface of inflamed and ulcerated gingival crevicular tissue around teeth is the portal of entry for the viridans streptococci bacteria that cause as much as 50 percent of the IE cases in the United States annually.[5–8] Some of these cases are found commonly or almost exclusively in the oral cavity (for example, *Streptococcus mitis*, *Streptococcus mutans*). Mansur and colleagues[9] estimated that *Streptococcus* species cause as much as 56 percent of recurrent endocarditis cases.

Although hundreds of bacterial species have been reported to enter a person's systemic circulation during dental procedures, we have found that toothbrushing is a more common source of bacteremia caused by IE-causing oral pathogens.[10,11] We could not, however, find a study in the literature in which the investigators identified a relationship between specific dental hygiene or gingival disease parameters and bacteremia resulting from routine daily events such as toothbrushing. We theorized that if such a relationship exists, it could lead to a more evidence-based, effective and universally accepted focus on preventing cases of IE caused by oral pathogens. This approach would target and benefit all patients at risk of developing IE, rather than focusing on administering AP before specific invasive procedures and to the small percentage of patients defined by the AHA as being at "higher" risk of experiencing a bad outcome from IE.

The main focus of our study was to determine if specific measures of oral hygiene, gingival disease or both would be risk factors for bacteremia caused by IE-associated oral species after toothbrushing or single-tooth extraction.

PARTICIPANTS AND METHODS

We initiated a double-blind, randomized, placebo-controlled study and enrolled patients who came to our hospital-based dental service and who needed to have at least one erupted tooth extracted. We excluded patients if they had fewer than 10 teeth; had taken a systemic antibiotic agent within the previous two weeks; needed AP on the basis of AHA guidelines; had an active viral infection, poorly controlled systemic disease, penicillin allergy, temperature greater than 100.5°F or facial cellulitis; had eaten or brushed their teeth within one hour before the study; or were immunocompromised by virtue of disease or medications. All participants provided written informed consent. We informed those who met the inclusion criteria about the study, which received Carolinas Medical Center's Institutional Review Board–approved consent.

Over a three-year study, we screened 600 patients and randomly assigned 290 to one of three groups: toothbrushing, single-tooth extraction with AP or single-tooth extraction with a placebo that was identical in look, feel, taste and so forth to AP, as described previously.[10] The results of some aspects of this study have been published previously.[10–12] We excluded from the results participants who were in the single-tooth extraction with AP group because we were interested only in the impact of oral hygiene and disease indexes on bacteremia.

We obtained demographic information and medical histories from the participants and conducted thorough clinical and radiographic examinations of their teeth and periodontia at baseline. We assessed oral hygiene and gingivitis statuses at four sites per tooth by using common dental plaque,[13] calculus[14] and gingivitis[15] indexes. We assessed periodontal status by means of probing depths, which we measured to the nearest whole millimeter at six sites per tooth by using a manual probe. We used tooth mobility[16] scores as a second, indirect measure of periodontal status. We scored dental caries as clinically present or absent for each tooth. We examined radiographs to determine the presence and depth of caries. We assigned a score of 0 if there were no caries or if the lesion was limited to the enamel or dentin. We assigned a score of 1 if there was gross caries or caries involving the tooth pulp. We determined the presence and depth of dental caries only for the teeth that were to be extracted. Also, for the teeth in the extraction group, we looked for radiographic evidence of periapical radiolucency, which most often indicates necrotic pulp tissue with an acute or chronic apical abscess. If a radiolucency was present, we recorded its diameter in millimeters.

We asked participants in the toothbrushing group to brush all tooth surfaces adjacent to the gingiva with a new toothbrush and without tooth-paste for a total of two minutes (30 seconds for each maxillary and mandibular quadrant). An examiner (J.N.) recorded any evidence of gingival bleeding after toothbrushing. If bleeding was present, she documented the extent of bleeding as localized (at a single site) or generalized (at multiple sites).

