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Chewing fails to induce oral bacteraemia in patients with periodontal disease

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Abstract

Aim: To investigate whether chewing in patients with untreated chronic periodontitis or plaque-induced gingivitis causes bacteraemia of oral origin.

Method: Twenty-one patients with untreated chronic periodontitis (32–75 years old) and 20 with plaque-induced gingivitis (26–54 years old) chewed a standard wax medium for 4 min. Blood samples were drawn before, during and 5 min. post-chewing. Aerobic and anaerobic Bactec system culturing was performed for 21 days and positive bottles were subcultured and isolates were identified to genus level. A full periodontal analysis was performed on all teeth and included probing depths, recession, attachment levels, bleeding on probing, mobility plaque index and gingival index. Radiographs were assessed for the severity of alveolar bone loss.

Results: No bacteraemia of oral origin was detected in any patient. Skin contaminants (*Staphylococcus epidermidis*, *Propionibacterium* spp.) were detected in blood samples from three patients (two periodontitis; one gingivitis).

Conclusion: Chewing did not cause bacteraemia in chronic periodontitis or plaque-induced gingivitis patients and may not be a risk factor for infective endocarditis in at-risk individuals with periodontal disease.

Key words: chewing; dental; infective endocarditis; oral bacteraemia; oral microbiology; periodontal disease

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Infective endocarditis may affect patients with congenital or acquired endocardial defects or cardiovascular prostheses due to colonization of those defects or prostheses by spread of bacteria into the blood stream (bacteraemia). Bacteraemia of oral origin is thought to be important in the pathogenesis of infective endocarditis as viridans Streptococci account for 20–30% of cases of native valve and late prosthetic valve endocarditis (Dwyer et al. 1994, Netzer et al. 2000, Moreillon & Que 2004). Infective endocarditis caused by Streptococci has been reported to have a mortality rate of 6–16% (Sandre & Shafran 1996, Netzer et al. 2000). Owing to the significant potential role of such oral microorganisms in the pathogenesis of infective endocarditis, antibiotic prophylaxis is recommended for patients in defined cardiac risk groups undergoing dental treatment such as periodontal therapy, which is likely to cause a bacteraemia of oral origin (Leport et al. 1995, Dajani et al. 1997, Ramsdale et al. 2004a).

In addition to dental treatment causing bacteraemia of oral origin, chewing has been cited as a cause of oral bacteraemia (Seymour et al. 2000, Pallasch 2003) and is considered to be implicated in the pathogenesis of infective endocarditis (Strom et al. 1998, Moreillon & Que 2004). Several recent reviews on infective endocarditis and dental implications (Gendron et al. 2000, Seymour et al. 2000, Pallasch 2003, Slots 2003, Singh et al. 2005) have stated that chewing causes bacteraemia, and yet base this only on review articles (Everett & Hirschmann 1977, Guntheroth 1984). In fact, evidence that chewing can cause bacteraemia is based on seven studies, the majority of which were carried out in the 1930s–1960s (Round et al. 1936, Murray & Moosnick 1941, Robinson et al. 1950, Cobe 1954, Diener et al. 1964, Degling 1972, Forner et al. 2006).

Of these seven studies, two reported no bacteraemia on chewing (Robinson et al. 1950, Degling 1972), while the other five studies reported incidences of 7–55% (Forner et al. 2006, 7%; Cobe 1954, 17%; Round et al. 1936, 20%; Diener et al. 1964, 22%; Murray & Moosnick 1941, 55%). Analysis of the methodology of these studies showed that in only one study was a specific periodontal diagnosis given (Forner et al. 2006), while the remaining six studies failed to provide any clinical data. In the most recent study, Forner et al. (2006) reported that four out of 20

periodontitis patients had a detectable bacteraemia 30 s after completion of chewing while no patients with gingivitis or periodontal health experienced a bacteraemia. However, in their identification of the microorganisms from the blood cultures, they reported that five oral microorganisms were identified in patients with gingivitis after completion of chewing and only two microorganisms were found in the periodontitis group. This confusion in the reporting of Forner et al.'s (2006) results makes interpretation of their findings difficult. Thus, it is not possible to determine whether individuals with chronic periodontitis may be at greater risk of bacteraemia due to chewing than those with plaque-induced gingivitis.

