Journal of Clinical Periodontology
Official Journal of The European Federation of Periodontology
Founded by the British, Dutch, French, German, Scandinavian, and Swiss Societies of Periodontology

J Clin Periodontol 2009; 36: 323–332 doi: 10.1111/j.1600-051X.2008.01372.x

Journal of Clinical Periodontology

Bacteraemia due to dental flossing

Kenneth Crasta[1], Christopher G. Daly[1], David Mitchell[2], Brad Curtis[3], Douglas Stewart[4] and Lisa J. A. Heitz-Mayfield[1]

[1]Discipline of Periodontics, Faculty of Dentistry, University of Sydney, Sydney, NSW, Australia; [2]Institute of Clinical Pathology and Medical Research, Westmead Hospital, Sydney, NSW, Australia; [3]Discipline of Epidemiology and Biostatistics, Faculty of Dentistry, University of Sydney, Sydney, NSW, Australia; [4]Conscious Sedation and Pain Control Unit, Westmead Centre for Oral Health, Sydney, NSW, Australia

Crasta K, Daly CG, Mitchell D, Curtis B, Stewart D, Heitz-Mayfield LJA. Bacteraemia due to dental flossing. J Clin Periodontol 2009; 36: 323–332. doi: 10.1111/j.1600-051X.2008.01372.x.

Abstract

Aims: The aims of this study were to (1) investigate the incidence of bacteraemia following flossing in subjects with chronic periodontitis or periodontal health; (2) identify the micro-organisms in detected bacteraemias; and (3) identify any patient or clinical factors associated with such bacteraemia.

Materials and Methods: Baseline blood samples were obtained from 30 individuals with chronic periodontitis (17 M:13 F, 29–75 years) and 30 with periodontal health (17 M:13 F, 28–71 years) following a non-invasive examination. Each subject's teeth were then flossed in a standardized manner and blood samples obtained 30 s and 10 min. after flossing cessation. Blood samples were cultured in a BACTEC™ system and positive samples subcultured for identification.

Results: Forty per cent of periodontitis subjects and 41% of periodontally healthy subjects tested positive for bacteraemia following flossing. Viridans streptococci, which are commonly implicated in infective endocarditis (IE), were isolated from 19% of positive subjects and accounted for 35% of microbial isolates. Twenty per cent of subjects had a detectable bacteraemia at 10 min. post-flossing. No patient or clinical factors were significantly associated with post-flossing bacteraemia.

Conclusions: Dental flossing can produce bacteraemia in periodontally healthy and periodontally diseased individuals at a rate comparable with that caused by some dental treatments for which antibiotic prophylaxis is given to prevent IE.

Key words: antibiotic prophylaxis; bacteraemia; flossing; infective endocarditis; oral hygiene; viridans streptococci

Accepted for publication 25 November 2008

Bacteraemia of oral origin is considered to be important in the pathogenesis of infective endocarditis (IE) because oral streptococci account for 20% of cases of native valve IE and 26% of cases of late prosthetic valve endocarditis (Moreillon & Que 2004). IE caused by viridans streptococci has been reported to have a mortality rate of 6–16% (Sandre & Shafran 1996, Netzer et al. 2000). Thus, European, American and Australian guidelines for prevention of IE recommend that antibiotic prophylaxis should be given to individuals in specified cardiac risk groups before having dental treatment likely to cause bacteraemia [Horstkotte et al. 2004, Wilson et al. 2007, Infective Endocarditis Prophylaxis (IEP) Expert Group 2008]. The recent British guidelines are the exception and do not recommend antibiotic prophylaxis for dental procedures [National Institute for Health and Clinical Excellence (NICE) Guideline Development Group 2008]. All of these guidelines have stressed that good oral hygiene and oral health are important factors in reducing the incidence of IE in susceptible individuals and therefore it is recommended that oral hygiene should be reinforced for patients at risk of IE and that they attend regular preventive dental check-ups (Duval & Leport 2008).

Paradoxically, the various national guidelines suggest that patient-performed oral hygiene procedures could be implicated in IE due to their potential to cause bacteraemia on a daily basis as compared with dental treatment, which might occur infrequently (Daly et al. 2008). Indeed, the most recent American guidelines go so far as to state that the vast majority of cases of IE caused by oral microflora most likely result from random bacteraemias caused by routine daily activities, including oral hygiene procedures (Wilson et al. 2007). Similarly, the UK guidelines concluded that it was ''biologically implausible'' that a dental procedure would lead to a greater risk of IE than an everyday oral hygiene activity such as regular tooth-

Conflict of interest and source of funding statement

The authors declare that they have no conflicts of interests.
Funding was provided by competitive grants from the Dental Board of New South Wales, Australia, and the Australian Periodontology Research Foundation.

brushing (NICE Guideline Development Group 2008). The question then arises as to which individuals may be most at risk of bacteraemia due to their oral hygiene activities. While it has been reported that bacteraemia caused by toothbrushing is more common in the presence of periodontal inflammation than health (Silver et al. 1977), the situation as regards dental flossing is unclear. Although anecdotal reports suggest that flossing may be implicated in causing specific cases of IE (Jenney et al. 2001), epidemiological studies have reported that subjects who floss daily appear to be less susceptible to IE (Strom et al. 2000).

Dental flossing is the most common method of inter-proximal cleaning recommended by dentists and the most common method of inter-proximal cleaning utilized by patients (Warren & Chater 1996). The risk of bacteraemia following flossing is not clear as there is little published evidence available. Only four studies have previously sought to address the issue of whether flossing causes bacteraemia (Lineberger & De Marco 1973, Ramadan et al. 1975, Wank et al. 1976, Carroll & Sebor 1980). A critical appraisal of these previous studies reveals a number of methodological shortcomings such as a lack of a periodontal diagnosis (Ramadan et al. 1975); the use of partial recording indices when assessing the amount of periodontal disease present (Wank et al. 1976); no indication as to when periodontal assessment was performed in relation to the flossing exercise (Lineberger & De Marco 1973); the presence of an interval of a week or more between measuring indices and the actual performance of the flossing exercise (Wank et al. 1976, Carroll & Sebor 1980); and failure to record whether the subject or the investigator performed the flossing (Lineberger & De Marco 1973). Some studies included commonly regarded skin contaminants as positive results in their bacteraemia analysis (Ramadan et al. 1975, Wank et al. 1976) or did not identify the bacteria present in positive blood cultures to ensure they were of oral origin (Carroll & Sebor 1980).

From the previous studies, it is not possible to determine whether individuals with periodontal disease are at higher risk of bacteraemia from flossing than those who are periodontally healthy. Although one study reported a decrease in the incidence of bacteraemia due to flossing from 29% to 14% in the same group of patients before and after non-surgical periodontal treatment, no data were presented on the degree of gingival inflammation, plaque accumulation, bleeding on probing or probing depths, which could assist in identifying individual risk for bacteraemia (Wank et al. 1976). One study sought to investigate whether daily flossing resulted in a lower incidence of bacteraemia as compared with flossing every two to three days (Carroll & Sebor 1980). However, as the study involved only four subjects (two gingivitis; two healthy), no reliable conclusions can be drawn as to the impact of periodontal status or plaque levels on bacteraemia caused by flossing. Another study that investigated bacteraemia due to dental flossing, use of stimudents or periodontal surgery did contain a periodontally healthy control group (Lineberger & De Marco 1973). Unfortunately, the investigators did not subject the control group to flossing on the basis that the incidence of bacteraemia was only 20% in the untreated, periodontitis group.