One hour after administration of the placebo and 15 minutes before surgery, we locally anesthetized participants in the extraction group with 1.8 milliliters of 2 percent lidocaine with 1:100,000 epinephrine. We administered mepivacaine hydrochloride 3 percent (Carbocaine, Cook-Waite, Novocol Pharmaceutical of Canada, Cambridge, Ontario, Canada) without vasoconstrictor if further local anesthesia was necessary. We then extracted the indicated tooth. Participants in the toothbrushing group underwent tooth extraction at the end of the study period, after the last blood sample was drawn or during a subsequent office visit.

For both study groups, we drew venous blood for bacterial culture and identification at six time points: before toothbrushing or extraction (draw 1); during and immediately after toothbrushing

or extraction (draws 2 and 3); and at 20, 40 and 60 minutes after toothbrushing or extraction (draws 4–6). The examiner, who was blinded as to participant randomization, placed the blood samples in culture media (BACTEC Plus Aerobic/F Becton, Dickinson, Sparks, Md., and Lytic/10 Anaerobic/F, Becton, Dickinson). We isolated bacterial colonies by using selective and nonselective media.[10] We isolated bacterial DNA and used it as the template for amplification of the 16S ribosomal RNA genes, followed by DNA sequencing and analysis of the genes for bacterial identification as described previously.[11,12] If we found a blood culture positive for bacteria at any time during or after the study procedure (that is, at blood draws 2–6), we determined that bacteremia was present.

For the toothbrushing group, we averaged the dental plaque, calculus, gingivitis, probing depth and tooth mobility scores within and then across participants. For the extraction group, we averaged the clinical scores from the tooth to be extracted because only this tooth and its surrounding tissue were manipulated during the study. We dichotomized participants' mean plaque, calculus and gingivitis scores by using the cutoff of 2.0 ($<$ versus $\geq$) to signify more severe disease. In all analyses, the dependent variable was the presence or absence of a positive blood culture at one or more of the time points, regardless of the number or nature of the species.

We reported descriptive statistics as means and standard deviations, or frequencies and percentages, as appropriate. We constructed logistic regression models to assess the association of demographic and dental disease parameters with the likelihood of bacteremia. We used a statistical significance of $< .05$ in all cases. We conducted all analyses by using a statistical software package (SAS 9.1, SAS Institute, Cary, N.C.).

RESULTS

Table 1 shows the demographic and clinical characteristics of the participants according to study group. The 194 participants in the two study groups (toothbrushing and extraction) had a mean age of 40.1 years (range, 18–83 years). A total of 54.6 percent were men, and 72.7 percent were African American. Twenty-six participants (13.4 percent) reported having had their teeth professionally cleaned within two years, whereas the remaining 168 participants (86.6 percent) had had their last professional cleaning more than two years earlier. Ten participants (10.2 percent) in the toothbrushing group and 16 participants (16.7 percent) in the extraction group had their teeth professionally cleaned within two years.

We identified 98 different bacterial species in the blood samples. We reported data on the diverse phylogeny of these species elsewhere.[10] Of these 98 species, 32 overlapped with a list of 275 bacterial species that we identified in an exhaustive search of the literature for bacterial species that were reported to cause IE.[10] Results in our study pertain exclusively to findings related to these 32 species. The participant-based cumulative incidence of IE-related bacteremia assessed from the five interventional/postinterventional blood draws was 22.5 percent and 60.4 percent for the toothbrushing and extraction groups, respectively ($P < .0001$). Ninety-three of the participants with detectable bacteremia had cultures negative for bacteria within 20 minutes after the study procedure. A total of 43.8 percent of the 32 IE-associated oral bacterial species were viridans streptococci. Thirteen of 27 (48.1 percent) cultures positive for bacteria in the toothbrushing group contained viridans streptococci, compared with 106 of 152 (69.7 percent) cultures positive for bacteria in the extraction group ($P = .0285$).