A major deficiency in five of the seven chewing-bacteraemia studies (Round et al. 1936, Murray & Moosnick 1941, Robinson et al. 1950, Cobe 1954, Degling 1972) was that baseline blood samples were not obtained from all patients to ensure that any bacteraemia found following chewing was actually caused by the chewing and not a pre-existing bacteraemia. The requirement to obtain a pre-procedure, baseline bacteraemia is now recognized as a strict requirement for any study investigating bacteraemia induced by dental manipulations (Ramsdale et al. 2004b).

A study by Geerts et al. (2002) reported that plasma levels of endotoxin (bacterial lipopolysaccharide) were elevated following chewing of a proprietary chewing gum. The investigators reported that the increases in the levels of endotoxin above baseline were related to the severity of the periodontal disease present. However, the Geerts et al. (2002) study did not attempt to identify microorganisms in the blood samples such that it cannot be determined whether the detected endotoxin was related to any oral bacteraemia.

Given the limitations of these previous studies, there is a need to investigate the occurrence of oral bacteraemia caused by chewing in individuals with periodontal disease in which baseline blood samples are obtained, adequate microbiological analysis is undertaken and periodontal indices are measured. Accordingly, the aims of this study were to: (1) determine whether chewing in patients with plaque-induced gingivitis or chronic periodontitis causes bacteraemia; (2) identify any clinical factors that might be significantly associated with such bacteraemia; and (3) identify the microorganisms present within positive blood cultures.

Material and Methods

Experimental design

This study was a cohort investigation in which 21 subjects with untreated chronic periodontitis and 20 with plaque-induced gingivitis were required to attend the clinic for a single visit during which a chewing exercise, blood sampling and recording of clinical data were undertaken. Ethical approval for the study was obtained from the Human Research Ethics Committees of the University of Sydney and the Sydney West Area Health Service. Research was conducted in accordance with the World Medical Association Declaration of Helsinki (Version VI, 2002, http://www.wma.net/e/policy/b3.htm [accessed on 31 March 2005]). Volunteers were given written and verbal advice about the study and were required to sign a witnessed consent form.

Population screening

Potential subjects for the study were identified from patients referred to the periodontal unit of Westmead Centre for Oral Health, Westmead Hospital, Sydney, for whom clinical and radiographic records were available. Westmead Hospital staff with plaque-induced gingivitis were also invited to participate. A total of 143 patient files or referral letters and accompanying orthopantomograms were reviewed. Prospective patients were contacted by telephone during the period from March 2005 until October 2005 to enquire as to their willingness to be involved in the study. Forty one subjects were recruited: 20 with plaque-induced gingivitis (10 males, 10 females: aged 26–54 years) and 21 with chronic periodontitis (nine males, 12 females: aged 32–75 years). There were two smokers, two former smokers and 16 never smokers in the plaque-induced gingivitis group and nine smokers, eight former smokers and four never smokers in the chronic periodontitis group.

Subjects selected for this study were diagnosed with either untreated chronic periodontitis as defined by the American Academy of Periodontology (2000a, b) or plaque-induced gingivitis (Mariotti 1999). Patients with chronic periodontitis were required to have at least four sites with both probing depths (PDs) and clinical attachment loss (CAL) of 5 mm or greater and evidence of radiographic crestal bone loss on orthopantomograms (Walsh et al. 1997). In order to be eligible for inclusion in the study, patients were required to have a minimum of four pairs of opposing posterior teeth, no history of periodontal treatment in the preceding 12 months, no treatment with antibiotics in the previous 3 months and a palpable vein in the ante-cubital fossa. Exclusion criteria included congenital or acquired cardiac defects, immune defects, haematological disorders, diabetes, pregnancy, upper respiratory tract infections or a history of corticosteroid or immunosuppressive medications.

Data recorded

All clinical measurements were performed immediately following chewing and blood sampling as periodontal probing has been shown to cause bacteraemia (Daly et al. 1997, 2001, Kinane et al. 2005). Age, gender and smoking status (never, former, current) were recorded for each subject. Clinical examination consisted of assessing the plaque index (PI; Silness & Loe 1964) at two sites per tooth (facial and oral); gingival index (GI; Loe & Silness 1963) at two sites per tooth (facial and oral); PD in millimetres at six sites per tooth (mesial, mid and distal on facial and oral aspects); bleeding on probing (BOP) at six sites per tooth; recession in millimetres at six sites per tooth; and mobility (Yes/No) and graded I, II or III (Miller 1938). All probing measurements were rounded to the nearest mm and were performed by one examiner (AM) with the use of a PCP-11 periodontal probe (Hu-Friedy, Chicago, IL, USA) and a P2N Nabers furcation probe (Hu-Friedy). The degree of interproximal bone loss in patients with chronic periodontitis was assessed by determining the height of the crestal bone on the mesial and distal surfaces of each tooth between a point on the root 2 mm below the cemento-enamel junction to the root apex. Bone loss was graded as mild if it was in the coronal 1/3 of the root length, moderate if in the middle 1/3 and severe if in the apical 1/3. Radiographic assessment of orthopantomograms was performed by an experienced Maxillofacial Radiologist who was blind as to the nature of the study.