Given the paucity of data on bacteraemia caused by flossing, there is a need to clarify whether patients with periodontal disease are more at risk of oral bacteraemia due to flossing than are patients with a healthy periodontium. Thus, the aims of this study were:

(1) To determine if there is an increased incidence of flossing-induced bacteraemia in patients with periodontitis as compared with periodontal health.
(2) To identify the micro-organisms present within positive blood cultures.
(3) To identify any factors that might be associated with such bacteraemia.

Materials and Methods

Study design

This study was a cross-sectional investigation in which 30 individuals with chronic periodontitis and 30 with periodontal health attended for a single visit during which the experimental flossing protocol was performed, blood samples collected and periodontal data gathered.

Ethics approval

The study was approved by the Human Research Ethics Committees of the University of Sydney and the Sydney West Area Health Service. Subjects were given written and verbal advice about the study and were required to sign a witnessed consent form. Research was conducted in accordance with the World Medical Association Declaration of Helsinki (version VI, 2002 http://www.wma.net/e/policy/b3.htm).

Population screening

Thirty volunteers with chronic periodontitis were sought by telephone interview from patients on the periodontal waiting list of the Westmead Centre for Oral Health (WCOH) and in person from patients presenting to clinics at the same institution, for whom clinical and radiographic records were available. The experimental procedures and purposes of the study were explained to all potential subjects and their medical and dental histories were reviewed to assess inclusion and exclusion criteria and suitable patients were then invited to attend for the clinical procedure. A total of 106 periodontitis subjects were screened of whom 58 did not meet the inclusion criteria, nine could not be contacted and seven declined to participate. Thirty subjects with periodontal health were recruited from the clinics and from staff and dental students at the WCOH, all of whom were contacted in person by the investigator (K. C.). A total of 53 subjects with periodontal health were approached of whom four did not meet the inclusion criteria and 18 declined to participate. Subjects in the periodontitis group were recruited first and then subjects in the periodontally healthy group were recruited on the basis of age and gender matching. Subject recruitment occurred between 05 March 2007 and 13 July 2008.

Selection criteria

Subjects in the periodontitis group were selected initially on the basis of radiographic evidence of inter-proximal bone loss viewed on an orthopantomogram (OPG) (Walsh et al. 1997). They were required to have had no history of periodontal treatment in the last 12 months. Subjects in the periodontally healthy group were selected on the basis of having no evidence of chronic periodontitis as defined by the American Academy of Periodontology (2000a, b) nor of having generalized

plaque-induced gingivitis (Mariotti 1999).

All subjects were required to have at least 10 teeth. Volunteers with significant medical problems (e.g. diabetes), cardiac defects or other conditions requiring prophylactic antibiotic cover, immune defects, haematological disorders, pregnancy, upper respiratory tract infections, mouth ulceration, pulpal/periapical infections or those taking immunosuppressive or corticosteroid medication or those who had taken antibiotics in the past 3 months were excluded from the study. Subjects were instructed not to brush or floss their teeth, chew any food or perform any intra-oral manipulations for at least 1 h before the experimental protocol.

Data collection

The experimental design is shown in Fig. 1. All experimental procedures and clinical data gathering were performed at the dental clinics at WCOH between 10 March 2007 and 17 July 2008. All data collection was performed by K. C. during a single visit. All invasive data-gathering procedures were performed immediately after the flossing and blood collection procedures, as periodontal probing has been shown to cause bacteraemia (Daly et al. 2001, Kinane et al. 2005). Data were gathered in three stages:

(1) Pre-flossing

(a) Patient questionnaire including medical history, personal inter-proximal cleaning regimen, age, gender and smoking status (never, former or current).
(b) Marginal Gingival Index (MGI; Lobene et al. 1986) on the vestibular and oral surfaces at all gingival papillae to assess gingival inflammation. The gingival tissue adjacent to a tooth bounding a saddle area was also considered a papilla.
(c) Plaque Index (PI; Silness & Löe 1964) at each approximal surface on facial and lingual/palatal aspects. A 1% erythrosine disclosing dye (Plaque Disclose, Professional Dental Supplies, Bayswater, Vic., Australia) was used to disclose plaque. No probing was performed as part of the PI.

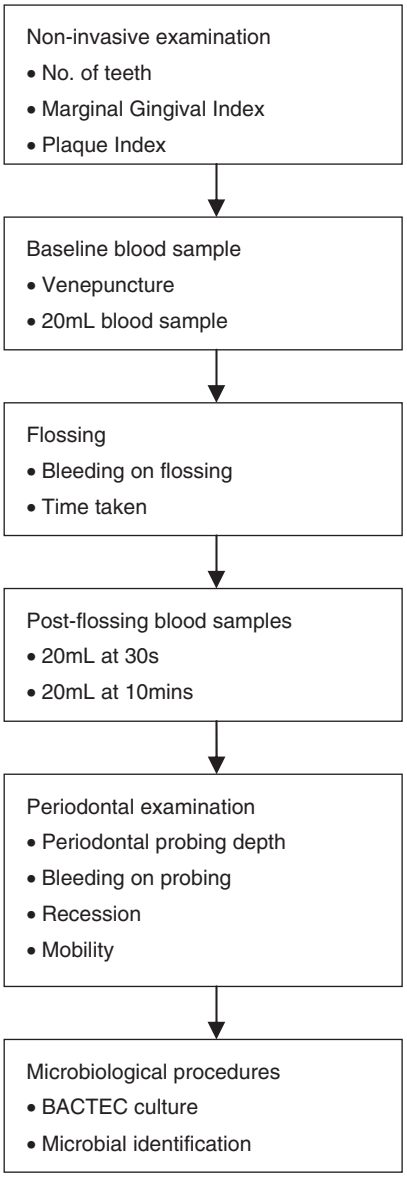

Fig. 1. Experimental design.

(2) During flossing

(a) Papillary bleeding on flossing (Carter & Barnes 1974). The presence or absence of bleeding on flossing was recorded within 30 s after flossing. Each papilla was treated as a single unit, regardless of whether bleeding was noted on the vestibular and/or oral aspect of the papilla or on the mesial and/or distal aspect of the papilla.
(b) Time taken to floss.

(3) Post-flossing

(a) Periodontal probing depth (PPD) and recession (REC) to the nearest millimetre were recorded using a PCP-11 periodontal probe (Hu-Friedy, Chicago, IL, USA) at six sites per tooth (mesial, mid and distal on vestibular and oral aspects).
(b) Bleeding on probing (BOP) assessed as Yes/No at six sites per tooth.
(c) Tooth mobility assessed as Yes/No and graded as Grade I, II or III (Miller 1938).