In the extraction group, we found no significant associations between oral hygiene or oral disease parameters and the incidence of IE-related bacteremia (Table 2). In the toothbrushing group, however, increased age was a predictor for developing IE-related bacteremia after toothbrushing ($P = .017$). The risk of developing bacteremia increased 6 percent for each additional year of age. In addition, all measures of oral hygiene (mean plaque score, plaque

score of ≥ 2, mean calculus score, calculus score of ≥ 2) and one measure of gingival bleeding (generalized bleeding with toothbrushing) were significantly associated with IE-related bacteremia. Participants with mean plaque or calculus scores of 2 or greater had 3.78- and 4.43-fold increased risks, respectively, of developing an IE-related bacteremia after toothbrushing ($P < .01$) (Table 2). In addition, the presence of generalized bleeding after toothbrushing was associated with an almost eightfold risk of developing bacteremia caused by oral bacterial species implicated in IE.

We further evaluated the impact of poor oral hygiene on the incidence of bacteremia by assessing the outcomes of participants in the toothbrushing group whose plaque and calculus scores were in the highest quartile. Nineteen of 98 participants in the toothbrushing group had both plaque and calculus scores that were in the highest quartile. The incidence of bacteremia after toothbrushing in this group was 42 percent (n = 8), which was significantly greater than the incidence among participants whose scores were in the lower three quartiles (18 percent, $P = .02$). The incidence of bacteremia after toothbrushing in the highest quartile was not significantly different than the incidence of bacteremia after a single-tooth extraction in 58 of 96 participants (60 percent, $P = .14$), as we reported previously.[10] We found no significant association between any measures of periodontal disease or caries or dental disease and the incidence of bacteremia with IE-related oral bacteria after toothbrushing or tooth extraction (Table 2).

DISCUSSION

Poor oral hygiene results in plaque and calculus accumulation around teeth that can lead to inflammation and ulceration of the gingival tissues (that is, gingivitis), which precedes periodontitis and eventual tooth loss.[17] Although it has long been assumed that poor oral hygiene and dental disease are important risk factors for IE, our study is the first to show a relationship between oral hygiene and gingival disease parameters and the risk of developing an IE-associated bacteremia after a routine daily event such as toothbrushing. We now have scientific evidence that good oral hygiene and gingival health are associated with a reduced risk of developing bacteremia, which may translate into a reduced risk of developing IE.

Investigators in previous studies of adults have reported conflicting results concerning the relationship between gingival or periodontal disease and bacteremia risk after tooth extraction.[18,19] In the extraction group in our study, neither the level of oral hygiene nor the gingival or periodontal health of the tooth to be extracted was associated with an increased incidence of bacteremia. One explanation for our negative finding for tooth extraction may be the significantly higher incidence of bacteremia associated with extraction compared with toothbrushing. Because of its invasive nature, tooth extraction, even when performed in a periodontally healthy mouth, is more likely to result in bacteremia than is toothbrushing.[10,18]

The oral cavity can be colonized by a wide range of bacteria; more than 700 species have been detected.[20] Streptococci constitute a significant proportion of the flora around the teeth, especially in the dental biofilm that grows above the gingival crest. Whereas toothbrushing results in relatively minor trauma to the potentially ulcerated gingival crevice, extractions disrupt both the superficial and deeper tooth-supporting tissues. The supragingival plaque also contains a higher proportion of IE-related bacterial species, whereas deeper periodontal pockets harbor more anaerobic and gram-negative species that are less commonly associated with IE. These differences may explain our finding that oral hygiene and gingivitis were more strongly associated with the risk of developing IE-associated bacteremia than were measures of periodontal disease (for example, probing depth and tooth mobility).