Calibration and reproducibility

Measurement calibration exercises were performed before the commencement of the study and reproducibility exercises were performed during the study. Intra-examiner reproducibility was evaluated on each patient with duplicate measurements at six sites randomly selected by the dental assistant. The intra-examiner reproducibility using the κ statistic was: PD (± 1 mm) 0.92; GI 0.80; PI 0.86; mobility 0.87; and radiographic assessment 0.82. A κ statistic above 0.75 is considered substantial (Thompson & Walter 1988).

Chewing

Patients were instructed not to brush or floss their teeth, eat any food, chew any gum or receive dental treatment for at least 30 min. before their appointment to avoid any possible baseline bacteraemia. A baseline blood sample was obtained immediately before chewing. The patient was then instructed to chew in their normal manner on a standardized paraffin wax sample (GC Saliva Test Kit, GC America, Alsip, IL, USA) for 4 min. The paraffin wax had been placed in a freezer for 15 min. before the experiment to provide consistency in the texture of the material. Patients were encouraged to chew on mobile teeth and teeth, that had radiographic evidence of severe bone loss. A second blood sample was taken during the second and third minute of chewing, and a final blood sample was taken 5 min. after cessation of chewing. Following the final blood sample, periodontal examination was performed.

Blood sampling

Blood samples were obtained from a vein in the antecubital fossa. Before each sampling, the skin was wiped with a sterile 70% isopropyl alcohol wipe (Medind® Alcohol Prep, Medical Industries Australia, Sydney, NSW, Australia). The venepuncture was performed via an intravenous cannula (Protectiv® Plus 22G × 25 mm, Cincinnati, OH, USA) inserted into the median cubital vein in the cubital fossa and left in place during the experiment. A one-way valve (RV1000NC Safsite®, Braun Medical Inc., Bethlehem, PA, USA) and minimum volume extension set (Tuta Healthcare, Lane Cove, NSW, Australia) were fitted and secured with a cannula dressing (Opsite IV3000™, 10 cm × 14 cm, Smith & Nephew, Hull, UK), allowing samples to be taken continuously through the same line. The cannula was flushed with 0.9% NaCl between each of the three samples along with the first 2 ml of blood drawn being discarded in a 5 ml syringe (Becton Dickinson, Singapore). Each blood sample was then collected in a single use 20 ml syringe (Becton Dickinson). A total of 60 ml of blood was collected from each patient: 20 ml of blood before chewing; 20 ml of blood during chewing; and a final 20 ml of blood taken 5 min. after completion of the chewing exercise.

Blood culture

The Bactec 9240 automated system (Becton Dickinson, Diagnostic Systems, Sparks, MD, USA) was used for culturing of blood samples in the microbiology laboratory of the Centre for Infectious Diseases and Microbiology, Westmead Hospital, Sydney, which is a tertiary referral centre and an anaerobic reference laboratory. Of the 20 ml of venous blood collected at each sampling, 10 ml was inoculated into an aerobic culture bottle (BD Bactec™ Plus Aerobic/F) and 10 ml into an anaerobic one (BD Bactec™ Lytic/10 Anaerobic/F). Bottles were incubated and continuously monitored in an automated processor (Bactec 9240, Becton Dickinson), and any bottles that signalled negative after 21 days were discarded.

Microbiological identification

Bottles that signalled positive were Gram-stained and subcultured onto Horse Blood Agar and Chocolate Agar plates (BioMedia Laboratories, Singapore), which were incubated at 5% CO_2 and 35°C; onto MacConkey agar plates that were incubated aerobically at 35°C and Brain Heart Infusion Agar supplemented with vitamin K (BioMedia Laboratories), which were incubated anaerobically at 35°C. Any isolated bacteria were identified to the genus level using conventional microbiological techniques (Murray et al. 2003). *Staphylococcus epidermidis* was identified using a tube coagulase test and the Phoenix identification and susceptibility system (Becton Dickinson). *Propionibacterium* spp. were identified using an API 20A system (Analytab products, Plainview, NY, USA).