Flossing technique

Flossing was performed by K. C. to ensure standardization of technique and because the blood sampling procedure precluded subjects being able to use their own hands for flossing. A 150 cm strand of waxed dental floss (DentaFloss, Caredent, Hornsby, NSW, Australia) was pre-measured and then wrapped around the index fingers of each hand. The floss was gently moved through the contact area with a back and forth action using a combination of the thumbs and/or middle fingers of both hands. A recommended flossing action was then utilized (Perry 1996, American Dental Association 2008). The floss was shaped into a ''C'' configuration and moved apically along the tooth surface from the contact area to a position under the gingival papilla where it could not penetrate any further and then back again to the contact area. A standardized, calibrated force of 50 g (0.5 N) was used when flossing (Smith et al. 1986). This flossing action was repeated three times on the tooth surface before the adjacent tooth surface on the other side of the gingival papilla was similarly flossed (Carter et al. 1975). The floss was then unravelled slightly and moved between the fingers before progressing from one inter-proximal area to the next so that there was no transfer of blood or plaque from one area to another. The mesial and distal surfaces of all teeth were each flossed in this manner.

Calibration and reproducibility

Calibration scoring of the PI, MGI, mobility, PPD and REC was undertaken before the study. Intra-examiner reproducibility was calculated during the study by randomly re-assessing six selected sites within each subject for each of the indices, and calculating the percentage agreement within those scores. Analysis of the Pearson correlation coefficient in each case revealed substantial intra-observer reliability:

0.91 for the PI; 0.92 for the GI; 0.97 for mobility; 0.89 for PPD; and 0.95 for REC. A score of $\geqslant 0.75$ for the Pearson statistic is considered substantial (Thompson & Walter 1988). The force used when flossing was standardized at 50 g (0.5 N) by performing repeated measurements with the floss on an electronic weighing scale (Tronic S, Maul, Bad Konig, Germany) before the study.

Blood sampling

Three blood samples (20 ml each in volume) were obtained from each subject by a registered nurse under the direct supervision of the investigator. The skin was initially wiped with a sterile 70% isopropyl alcohol wipe (Medind® Alcohol Prep, Medical Industries Australia, Sydney, NSW, Australia) and venepuncture performed with an intravenous 25 mm/22 gauge cannula (Protectiv® Plus, Cincinnati, OH, USA) inserted into the median cubital vein. The cannula was fitted with a one-way valve (RV1000NC Safsite®, Braun Medical Inc., Bethlehem, PA, USA) and minimum volume extension set (Tuta Healthcare, Lane Cove, NSW, Australia). A 20 ml baseline blood sample was then taken with a 20 ml syringe (Becton Dickinson, Singapore, Singapore). After taking the baseline sample, the cannula was taped and secured with a sterile dressing (OPSITE IV3000™ 10 × 14 cm, Smith & Nephew, Hull, UK) to prevent contamination of the venepuncture site. The cannula was then left in place in the subject's outstretched and supported arm while the flossing procedure was performed.

Subsequent collections of 20 ml blood samples were initiated at 30 s and again at 10 min. after cessation of flossing. Immediately before these samples being obtained, 4 ml of 0.9% saline (Sodium Chloride Injection BP, Pfizer Australia Pty Ltd., West Ryde, NSW, Australia) was flushed into the vein with a 5 ml disposable syringe (Becton Dickinson) and the first 4 ml of blood was aspirated and discarded in order to avoid any contamination of the blood sample with saline. From each 20 ml blood sample, 10 ml was immediately inoculated into a BACTEC™ Plus Aerobic/F* 9240 media culture bottle (Becton Dickinson Diagnostic Systems, Sparks, MD, USA) and 10 ml into a BACTEC™ Lytic/10 Anaerobic/F* 9240 media culture bottle (Becton Dickinson Diagnostic Systems).

Microbiological procedures

All microbiological procedures were carried out in the microbiology laboratory of the Centre for Infectious Diseases and Microbiology, Institute of Clinical Pathology and Medical Research, Westmead Hospital, Sydney, which is a tertiary referral centre and an anaerobic reference laboratory. All bottles were transported to the laboratory within 30 min. of collection. The blood culture bottles were then incubated in the BACTEC™ 9240 automated processor (Becton Dickinson Diagnostic Systems). The staff at the microbiology laboratory were blinded as to whether the blood samples belonged to a subject with periodontitis or to one with periodontal health. Bottles were monitored continuously for 14 days before being discarded if they failed to signal positive.

Bottles that signaled positive were Gram-stained and subcultured onto Horse Blood Agar and Chocolate Agar plates (BioMedia Laboratories, Singapore, Singapore), which were incubated at 5% CO_2 and 35°C; onto MacConkey agar plates (Biomedia Laboratories) that were incubated aerobically at 35°C; and Brain Heart Infusion agar plates supplemented with vitamin K (BioMedia Laboratories), which were incubated anaerobically at 35°C. Any isolated bacteria were identified to at least genus level using conventional microbiological techniques (Murray et al. 2003) including colonial morphology, Gram stain appearance, catalase and oxidase reactions, and where appropriate, reactions in kit systems, such as API (Analytab Products, Plainview, NY, USA).

Interpretation of results and statistical analysis

The primary outcome measure for this study was the incidence of bacteraemia following flossing. A priori, sample size estimation was undertaken based on an expected difference in the incidence of post-flossing bacteraemia levels of 25% between subjects with chronic periodontitis and healthy controls. Based on that difference, a sample size of 28 subjects per group was calculated necessary to detect a significant difference ($\alpha = 0.05$) with 80% power and therefore 30 subjects were recruited in each group.

A correlation matrix was used to examine associations between subject and clinical variables. The Spearman (non-parametric) analysis was used in order to account for the relatively small sample size and high variance in the data. Mean scores for clinical measures that demonstrated normality of distribution are presented, otherwise median values are represented.

Appropriate statistical methods, such as χ^2 tests for proportions and *t*-tests (or the non-parametric equivalent), for continuous outcomes were used to explore differences for binary and continuous outcomes. In all cases α of <0.05 was considered to be statistically significant. All data analysis was undertaken using SPSS® Version 14.0 (SPSS Inc., Chicago, IL, USA) and SAS® Version 8.2 (SAS Institute Inc., Cary, NC, USA).

Results

Data for subject groups

The age and clinical data for the periodontitis group and the periodontally healthy group are shown in Table 1. All subjects in the periodontitis group were found to fulfil the diagnosis of severe chronic periodontitis as defined by Page & Eke (2007). They all had at least two inter-proximal sites on non-adjacent teeth and not on the same tooth, with clinical attachment loss (CAL) $\geqslant 6$ mm with at least one of these sites exhibiting concomitant PPD of $\geqslant 5$ mm. All subjects in the periodontally healthy group were found to fulfil the criteria for periodontal health of $\leqslant 20\%$ BOP and $\leqslant 10\%$ sites with PPD $\geqslant 4$ mm (Hugoson et al. 2008).