There were two limitations to our study. One limitation was that all participants were in need of a tooth extraction and were recruited from a hospital-based dental clinic. Therefore, they may not be representative of the broader adult population with regard to demographics and level of oral disease. For example, when compared with local (28 percent) and national (12 percent) population demographics, our study sample had a higher proportion of blacks (69.4 percent).[21] The results of at least one report suggest differences between racial groups in terms of oral bacterial flora in disease and health,[22] but we believe that our data can be extrapolated to other racial and ethnic groups because these differences should have no bearing on the incidence and nature of bacteria that enter the circulation from the gingival crevicular tissues.

The other limitation was that we did not design our study to test for associations between gingival disease parameters and bacteremia risk; we did not determine the power for this secondary purpose. Despite this limitation, we were able to detect clinically relevant and statistically significant associations between measures of oral hygiene and bacteremia risk. Before we conducted our study, there was no consensus regarding which clinical measures were most appropriate to study with regard to risk of developing a bacteremia, the relationship between oral and general health or the risk of developing an infection distant from the mouth (for example, IE). With regard to calculus, Albandar and Kingman[17] reported on a group of 9,689 people 30 years and older in the 1988–1994 Third National Health and Nutrition Examination Survey. This weighted sample is representative of 105.8 million civilian, noninstitutionalized adults in the United States. Calculus was assessed at two sites per tooth in two randomly selected dental quadrants, one maxillary and one mandibular. The prevalence of subgingival calculus in the population was 55.1 percent, and the extent was 27.4 percent of teeth per person. For people with subgingival calculus at at least one site, the percentage of teeth with subgingival calculus was 54.7 percent. The results of a 1985 through 1986 survey showed that 53.5 percent of the population 18 to 64 years of age had subgingival calculus, which increased to 65.6 percent for people 65 years and older.[23]

In our study, we found that 244 of 290 participants (84.1 percent) had subgingival calculus, and the extent was 31.4 percent of teeth for all participants. We found that 38.9 percent of the teeth in these 244 participants had subgingival calculus. We measured calculus at the same two sites as Albandar and Kingman[17] did, but we evaluated all of the teeth, rather than those in two of the four quadrants. The larger (84.1 versus 55.1 percent) prevalence of subgingival calculus in our total study population compared with that in Albandar and Kingman's[17] may reflect the hospital-based dental clinic population in our study, which may have been demographically distinct from and which may have had a greater burden of dental disease than did the general population. Our findings for the percentage of teeth with subgingival calculus in our total study population, however, were close to those of Albandar and Kingman[17] (27.4 versus 31.4 percent). Albander and Kingman[17] reported finding a higher percentage (54.7 percent) of teeth with subgingival calculus in their group with calculus compared with that in our study (38.9 percent).

The participants in our study had a higher mean probing depth than that reported as the national average by the Centers for Disease Control and Prevention (CDC) (3.54 mm versus 1.04 mm, respectively).[24] The CDC's data may be a low estimate, however, since its investigation involved probing two locations on a limited number of teeth, and our study involved probing six locations on all teeth. With regard to gingival bleeding, national data suggests that 50.3 percent of the adult population has some gingival bleeding after probing.[17] We measured bleeding after probing and found a 70 percent incidence, which is higher than the national average but difficult to compare because of the use of different measurements.

CONCLUSIONS

Long-standing oral disease prevention protocols reduce the risk of developing periodontal disease.[25] Our data suggest that methods used to prevent cases of IE that originate from oral bacteria should focus on improving oral hygiene and reducing or eliminating gingivitis, which should reduce the incidence of bacteremia after toothbrushing and the need to extract teeth owing to periodontal disease and caries. These findings also may provide measures by which physicians and dentists can monitor the risk of developing IE from oral pathogens by means of both patient histories (assessment of bleeding with toothbrushing) and clinical examinations (general assessment of plaque and calculus). Although a large clinical study would be necessary to substantiate this claim, our data suggest that maintenance of optimal oral hygiene and the absence of gingival disease should result in fewer cases of IE.