Statistical analysis

The minimum desired sample size (12 per experimental group) was determined by the ability to detect a 30% difference in the proportion of patients exhibiting a chewing-induced bacteraemia between the groups with 80% power and $\alpha = 0.05$. Appropriate statistical methods – χ^2 test for proportions and independent *t*-test for continuous outcomes – were used to explore differences for binary and continuous outcomes. The statistical tests for continuous outcomes were undertaken in both the parametric (independent *t*) and distribution-free form (Mann–Whitney *U*) in order to account for the small sample size and inequality of variance. All analysis was undertaken using SPSS™ Version 13.0. An α of <0.05 was considered to be statistically significant.

Results

Subjects

The age and clinical data are shown in Table 1. All periodontitis patients were found to satisfy the inclusion criteria following periodontal probing. The mean PD ($p<0.001$), CAL ($p<0.001$), BOP percentage ($p<0.001$), PI ($p<0.001$) and GI ($p<0.001$) were significantly higher in the chronic periodontitis group compared with the plaque-induced gingivitis group, while the mean number of teeth was significantly lower ($p<0.002$). The chronic periodontitis group had significantly more mobile teeth than the plaque-induced gingivitis group ($p<0.001$). Patients diagnosed with chronic periodontitis had 255 teeth with grade I mobility, 93 with grade II mobility and 41 with grade III mobility, while the plaque-induced gingivitis group had 23 mobile teeth (all grade I). Radiographic assessment of interproximal bone loss in subjects with chronic periodontitis revealed that 44% of assessable sites had mild bone loss, 46% had moderate loss and 10% had severe loss.

Bacteraemia

The bacteraemias detected in the blood samples are shown in Table 2. Three patients had a baseline bacteraemia detected: two in the chronic periodontitis group (AN 16, TV 27) and one in the plaque-induced gingivitis group (JM 42). Two patients in the chronic

Table 1. Age and clinical data are shown for patients with plaque-induced gingivitis and chronic periodontitis

	Mean ± SD		*p* value*
	Plaque-induced gingivitis ($n = 20$)	Chronic periodontitis ($n = 21$)	
Age (years)	39.9 ± 7.0	49.7 ± 11.5	0.003‡
Teeth (*n*)	28.3 ± 1.8	25.4 ± 3.5	<0.002‡
Probing depths (mm)	2.3 ± 0.4	3.5 ± 0.6	<0.001‡
Clinical attachment level (mm)	2.4 ± 0.3	4.3 ± 0.9	<0.001‡
Bleeding on probing (%)	18.8 ± 11	46.5 ± 14	<0.001‡
Plaque index	1.2 ± 0.5	1.7 ± 0.4	<0.001‡
Gingival index	1.1 ± 0.4	1.7 ± 0.2	<0.001‡
Mobility grade I	1.3 ± 1.9	12.1 ± 4.5	<0.001‡
Grade II	0.00	4.4 ± 3.9	N/A
Grade III	0.00	1.9 ± 2.5	N/A

N/A, not applicable. Not possible to compute correctly when there are zero cells.
*Both the independent sample *t*-test and the Mann–Whitney *U*-test were used.
‡The *t*-test if the data satisfied the criteria of normal distribution and equality of variance
†The Mann–Whitney *U*-test if data did not satisfy the criteria of normal distribution and equality.

Table 2. Total number of positive samples obtained from the chewing exercise.

	Baseline	Chewing	Post-chewing
Plaque-induced gingivitis ($n = 20$)	1 (JM 42)	0	0
Chronic periodontitis ($n = 21$)	2 (AN 16, TV 27)	2 (AN 16, TV 27)	0

Initials and numbers identify the individual patients with positive bacteraemia.

Table 3. Identification of the bacterial species present in each of the three patients with positive bacteraemia

Patient	Baseline	Chewing	Post-chewing
AN 16	*Propionibacterium* spp.	*Propionibacterium* spp.	
TV 27	*Staphylococcus epidermidis*	*P. acnes*	
JM 42	*Propionibacterium* spp.		

periodontitis group had a bacteraemia detected during chewing, with both these patients also having a positive baseline bacteraemia (AN 16, TV 27). The bacterial species detected in the positive blood samples are shown in Table 3. No bacteria of oral origin were detected in any blood sample. Each of the bacterial species recovered from the blood samples (*P. acnes, Propionibacterium* spp. and *S. epidermidis*) was considered to be skin contaminants from the blood sampling procedure (Cockerill et al. 1997, McBryde et al. 2005). In the baseline bacteraemia, the skin commensal *Propionibacterium* spp. was detected in two patients (AN 16, JM 42) after 7 and 10 days of incubation, respectively, and *S. epidermidis* was detected in one patient (TV 27) after 3 days. The two bacteraemias recovered during chewing were *Propionibacterium* spp. in patient AN 16 and *P. acnes* in patient TV 27. These were detected after 7 and 10 days of incubation, respectively.