The periodontitis and periodontally healthy groups were age and gender matched. Both groups consisted of 17 males and 13 females with a mean age of 49.4 (± 12.0) years in the periodontitis group and 48.6 (± 12.1) years in the periodontally healthy group ($p = 0.9$). There were five current smokers, eight former smokers and 17 never smokers in the periodontitis group with the corresponding numbers in the periodontally healthy group being two, seven and 21. This difference was not statistically significant ($p = 0.4$). There were six daily, three less than daily and 21 never flossers in the periodontitis group, the corresponding figures being 15, nine and six in the periodontally healthy

Table 1. Clinical and patient data for the periodontitis and periodontally healthy groups

Variable	Periodontitis	Periodontal health	*p* value
Age (years) (mean ± SD)	49.4 (± 12.0)	48.6 (± 12.1)	0.9*
Teeth (mean ± SD)	26.2 (± 4.7)	26.9 (± 3.6)	0.3*
Gingival Index (median ± SD)	1.9 (± 0.4)	0.5 (± 0.6)	<0.001^
Plaque Index (median ± SD)	2.0 (± 0.6)	0.9 (± 0.9)	<0.001^
Periodontal probing depth (mm) (mean ± SD)	3.4 (± 0.9)	2.3 (± 0.2)	<0.001*
Periodontal probing depth sites ⩾5 mm (*n*) (mean ± SD)	28.9 (± 27.6)	0	<0.001*
Clinical attachment level (mm) (mean ± SD)	3.9 (± 1.0)	2.5 (± 0.4)	<0.001^
Bleeding on probing (%) (mean ± SD)	41 (± 19.4)	6 (± 3.8)	<0.001^
Teeth with mobility Grade II/III (mean ± SD)	0.9 (± 1.4)	0	<0.001*
Bleeding on flossing (%) (mean ± SD)	30 (± 16.5)	8 (± 6.9)	<0.001^
Bleeding on flossing (no. of sites) (mean ± SD)	9.2 (± 5.3)	2.5 (± 2.1)	<0.001*

*Independent t-test. ^Mann–Whitney U test.

group. This difference was statistically significant ($p = 0.001$).

The median GI ($p < 0.001$), median PI ($p < 0.001$), mean PPD ($p < 0.001$), mean number of sites with PPD ⩾5 mm ($p < 0.001$), mean CAL ($p < 0.001$), percentage of sites with BOP ($p < 0.001$) and number of teeth with mobility Grade II/III ($p < 0.001$) were significantly greater for the periodontitis group compared with the periodontally healthy group. There was no statistically significant difference in the number of teeth ($p = 0.3$) between both groups.

Flossing

It was possible to floss all inter-proximal areas of all teeth and no discomfort or adverse sequelae were reported by any of the subjects during or after flossing. The mean time taken to floss the subjects' teeth was 280.1 s (± 52.3) in the periodontitis group and 258.7 s (± 50.2) in the periodontally healthy group. This difference was not significant ($p = 0.8$). The occurrence of bleeding on flossing is shown in Table 1. The percentage of papillae that bled on flossing was higher in the periodontitis group (30%) when compared with the periodontally healthy group (8%). This difference was statistically significant ($p < 0.001$). The mean number of papillae that bled on flossing was higher in the periodontitis group (9.2 ± 5.3) than in the periodontally healthy group (2.5 ± 2.1) and this difference was statistically significant ($p < 0.001$).

Bacteraemia

One subject with periodontal health tested positive for bacteraemia of oral origin at baseline. The organism isolated before flossing in this subject was *Actinomyces odontolyticus*. The subject confirmed that he had avoided any brushing, flossing or chewing before the experimental procedure and there was no evidence of any periapical pathology on his OPG. This positive result at baseline necessitated that the subject's results be excluded from further analysis.

Four blood samples tested positive for bacteraemia of non-oral origin. A *Staphylococcus* spp. was isolated from one subject with periodontal health and *Propionibacterium* spp. were isolated from one subject with periodontitis and two subjects with periodontal health following flossing. These isolates were considered skin contaminants from the blood sampling procedure and were excluded from analysis (Cockerill et al. 1997, McBryde et al. 2005).

Table 2 summarizes the microbiological findings for those subjects who demonstrated a positive oral bacteraemia at 30 s and/or 10 min. post-flossing. In the periodontitis group, 12/30 (40%) of the subjects were positive for bacteraemia of oral origin following flossing whereas in the periodontally healthy group 12/29 (41%) of the subjects were positive. This difference was not statistically significant ($p = 0.8$). Ten minutes following flossing, 8/30 of the periodontitis group (27%) and 4/29 of the periodontally healthy group (14%) tested positive for bacteraemia of oral origin (20% of all subjects). This difference, however, was not significant ($p = 0.2$). Those subjects who tested positive for bacteraemia at 10 min. post-flossing did not necessarily test positive at 30 s post-flossing. In those subjects who exhibited a bacteraemia at both 30 s and 10 min., the microbial isolates were often different at the two time points.

Microbiological findings

A wide variety of oral micro-organisms was identified in the isolates. Viridans streptococci were identified in positive cultures from 23% (7/30) of the periodontitis group and 14% (4/29) of the periodontally healthy group. This difference was not statistically significant ($p = 0.3$). Overall, viridans streptococci were found in 19% of all positive subjects, and accounted for 35% (15/43) of all microbes isolated.

Data for bacteraemia subjects

Correlations between subject or clinical indices and the occurrence of flossing-induced bacteraemia are summarized in Table 3. No significant correlations were found between any of the subject or clinical indices assessed and the occurrence of bacteraemia following flossing (Table 3). Neither the GI ($p = 0.09$), PI ($p = 0.6$), number of sites bleeding on flossing ($p = 0.2$) nor the percentage of sites bleeding on flossing ($p = 0.2$) were found to be significantly associated with the occurrence of bacteraemia and no other statistically significant predictors of outcome were identified.

Discussion

This study found that the incidence of bacteraemia caused by dental flossing was not significantly different in subjects with periodontitis (40%) when compared with those with periodontal health (41%). This incidence is comparable to the rate of bacteraemia occurring with periodontal treatment procedures in adults such as periodontal probing (Daly et al. 2001), ultrasonic scaling (Reinhardt et al. 1982, Cherry et al. 2007), subgingival irrigation (Lofthus et al. 1991) and scaling and prophylaxis (Baltch et al. 1982).

When compared with previous investigations of bacteraemia caused by flossing, the incidence found in the present study is similar to the 38% reported by Carroll & Sebor (1980) but higher than those reported by Lineberger & De Marco (1973) (20%), Ramadan et al. (1975) (18%) and Wank et al. (1976) (22%). Comparison with these previous studies is complicated by the fact that none of the studies described either the technique of flossing which was used nor was there any indication of the force utilized for flossing. Flossing of the subjects' teeth was performed by several

Table 2. Microbiological findings of those subjects with positive oral bacteraemia post-flossing

Periodontitis group			Periodontally healthy group*		
subject	30 s	10 min.	subject	30 s	10 min.
7	*Neisseria subflava*		1	*Actinomyces odontolyticus*	
10	*Streptococcus mitis*, *Neisseria subflava*		25	*Prevotella nigrescens*	
11	*Streptococcus spp*, *Neisseria* spp.	*Eikenella corrodens*	29		Bovis group streptococci
14	*Capnocytophaga sputigena*		30	*Gemella morbillorum*	
16	*Streptococcus anginosus*, *Corynebacterium* spp.	*Corynebacterium* spp.	38	Anginosus group streptococci	
17	Viridans streptococcus × 2, *Corynebacterium* spp.	*Corynebacterium* spp., Viridans streptococcus	41	Bovis group streptococci	
18	*Aggregatibacter actinomycetemcomitans*		45	Diphtheroids, *Streptococcus intermedius*	
20		*Corynebacterium* spp.	47	Anginosus group streptococci	
21	*Streptococcus* spp., *Micrococcus* spp., *Eikenella* spp.	*Streptococcus intermedius*, *Peptostreptococcus anaerobius*	50		*Neisseria mucosa*
23	Anginosus group streptococci	*Peptostreptococcus anaerobius*	51	*Gemella haemolysans*	
26	Anginosus group streptococci	*Peptostreptococcus anaerobius*	52	*Actinomyces gerencseriae*	*Micromonas micros*
35	*Neisseria subflava*	*Selemonas* spp.	54		*Streptococcus gordonii*, Diphtheroids

*One subject not included due to positive oral bacteraemia at baseline.