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ABBREVIATION KEY

AHA
American Heart Association

AP
Antibiotic prophylaxis

CDC
Centers for Disease Control and Prevention

IE
Infective endocarditis

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TABLE 1

Demographic and clinical characteristics of the study participants, according to group.

CHARACTERISTIC	TOOTHBRUSHING GROUP (n = 98)	TOOTH EXTRACTION WITH PLACEBO GROUP (n = 96)	*P* VALUE
Sex (No. of Participants [%])			.675
Male	55 (56.1)	51 (53.1)	
Female	43 (43.9)	45 (46.9)	
Ethnicity (No. of Participants [%])			.609
White	27 (27.6)	23 (24.0)	
African American	68 (69.4)	72 (76.0)	
Hispanic	2 (2.0)	1 (1.0)	
Other	1 (1.0)	0 (0.0)	
Age (Years) (Mean [SD*])	39.7 (11.7)	40.5 (10.9)	.721
Plaque Index Score (Mean ≥ 2 [%])	34 (34.7)	31 (32.3)	.885
Gingival Index Score (Mean ≥ 2 [%])	49 (50.0)	41 (42.7)	.453
Calculus Index Score (Mean ≥ 2 [%])	25 (25.5)	18 (18.8)	.348
Average Pocket Depth (Millimeters) Mean (SD)	3.54 (1.17)	3.80 (1.46)	.188
Bleeding (Any Gingival Score = 3 [%])	69 (70.4)	64 (66.7)	.575
Bleeding With Toothbrushing (No. of Participants [%])	47 (48.0)	—	—

* SD: Standard deviation.

TABLE 2

The relationships between parameters and incidence of bacteremia with infective endocarditis–related bacterial species, according to group.

PARAMETER	TOOTHBRUSHING GROUP (n = 98)			TOOTH EXTRACTION WITH PLACEBO GROUP (n = 96)		
	Odds Ratio	95% CI*	*P* Value	Odds Ratio	95% CI	*P* Value
Demographic Measure						
Age	1.06	1.01–1.10	.017	1.03	0.99–1.07	.211
Sex (risk level = female)	1.09	0.42–2.82	.866	1.64	0.72–3.77	.241
Body mass index	0.99	0.93–1.05	.749	0.99	0.94–1.04	.630
Oral Hygiene Measure						
Mean plaque score	2.53	1.25–5.10	.010	0.74	0.44–1.22	.236
Plaque score ≥ 2	3.78	1.41–10.16	.008	0.90	0.37–2.16	.811
Mean calculus score	1.77	1.01–3.11	.048	0.93	0.60–1.42	.724
Calculus score ≥ 2	4.43	1.60–12.25	.004	0.82	0.29–2.33	.715
Gingivitis Measure						
Mean gingival score	1.62	0.77–3.40	.203	0.71	0.42–1.22	.217
Gingival score ≥ 2	1.61	0.61–4.20	.335	0.76	0.33–1.75	.518
Bleeding with toothbrushing (yes, no)‡	0.89	0.33–2.38	.810	NA†	NA	NA
Bleeding type with toothbrushing (generalized versus localized bleeding)	7.96	1.49–42.56	.015	NA	NA	NA
Periodontal Measure§						
Mean probing depth	1.02	0.68–1.53	.918	0.95	0.71–1.27	.735
Tooth mobility score	1.93	0.71–5.26	.200	1.01	0.44–2.34	.978
Caries or Dental Disease Measure						
Dental caries	4.40	0.54–35.72	.165	1.66	0.45–6.17	.452
Depth of dental caries	0.43	0.13–1.38	.155	0.21	0.03–1.81	.156
Apical lucency	2.37	0.89–6.34	.086	0.86	0.37–1.99	.724
Apical lucency size (millimeters)	0.87	0.48–1.58	.647	1.00	0.72–1.39	.995

*CI: Confidence interval.

†NA: Not applicable.

‡Those who had bleeding were counted as “yes.”

§Per millimeter change in probing depth.