Discussion

This study found that chewing failed to induce a bacteraemia of oral origin in patients with either plaque-induced gingivitis or chronic periodontitis. Instead, the only bacteraemias detected were considered to be skin contaminants that were obtained from the blood sampling procedure (Cockerill et al. 1997, McBryde et al. 2005). The number of positive skin contaminants obtained in our study is similar to those obtained in two recent studies (Tomas et al. 2004, 10%, Kinane et al. 2005, 7%) using the same Bactec 9240 continuous monitoring system to assess oral bacteraemia. Our results are in accordance with two previous studies that failed to show any bacteraemia due to chewing (Robinson et al. 1950, Degling 1972) but are in disagreement with the five studies that did report bacteraemia (Round et al. 1936, Murray & Moosnick 1941, Cobe 1954, Diener et al. 1964, Forner et al. 2006).

Our study obtained baseline, pre-chewing blood samples from all patients as in order to show a significant bacteraemia following a dental procedure, it must be demonstrated that such a bacteraemia is statistically significantly different from the pre-procedure bacteraemia (Ramsdale et al. 2004b). In the current British recommendations for antibiotic prophylaxis for infective endocarditis in the dental setting (Ramsdale et al. 2004a), any study that did not obtain pre-procedure blood samples was excluded from the list of studies on which evidence-based recommendations were made. This would mean that three of the five positive chewing studies (Round et al. 1936, Murray & Moosnick 1941, Cobe 1954) and both of the negative studies (Robinson et al. 1950, Degling 1972) cannot be used to verify the occurrence or otherwise of bacteraemia caused by chewing. Thus, the present study fulfils the strict requirement of Ramsdale et al. (2004b) to ensure that pre-procedural blood samples are obtained.

Possible reasons for our inability to detect a positive bacteraemia include the timing of the blood samples, the consistency of the chewing medium or lack of sensitivity in our microbiological culture techniques. Previous studies on animals and children have reported that the highest yields of microorganisms occur between 30 and 90 s after inoculation with bacteria or initiation of a specific dental procedure (Silver et al. 1975, Roberts et al. 1992). Forner et al. (2006) obtained blood samples 30 s following completion of a 10 min. chewing exercise. Blood samples in our study were taken while the patient was chewing and were obtained during the second and third minutes of 4 min. of continuous chewing. This was thought to provide ample opportunity to detect a chewing-induced bacteraemia. Previous studies that have detected a positive bacteraemia following periodontal probing, dental extractions or endodontic treatment obtained blood samples either immediately following (Daly et al. 1997, 2001), at 30 s to 2 min. post-treatment (Okabe et al. 1995, Tomas et al. 2004) and up to 5 min. after completion of the dental treatment (Savarrio et al.

2005). In some instances, bacteraemia has been detected at up to 10-30 min. after completion of dental procedures (Lofthus et al. 1991, Messini et al. 1999, Rajasuo et al. 2004). This would suggest that any bacteraemia not detected while chewing would have been detected in the final blood sample obtained 5 min. after completion of chewing.

To provide a constant and firm chewing medium in our study, a high melting point paraffin wax from a commercial salivary testing kit, which had been cooled in a freezer, was selected. Use of this defined medium was considered preferable to some of the chewing mediums utilized in previous studies with vague descriptions such as ''hard candy'' (Cobe 1954) or ''mint lumps'' (Round et al. 1936). Geerts et al. (2002) used several pieces of soft chewing gum and a controlled gentle chewing action to detect increased plasma endotoxin levels, while Forner et al. (2006) used three pieces of chewing gum to detect bacteraemia. Although high melting point paraffin is a soft medium, it has been used in previous studies that have reported chewing-induced bacteraemia (Murray & Moosnick 1941, Diener et al. 1964). It is possible that differing consistencies among the various chewing mediums might have contributed to the differences in bacteraemia rates reported.