Table 3. Correlation between occurrence of bacteraemia following flossing and subject or clinical variables

Variable	Post-flossing bacteraemia (Spearman's correlation co-efficient)	*p* value
Group	0.17	0.2
Age	0.18	0.2
Gender	−0.08	0.5
Smoking status	−0.04	0.7
Time spent flossing	−0.04	0.8
Gingival Index	0.22	0.09
Plaque Index	0.07	0.6
% of sites bleeding on flossing	0.17	0.2
No. sites bleeding on flossing	0.17	0.2
% of sites bleeding on probing	0.16	0.2
Pocket depth	0.09	0.5
Clinical attachment loss	0.06	0.6
Self-reported daily flossing	−0.12	0.4

investigators in one study (Ramadan et al. 1975), by the subjects themselves in two studies (Wank et al. 1976, Carroll & Sebor 1980) and the remaining study did not describe whether flossing was performed by the investigator or the subject (Lineberger & De Marco 1973). One of the studies involved only four subjects meaning that it was underpowered (Carroll & Sebor 1980). Whereas previous studies used broths or agar plates for bacterial culture, the present study used the automated, continuous culture BACTEC™ system, which has been shown to be effective in detecting oral micro-organisms in bacteraemias caused by oral procedures (Daly et al. 2001, Lucas et al. 2002, Tomas et al. 2004, Kinane et al. 2005). The BACTEC™ system can detect odontogenic bacteraemia in 24–30 h (Pauli et al. 1999, Lucas et al. 2002) and thus the 14-day continuous culture used in our study would have permitted ample time for the detection of bacteria present within the blood samples. While polymerase chain reaction (PCR) has been shown to be more sensitive than BACTEC™ in the detection of oral micro-organisms in blood samples (Kinane et al. 2005), PCR does not discriminate between live and dead bacteria. However, PCR is acknowledged as an accurate and sensitive method for identification of bacterial isolates compared with the procedures used in the present study.

The finding of a 41% incidence of bacteraemia in the periodontally healthy group was unexpected. However, with the exception of Carroll & Sebor's (1980) investigation of only two periodontally healthy subjects, no previous study has investigated bacteraemia due to flossing in a periodontally healthy group. Our findings in the periodontally healthy group differ from previous studies of toothbrushing in adult subjects with periodontal health in which the incidence of bacteraemia has been found to be low (8%; Silver et al. 1979) or zero (Sconyers et al. 1979, Hartzell et al. 2005, Forner et al. 2006) regardless of whether toothbrushing was performed by the investigator (Silver et al. 1979, Sconyers et al. 1979) or by the subject (Hartzell et al. 2005, Forner et al. 2006). The 40% incidence of bacteraemia we found for flossing in the periodontitis group was similar to the 43% incidence reported for adult subjects with periodontal disease who underwent investigator-brushing (Silver et al. 1977) and also to the overall incidence of 32% found in patients with a range of periodontal indices who self-brushed (Lockhart et al. 2008). However, it was higher than that reported in other studies of toothbrushing (17%; Sconyers et al. 1973, 3%; Kinane et al. 2005, 2%; Forner et al. 2006).

The generally higher incidence of bacteraemia found for flossing as compared with toothbrushing may indicate

that flossing is not as benign a procedure as toothbrushing. The act of flossing involves mechanically disrupting the approximal and inter-proximal bacterial biofilm such that subgingival displacement of bacteria into the tissue may occur. To compound this, disruption of the junctional epithelium can also occur as the floss slides 2–3 mm below the inter-proximal gingival margin (Waerhaug 1981). In contrast, toothbrush bristles cannot reach the inter-proximal regions of teeth and are restricted to the approximal tooth surfaces (Egelberg & Claffey 1998). Thus, flossing may have a greater potential to introduce bacteria into the circulation than toothbrushing.

A similar observation has been made in relation to rubber dam placement in children in which an incidence of bacteraemia of 31.4% was found (Roberts et al. 2000). The investigators in that study ascribed this unexpectedly high incidence to the technique of rubber dam placement, which involved ''inter-proximal rubber being forced down between the teeth and at the same time pushing the dental plaque ahead of it and then squashing it onto the inflamed gingivae''. The act of flossing involves a similar action to rubber dam placement and thus tissue trauma and bacterial displacement caused by flossing may be factors accounting for a higher rate of bacteraemia than might be caused by toothbrushing. Use of inter-dental woodsticks for inter-proximal cleaning may also cause trauma and bacterial displacement and may account for the 30% incidence of bacteraemia found following the use of inter-dental woodsticks in subjects with periodontitis (Lineberger & De Marco 1973).

The incidence of flossing-induced bacteraemia found in the present study is similar to that following investigator-performed manual toothbrushing in children (39%; Roberts et al. 1997, 46%; Bhanji et al. 2002) but less than that for investigator-performed powered toothbrushing in children (78%; Bhanji et al. 2002). However, it should be noted that children may demonstrate higher rates of bacteraemia than adults due to their smaller blood volume, which means that bacteraemia can be of a higher concentration than in adults and thus more easily detectable. Children also have an immature immune system such that there may also be a difference in the rate of bacteraemia clearance as compared with adults (Yagupsky & Nolte 1990). Thus, it is not possible to directly compare the findings of the present investigation of adults with studies involving children.

In previous studies of bacteraemia due to toothbrushing, the toothbrushing has sometimes been performed by the one investigator in order to standardize the brushing action among the subjects (Silver et al. 1977, Sconyers et al. 1979). In this study, flossing was performed by the one investigator (K. C.), which ensured that a standardized, recommended technique was used in all subjects (Perry 1996, American Dental Association 2008) and that a standardized flossing force of 50 g was utilized (Smith et al. 1986). In addition, because each subject had an intravenous cannula present in one of their arms for the entire experimental visit, it was physically impossible for them to perform flossing without the cannula being removed and then re-inserted after the flossing exercise. However, it has been shown that bacteraemia peaks at 30 s following a dental procedure and so blood samples should be taken then in order to maximize chances of bacteraemia detection (Roberts et al. 1992). If subjects carried out their own flossing, the time then required to perform the venepuncture procedure and obtain the blood sample would be too long, thus lowering the chances of detecting bacteraemia. The average time taken to floss all the teeth was 4.5 min., which is similar to the average of 4 min. reported in a previous study of whole mouth, subject-performed flossing (Gjermo & Flotra 1970). The fact that flossing was investigator-performed and that the investigator could not be masked as to the subject's periodontal status are acknowledged as limitations of this study.