In previous studies in our laboratory, we have shown that the Bactec 9240 culture and continuous monitoring system is effective in detecting oral bacteraemia, including Gram negatives and fastidious oral organisms, caused by periodontal probing (Daly et al. 1997, 2001). The same Bactec system has also been shown to be effective in detecting these types of micro-organisms in oral bacteraemias caused by a range of procedures (Lucas et al. 2002, Tomas et al. 2004, Kinane et al. 2005). Studies on Bactec continuous monitoring systems have shown that 5 days are sufficient in routine circumstances to recover 97–99% of isolates (Wilson et al. 1993, Doern et al. 1997, Cockerill et al. 2004). Seventy-seven percent of positive blood cultures can be detected by day 4 and almost 90% by day 5 in patients with bacteraemia (Kara et al. 2004). It has been proposed that oral microorganisms present in bacteraemia are fastidious and may require a longer incubation period of 7–21 days (Daly et al. 1997, 2001, Ramsdale et al. 2004a, Kinane et al. 2005). However, other studies have found that odontogenic bacteraemia can be detected within 24–30 h when using the Bactec 9240 continuous monitoring system (Pauli et al. 1999, Lucas et al. 2002). Therefore, in order to provide consistency in methodology with previous oral bacteraemia studies, a 21-day continuous culture was used in this study, which would have provided ample time to detect any microbial growth. The Lysis filtration technique was not used as we did not aim to study the magnitude of bacteraemia but rather the incidence and, for this purpose, Bactec and Lysis filtration have been shown to be equivalent for detecting post-procedural bacteraemia (Lucas et al. 2002).

The results of our study challenge the notion of a background transient bacteraemia caused by chewing, and question the assertions of Guntheroth (1984) and Roberts (1999) that chewing and oral hygiene procedures could together account for up to 630 min. of bacteraemia in 1 month whereas a dental extraction might account for only 5 min. of bacteraemia (Guntheroth 1984), or that chewing has a 100,000 times greater risk of a bacteraemia than a single extraction (Roberts 1999). Although bacteraemia due to oral hygiene practices such as toothbrushing is well documented (Sconyers et al. 1973, Kinane et al. 2005), it would appear from our findings that chewing may not be a source of bacteraemia, even in patients with severe periodontal disease. This questions the suggestion that transient bacteraemia caused by chewing may be equal to invasive dental procedures as a risk factor for infective endocarditis in susceptible patients (Strom et al. 1998, Moreillon & Que 2004).

Patient-performed procedures such as toothbrushing, flossing or using oral irrigation devices have the potential to traumatize the gingival epithelial lining, thus allowing bacteria to gain access into the underlying tissue and hence the vascular system. However, the mode of entry due to chewing appears to have no clear mechanism. Theories to explain how bacteria might penetrate into the underlying tissues during chewing include a ''pumping action'' created by the movement of the tooth within the socket (Fish & MacLean 1936), rupture of small blood vessels and capillaries (Round et al. 1936) or creation of sudden negative pressure allowing aspiration of bacteria into the gingival blood vessels (Roberts 1999). It has also been hypothesized that a ''pumping effect'' caused by rubbing and depressing ulcerated periodontal tissues might allow the passage of subgingival bacteria through the ulcerated epithelial lining of periodontal pockets (Roberts 1999, Geerts et al. 2002). In light of these suggested mechanisms of microbial entry, it would be expected that individuals with chronically inflamed gingiva and especially with tooth mobility would have a high risk of bacteraemia due to chewing. However, despite the presence of 389 teeth with some degree of mobility, no oral bacteraemia was detected during or following chewing in any of the subjects with moderate to severe periodontal destruction.

Within the experimental limitations of this study, it appears that chewing in patients with either plaque-induced gingivitis or chronic periodontitis fails to induce a bacteraemia. This finding questions the accepted notion that transient bacteraemia caused by chewing may be a risk factor for infective endocarditis.

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Clinical Relevance

Scientific rationale for study: Chewing has been cited as a source of transient bacteraemia with the potential to cause infective endocarditis in at-risk cardiac patients. The evidence to support chewing as a cause of oral bacteraemia is inconclusive.

Principal findings: Chewing failed to induce a bacteraemia of oral origin in patients with either untreated chronic periodontitis or plaque-induced gingivitis. The only bacteria detected were considered to be skin contaminants due to the blood sampling procedure.

Practical implications: The notion that infective endocarditis caused by oral Streptococci may be initiated by a transient bacteraemia due to chewing appears unfounded. Instead, other causes of oral bacteraemia such as dental procedures or oral hygiene activities may play a more significant role in the pathogenesis of infective endocarditis in susceptible patients.