No clinical variables, including gingival inflammation, plaque levels or bleeding on flossing, were found to be associated with bacteraemia in this study. In contrast, the incidence of bacteraemia caused by toothbrushing has been reported to increase significantly when gingival inflammation increased (Silver et al. 1977) and a positive but weak correlation between gingival inflammation and the incidence of bacteraemia due to scaling has also been reported (Forner et al. 2006). However, the role of gingival inflammation and plaque control in bacteraemia is contentious and it is considered that it cannot be assumed that a healthy mouth reduces the risk of odontogenic bacteraemia (Wilson et al. 2007, NICE Guideline Development Group 2008). No associations have been found between bacteraemia and gingival inflammation for scaling (Cherry et al. 2007), nor for scaling and root planing (Lafaurie et al. 2007) and no associations between plaque levels and bacteraemia have been found for toothbrushing (Silver et al. 1977), periodontal probing (Daly et al. 2001), ultrasonic scaling (Cherry et al. 2007) or extractions (Wahlmann et al. 1999, Tomas et al. 2008). The finding that the presence of bleeding on flossing was not associated with an increased risk of bacteraemia is in agreement with a previous report that gingival bleeding caused by various oral hygiene and dental procedures is not predictive of odontogenic bacteraemia (Roberts 1999).

None of the periodontitis group had a positive bacteraemia of oral origin in the baseline blood sample but one of the periodontally healthy subjects did (*A. odontolyticus*) giving a baseline bacteraemia rate of 2%. Previous studies of flossing have not found any baseline, pre-flossing oral bacteraemia (Lineberger & De Marco 1973, Ramadan et al. 1975, Wank et al. 1976, Carroll & Sebor 1980) nor have any toothbrushing studies in adults (Sconyers et al. 1973, Silver et al. 1977, Sconyers et al. 1979, Silver et al. 1979, Hartzell et al. 2005, Forner et al. 2006, Lockhart et al. 2008). A baseline bacteraemia is usually absent (Heimdahl et al. 1990, Okabe et al. 1995, Cherry et al. 2007) or low (5%; Kinane et al. 2005, 2.5%; Lafaurie et al. 2007, 2%; Tomas et al. 2008). The rate of spontaneous oral bacteraemia has been reported to range from $<1\%$ in adults (Everett & Hirschmann 1977) to 9.3% for children (Roberts et al. 2000). However, in this latter study, the child subjects had undergone nasotracheal intubation for general anaesthesia that can cause bacteraemia, most likely due to trauma of the pharyngeal and tracheal mucosa (McShane & Hone 1986, Dinner et al. 1987) and so the high rate of baseline bacteraemia should be interpreted with caution.

The finding that viridans streptococci were found in positive post-flossing blood samples of 23% of the periodontitis group and 14% of the periodontally healthy group is notable because this group of bacteria is a principal pathogen in IE (Netzer et al. 2000) and the incidence of viridans

streptococcal bacteraemia (VSB) is considered an important factor in IE (Shanson 2008). The occurrence of VSB following dental flossing raises the question as to whether individuals in risk groups for IE should avoid flossing, even if they have a healthy periodontium, so as to limit their exposure to VSB. This proposal would be at odds with an epidemiological study, which found a trend for a reduced risk of IE in those who flossed daily and which recommended that patients with cardiac valvular abnormalities should be vigilant about oral hygiene (Strom et al. 2000). It would also be negated by the fact that the proportion of positive cultures found to consist of viridans streptococci in our study (35%) is less than the 48% reported in a recent study of toothbrushing in 89 subjects (Lockhart et al. 2008). The incidence of VSB caused by flossing in the present study is much lower than that reported for more invasive dental procedures such as extractions (85%; Heimdahl et al. 1990, 70%; Lockhart et al. 2008).

As well as the incidence of bacteraemia, its magnitude may also be important in the development of IE. Although the incidence of flossing-induced bacteraemia was the same between the periodontitis and periodontally healthy groups, it is possible that the magnitude of bacteraemia may have been higher in the periodontitis group. As the BACTEC™ system does not permit quantitation of bacteraemia, determining the magnitude of bacteraemia was beyond the scope of this study. However, the finding of a trend for a longer duration of bacteraemia in the periodontitis group may have been due to a higher magnitude of bacteraemia, which would require a longer period of time for clearance from the circulation. As such, daily flossing to maintain good oral health may still be important to reduce the quantity of bacteria that enter the bloodstream although there are no data to demonstrate that a greater magnitude of bacteraemia is more likely to cause IE in humans (Wilson et al. 2007).

The findings of the present study are important in light of recent changes to national guidelines, which have reduced or abolished the need for antibiotic prophylaxis for IE, based in part on the contention that ''everyday procedures'', such as daily oral hygiene, may be of more importance in the aetiology of IE than bacteraemia caused by dental procedures (Wilson et al. 2007, NICE Guideline Development Group 2008, IEP Expert Group 2008). As stated by Wilson et al. (2007), there may not be a clinically significant difference in the frequency, nature, magnitude and duration of bacteraemia associated with a dental procedure compared with that resulting from routine daily activities and so it is inconsistent to recommend prophylaxis for dental procedures but not for these same patients during routine daily procedures. The present study has shown that the incidence of bacteraemia due to flossing is similar to that for many dental procedures for which antibiotic prophylaxis is routinely given, but that the incidence of VSB is less than that caused by more invasive procedures such as tooth extraction (Heimdahl et al. 1990, Lockhart et al. 2008).

In conclusion, given the experimental constraints of this study, it would appear that dental flossing can produce bacteraemia in periodontally healthy as well as periodontally diseased individuals and that there are no patient or clinical factors that would assist the clinician in predicting who are most likely to experience such a bacteraemia. In addition, the occurrence of VSB suggests that flossing has the potential to be implicated as a causative factor in cases of streptococcal IE. Studies investigating the magnitude of bacteraemia due to flossing are required to further clarify the role of flossing in contributing to the daily cumulative exposure of an individual to odontogenic bacteraemia.

Acknowledgements

This study was funded by the Dental Board of New South Wales and the Australian Periodontology Research Foundation.

References

American Academy of Periodontology (2000a) Parameter on chronic periodontitis with advanced loss of periodontal support. *Journal of Periodontology* **71**, 856–858.

American Academy of Periodontology (2000b) Parameter on chronic periodontitis with slight to moderate loss of periodontal support. *Journal of Periodontology* **71**, 853–855.

American Dental Association (2008) Oral Health topics A-Z. Cleaning your teeth and gums (oral hygiene). How do I floss my teeth? Available at http://www.ada.org/public/topics/cleaning_faq.asp#4 [accessed on 6 July 2008].

Baltch, A.L, Schaffer, C., Hammer, M. C., Sutphen, N. T., Smith, R. P., Conroy, J. & Shayegani, M. (1982) Bacteremia following dental cleaning in patients with and without penicillin prophylaxis. *American Heart Journal* **104**, 1335–1339.

Bhanji, S., Williams, B., Sheller, B., Elwood, T. & Mancl, L. (2002) Transient bacteremia induced by toothbrushing: a comparison of the Sonicare toothbrush with a conventional toothbrush. *Pediatric Dentistry* **24**, 295–299.

Carroll, G. C. & Sebor, R. J. (1980) Dental flossing and its relationship to transient bacteremia. *Journal of Periodontology* **51**, 691–692.

Carter, H. G. & Barnes, G. P. (1974) The Gingival Bleeding Index. *Journal of Periodontology* **45**, 801–805.

Carter, H. G., Barnes, G. P., Radentz, W. H., Levin, M. P. & Bhaskar, S. N. (1975) Effects of using various types of dental floss on gingival sulcular bleeding. *Virginia Dental Journal* **52**, 18–32.

Cherry, M., Daly, C. G., Mitchell, D. & Highfield, J. (2007) Effect of rinsing with povidone-iodine on bacteraemia due to scaling: a randomized-controlled trial. *Journal of Clinical Periodontology* **34**, 148–155.

Cockerill, F., Hughes, J., Vetter, E., Mueller, R., Weaver, A., Ilstrup, D., Rosenblatt, J. & Wilson, W. (1997) Analysis of 281,797 consecutive blood cultures performed over an eight-year period: trends in microorganisms isolated and the value of anaerobic culture of blood. *Clinical Infectious Diseases* **24**, 403–418.

Daly, C. G., Currie, B. J., Jeyasingham, M. S., Moulds, R. F., Smith, J. A., Strathmore, N. F., Street, A. C. & Goss, A. N. (2008) A change of heart: the new infective endocarditis prophylaxis guidelines. *Australian Dental Journal* **53**, 196–200.

Daly, C. G., Mitchell, D. H., Highfield, J. E., Grossberg, D. E. & Stewart, D. (2001) Bacteremia due to periodontal probing: a clinical and microbiological investigation. *Journal of Periodontology* **72**, 210–214.

Dinner, M., Tjeuw, M. & Artusio, J. F. Jr. (1987) Bacteremia as a complication of nasotracheal intubation. *Anaesthetics and Analgesia* **66**, 460–462.

Duval, X. & Leport, C. (2008) Prophylaxis of infective endocarditis: current tendencies, continuing controversies. *Lancet Infectious Diseases* **8**, 225–232.

Egelberg, J. & Claffey, N. (1998) Role of mechanical dental plaque removal in prevention and therapy of caries and periodontal diseases. Consensus Report of Group B. In: Lang, N., Attstrom, R. & Löe, H. (eds). *Proceedings of the European Workshop on Mechanical Plaque Control*, pp. 169–172. London: Quintessence.

Everett, E. D. & Hirschmann, J. V. (1977) Transient bacteremia and endocarditis prophylaxis. A review. *Medicine* **56**, 61–77.

Forner, L., Larsen, T., Kilian, M. & Holmstrup, P. (2006) Incidence of bacteremia after chewing, tooth brushing and scaling in individuals

with periodontal inflammation. *Journal of Clinical Periodontology* **33**, 401–407.

Gjermo, P. & Flotra, L. (1970) The effect of different methods of interdental cleaning. *Journal of Periodontal Research* **5**, 230–236.

Hartzell, J., Torres, D., Kim, P. & Wortmann, G. (2005) Incidence of bacteremia after routine tooth brushing. *American Journal of Medical Science* **329**, 178–180.

Heimdahl, A., Hall, G., Hedberg, M., Sandgerg, H., Soder, P., Tuner, K. & Nord, C. (1990) Detection and quantitation by lysis-filtration of bacteremia after different oral surgical procedures. *Journal of Clinical Microbiology* **28**, 2205–2209.

Horstkotte, D., Follath, F., Gutschik, E., Lengyel, M., Oto, A., Pavie, A., Soler-Soler, J., Thiene, G., von Graevenitz, A., Priori, S. G., Garcia, M. A. A., Blanc, J.-J., Budaj, A., Cowie, M., Dean, V., Deckers, J., Fernandez Burgos, E., Lekakis, J., Lindahl, B., Mazzotta, G., Morais, J., Oto, A., Smiseth, O. A., Lekakis, J., Vahanian, A., Delahaye, F., Parkhomenko, A., Filipatos, G., Aldershvile, J. & Vardas, P. (2004) Guidelines on prevention, diagnosis and treatment of infective endocarditis executive summary; the task force on infective endocarditis of the European society of cardiology. *European Heart Journal* **25**, 267–276.

Hugoson, A., Sjodin, B. & Norderyd, O. (2008) Trends over 30 years, 1973–2003, in the prevalence and severity of periodontal disease. *Journal of Clinical Periodontology* **35**, 405–414.

Infective Endocarditis Prophylaxis Expert Group (2008) *Prevention of Endocarditis. 2008 update from Therapeutic Guidelines: Antibiotic. Version 13*. North Melbourne: Therapeutic Guidelines Ltd.

Jenney, A. W., Cherry, C. L., Davis, B. & Wesselingh, S. L. (2001) ''Floss and (nearly) die'': dental floss and endocarditis. *Medical Journal of Australia* **174**, 107–108.

Kinane, D. F., Riggio, M. P., Walker, K. F., MacKenzie, D. & Shearer, B. (2005) Bacteraemia following periodontal procedures. *Journal of Clinical Periodontology* **32**, 708–713.

Lafaurie, G. I., Mayorga-Fayad, I., Torres, M. F., Castillo, D. M., Aya, M. R., Baron, A. & Hurtado, P. A. (2007) Periodontopathic microorganisms in peripheric blood after scaling and root planing. *Journal of Clinical Periodontology* **34**, 873–879.

Lineberger, L. T. & De Marco, T. J. (1973) Evaluation of transient bacteremia following routine periodontal procedures. *Journal of Periodontology* **44**, 757–762.

Lobene, R. R., Weatherford, T., Ross, N. M., Lamm, R. A. & Menaker, L. (1986) A modified gingival index for use in clinical trials. *Clinical Preventive Dentistry* **8**, 3–6.

Lockhart, P., Brennan, M., Sasser, H., Fox, P., Paster, B. & Bahrani-Mougeot, F. (2008) Bacteremia associated with toothbrushing and dental extraction. *Circulation* **117**, 3118–3125.

Lofthus, J. E., Waki, M. Y., Jolkovsky, D. L., Otomo-Corgel, J., Newman, M. G., Flemmig, T. & Nachnani, S. (1991) Bacteremia following subgingival irrigation and scaling and root planing. *Journal of Periodontology* **62**, 602–607.

Lucas, V., Lytra, V., Hassan, T., Tatham, H., Wilson, M. & Roberts, G. (2002) Comparison of lysis filtration and an automated blood culture system (BACTEC) for detection, quantification and identification of odontogenic bacteremia in children. *Journal of Clinical Microbiology* **40**, 3416–3420.

Mariotti, A. (1999) Dental plaque-induced gingival diseases. *Annals of Periodontology* **4**, 7–19.

McBryde, E., Tilse, M. & McCormack, J. (2005) Comparison of contamination rates of catheter-drawn and peripheral blood cultures. *Journal of Hospital Infection* **60**, 118–121.

McShane, A. J. & Hone, R. (1986) Prevention of bacterial endocarditis: does nasal intubation warrant prophylaxis? *British Medical Journal* **292**, 26–27.

Miller, S. (1938) *Textbook of Periodontia*, 1st edition. Philadelphia: Blakiston.

Moreillon, P. & Que, Y.-A. (2004) Infective endocarditis. *Lancet* **363**, 139–149.

Murray, P., Baron, E. & Pfallar, M. (2003) *Manual of Clinical Microbiology*, 8th edition. Washington: American Society for Microbiology.

National Institute for Health and Clinical Excellence Guideline Development Group (2008) Prophylaxis against infective endocarditis: antimicrobial prophylaxis against infective endocarditis in adults and children undergoing interventional procedures. Available at http://www.nice.org.uk/CG064 [accessed on 22 March 2008].

Netzer, R. O., Zollinger, E., Seiler, C. & Cerny, A. (2000) Infective endocarditis: clinical spectrum, presentation and outcome. An analysis of 212 cases 1980–1995. *Heart* **84**, 25–30.

Okabe, K., Nakagawa, K. & Yamamoto, E. (1995) Factors affecting the occurrence of bacteremia associated with tooth extraction. *International Journal of Oral and Maxillofacial Surgery* **24**, 239–242.

Page, R. C. & Eke, P. I. (2007) Case definitions for use in population-based surveillance of periodontitis. *Journal of Periodontology* **78**, 1387–1399.

Pauli, I., Shekhawat, P., Kehl, S. & Sasidharan, P. (1999) Early detection of bacteremia in the neonatal intensive care unit using the new BACTEC system. *Journal of Perinatology* **19**, 127–131.

Perry, D. A. (1996) Plaque control for the periodontal patient. In: Newman, M. G., Takei, H. H. & Carranza, F. A. (eds). *Carranza's Clinical Periodontology*, 9th edition, pp. 660–661. Philadelphia: WB Saunders Co.

Ramadan, A. E., Zaki, S. A. & Nour, Z. M. (1975) A study of transient bacteremia following the use of dental floss silk and interdental stimulators. *Egyptian Dental Journal* **21**, 19–28.

Reinhardt, R. A., Bolton, R. W. & Hlava, G. (1982) Effect of nonsterile versus sterile water irrigation with ultrasonic scaling on postoperative bacteremias. *Journal of Periodontology* **53**, 96–100.

Roberts, G. J. (1999) Dentists are innocent! ''Everyday'' bacteremia is the real culprit: a review and assessment of the evidence that dental surgical procedures are a principal cause of bacterial endocarditis in children. *Pediatric Cardiology* **20**, 317–325.

Roberts, G. J., Gardner, P., Longhurst, P., Black, A. E. & Lucas, V. S. (2000) Intensity of bacteraemia associated with conservative dental procedures in children. *British Dental Journal* **188**, 95–98.

Roberts, G. J., Gardner, P. & Simmons, N. A. (1992) Optimum sampling time for detection of dental bacteraemia in children. *International Journal of Cardiology* **35**, 311–315.

Roberts, G. J., Holzel, H. S., Sury, M. R., Simmons, N. A., Gardner, P. & Longhurst, P. (1997) Dental bacteremia in children. *Pediatric Cardiology* **18**, 24–27.

Sandre, R. M. & Shafran, S. D. (1996) Infective endocarditis: review of 135 cases over 9 years. *Clinical Infectious Diseases* **22**, 276–286.

Sconyers, J. R., Albers, D. D. & Kelly, R. (1979) Relationship of bacteremia to toothbrushing in clinically healthy patients. *General Dentistry* **27**, 51–52.

Sconyers, J., Crawford, J. & Moriarty, J. (1973) Relationship of bacteremia to toothbrushing in patients with periodontitis. *Journal of the American Dental Association* **87**, 616–622.

Shanson, D. (2008) New British and American guidelines for the antibiotic prophylaxis of infective endocarditis: do the changes make sense? A critical review. *Current Opinion in Infectious Diseases* **21**, 191–199.

Silness, J. & Löe, H. (1964) Periodontal disease in pregnancy II. Correlation between oral hygiene and periodontal condition. *Acta Odontologica Scandinavia* **22**, 121–135.

Silver, J. G., Martin, A. W. & McBride, B. C. (1977) Experimental transient bacteraemias in human subjects with varying degrees of plaque accumulation and gingival inflammation. *Journal of Clinical Periodontology* **4**, 92–99.

Silver, J., Martin, A. & McBride, B. (1979) Experimental transient bacteraemias in human subjects with clinically healthy gingivae. *Journal of Clinical Periodontology* **6**, 33–36.

Smith, B. A., Collier, C. M. & Caffesse, R. G. (1986) In vitro effectiveness of dental floss in plaque removal. *Journal of Clinical Periodontology* **13**, 211–216.

Strom, B. L., Abrutyn, E., Berlin, J. A., Kinman, J. L., Feldman, R. S., Stolley, P. D., Levison, M. E., Korzeniowski, O. M. & Kaye, D. (2000) Risk factors for infective endocarditis: oral hygiene and nondental exposures. *Circulation* **102**, 2842–2848.

Thompson, W. & Walter, S. (1988) Kappa and the concept of independent errors. *Journal of Clinical Epidemiology* **41**, 969–970.

Tomas, I., Alvarez, M., Limeres, J., Otero, J., Saavedra, E., Lopez-Melendez, C. & Diz, P.

(2004) In vitro activity of moxifloxacin compared to other antimicrobials against streptococci isolated from iatrogenic oral bacteremia in Spain. *Oral Microbiology and Immunology* **19**, 331–335.

Tomas, I., Pereira, F., Llucian, R., Poveda, R., Diz, P. & Bagan, J. V. (2008) Prevalence of bacteraemia following third molar surgery. *Oral Diseases* **14**, 89–94.

Waerhaug, J. (1981) Healing of the dento-epithelial junction following the use of dental floss. *Journal of Clinical Periodontology* **8**, 144–150.

Wahlmann, U., Al-Nawas, B., Jutte, M. & Wagner, W. (1999) Clinical and microbiological efficacy of single dose cefuroxime prophylaxis for dental surgical procedures. *International Journal of Antimicrobial Agents* **12**, 253–256.

Walsh, T. F., Al-Hokail, O. S. & Fosam, E. B. (1997) The relationship of bone loss observed on panoramic radiographs with clinical periodontal screening. *Journal of Clinical Periodontology* **24**, 153–157.

Wank, H. A., Levison, M. E., Rose, L. F. & Cohen, D. W. (1976) A quantitative measurement of bacteremia and its relationship to plaque control. *Journal of Periodontology* **47**, 683–686.

Warren, P. R. & Chater, B. V. (1996) An overview of established interdental cleaning methods. *Journal of Clinical Dentistry* **7** (Spec. No.), 65–69.

Wilson, W., Taubert, K. A., Gewitz, M., Lockhart, P. B., Baddour, L. M., Levison, M., Bolger, A., Cabell, C. H., Takahashi, M., Baltimore, R. S., Newburger, J. W., Strom, B. L., Tani, L. Y., Gerber, M., Bonow, R. O., Pallasch, T., Shulman, S. T., Rowley, A. H., Burns, J. C., Ferrieri, P., Gardner, T., Goff, D. & Durack, D. T. (2007) Prevention of infective endocarditis: Guidelines from the American Heart Association: A Guideline from the American Heart Association Rheumatic Fever, Endocarditis and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. *Journal of the American Dental Association* **138**, 739–760.

Yagupsky, P. & Nolte, F. S. (1990) Quantitative aspects of septicaemia. *Clinical Microbiology Reviews* **3**, 269–279.

Address:
C. *G. Daly*
Faculty of Dentistry
University of Sydney
2 Chalmers St
Surry Hills 2010
Australia
E-mail: cdaly@dentistry.usyd.edu.au

Clinical Relevance

Scientific rationale for the study: Bacteraemia of oral origin is implicated in the pathogenesis of IE. This study investigated whether dental flossing caused a higher incidence of bacteraemia in health as compared with chronic periodontitis.

Principal findings: Periodontally healthy individuals experienced oral bacteraemia due to flossing at the same rate as those with untreated periodontitis. This rate was the same as that of some dental treatments for which antibiotic prophylaxis is given.

Practical implications: Dental flossing is a potential source of bacteraemia, even in periodontal health, and may therefore be implicated in cases of infective endocarditis.