doi: 10.1111/j.1365-2591.2006.01124.x

REVIEW

Endodontics and infective endocarditis – is antimicrobial chemoprophylaxis required?

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Abstract

Brincat M, Savarrio L, Saunders W. Endodontics and infective endocarditis – is antimicrobial chemoprophylaxis required? *International Endodontic Journal*, **39**, 671–682, 2006.

The purpose of this review is to evaluate the evidence implicating nonsurgical endodontic procedures in inducing infective endocarditis (IE). The literature is reviewed and findings about dental procedures that elicit bacteraemia [in particular root canal treatment (RCT)], sequelae of bacteraemia, relationship between IE and RCT and variation between antibiotic prophylaxis (AP) guidelines are highlighted. At present, there is still significant debate as to which dental procedures require chemoprophylaxis and what antibiotic regimen should be prescribed. Currently, there are insufficient primary data to know whether AP is effective or ineffective against IE. Practitioners are bound by current guidelines and medico-legal considerations. Thus, the profession requires clear, uniform guidelines that are evidence-based.

Keywords: antibiotic prophylaxis, bacteraemia, endocarditis, endodontics, guidelines.

Received 30 January 2006; accepted 31 January 2006

Introduction

It is well established that manipulation of the oral tissues may be associated with a transient bacteraemia (Bender & Montgomery 1986). This can occur in everyday life during oral hygiene measures (Everett & Hirschmann 1977) as well as during dental treatment. The amount of trauma sustained during a procedure does have a bearing on the potential for a bacteraemia to be caused and traumatic procedures such as multiple extractions have been shown to do so (Bender *et al.* 1984). Bacteraemia is usually eradicated by the reticulo-endothelial system within a few minutes and poses no threat to the healthy patient. However, some medically compromized patients may be at risk from this transient blood-borne infection, most notably infective endocarditis (IE) (Dajani *et al.* 1997). Thus, implementation of antibiotic prophylaxis (AP) has been advocated widely in an attempt to provide some degree of protection for 'at-risk' patients.

Antibiotic prophylaxis may be defined as the use of an antimicrobial agent before any infection has occurred for the purpose of preventing a subsequent infection (Gerding 1996, Titsas & Ferguson 2001). Because of the high morbidity and mortality related to IE, it has long been advised that AP is required before dental procedures likely to induce bacteraemia (Tomas Carmona *et al.* 2002). However, the incidence of IE is low and there is no evidence that AP is either effective or ineffective against IE in people at risk about to undergo an invasive dental procedure (Oliver *et al.* 2004). Some authorities have questioned the routine use of antibiotics for endocarditis prophylaxis (Strom *et al.* 1998), arguing that the adverse effects outweigh the potential benefits. Many dental procedures have

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been investigated and some have been shown definitely to produce bacteraemia and are thus thought to be implicated in IE. Others, such as nonsurgical root canal treatment, are less certain.

Literature review methodology

A literature search was conducted utilizing Pubmed, Cochrane Library, e-library and Medline. Various combinations of the following keywords were used: infective endocarditis, bacteraemia/bacteremia, endodontics, root canal treatment, rubber dam, pulpotomy, dental procedures, dentistry, antibiotics, antimicrobials and prophylaxis.

Studies that show IE during dental procedures

Strategies used to support the link amongst dental procedures, bacteraemia and subsequent infective endocarditis are:

- Use of animal models.
- Evidence that antibiotic prophylaxis preoperatively is protective against infective endocarditis with the inference that the procedure was the cause of the disease in the unprotected.
- To ascertain whether a bacteraemia is produced during a dental procedure.

The latter method is the most widely studied and will be discussed in more detail.

Bacteraemia in dentistry

It was in the early 20th century that oral bacteria were first implicated in IE (Horder 1908). Since then, interest has grown in the relationship amongst dental procedures, subsequent bacteraemia and IE. The reported incidence of bacteraemia during dental interventions ranges from 17% to 94%; these varying results have been attributed to patient selection, the procedure itself and the microbiological techniques used (Heimdahl *et al.* 1990).

The procedure itself was studied by Roberts *et al.* (1997). The study reported on 13 dental operative procedures used routinely in paediatric dentistry as to whether they caused bacteraemia. Children in each procedure group were treated under general anaesthesia and an 8-mL blood sample was taken from each patient 30 s after each procedure. For the baseline group, blood was drawn after anaesthesia had been induced but before any dental procedure was carried out. Baseline values were not investigated for all the children in the study. Two commercial blood culture systems were used and the results were expressed as the percentage of samples that yielded bacteria; no investigations were carried out to assess the microbial load following these procedures. Four of these procedures used for conservative dentistry caused bacteraemia significantly more often than the baseline value of 9.4%. In comparison, toothbrushing alone, a procedure usually carried out on a daily basis, caused a bacteraemia in 38.5% of occasions. Results are shown in Table 1.

Table 1 Incidence of bacteraemia following various dental procedures (Roberts *et al.* 1997)

Procedure	%
Baseline rate	9
Intraligamentary injection	96.6
Multiple extractions (4)	51
Single extraction	39
Mucoperiosteal flap	39
Tooth brushing	38.5
Matrix band placement with wedge	32
Rubber dam placement	29.4
Ultrasonic scaling of teeth	25
Polishing teeth	24.5
Slow-speed drill	13
High-speed drill	4

Another study (Roberts *et al.* 2000) investigated the procedures involved in a two surface restoration, to determine which, if any, was associated with a bacteraemia. These were placement of rubber dam, use of high speed and slow speed drills and placement of a matrix band and wedge. In this study, besides the broth culture system, the authors also investigated the number of colony forming units (CFU) per millilitre of blood in each procedure group. The incidence of bacteraemia for these procedures is shown in Table 2.

Table 2 Number of positive blood cultures, and total number of samples with percentage positive blood cultures and colony forming units per millilitre for each dento-gingival manipulative procedure (Roberts *et al.* 2000)

Group	Number positive/number in group	% Positive	CFU mL^{-1}
Baseline (no procedure)	5/54	9.3	1.2
Rubber dam placement	16/51	31.4	1.962
Slow drill (60 s)	6/49	12.2	0.3
Fast drill (60 s)	2/47	4.3	1.9
Matrix band and wedge	18/56	32.1	4.8

Statistical calculations showed that the placement of matrix band and wedge caused a percentage prevalence of bacteraemia significantly greater than the other procedures. However, there were no statistically significant differences in the microbial load between the groups. The conclusions from this study were that the placement of rubber dam and a matrix band with a wedge resulted in a bacteraemia comparable with that encountered following a dental extraction, thus providing evidence that these procedures should be covered by AP. However, this similarity was related to the percentage incidence of samples that yielded bacteria and did not take into account the number of CFU in the original sample.

Roberts (2004) recommends AP only for procedures, where it has been shown that there is a statistically significant difference in bacteraemia between pre- and post-procedure blood samples. Dental procedures associated with bleeding are no longer exclusively indicated for AP as many procedures cause bacteraemia without discernible bleeding (Roberts 2004).

The cumulative exposure to bacteraemia is significantly greater from everyday procedures such as tooth cleaning and mastication when compared with dental operative procedures. It is far more likely that such everyday procedures are the cause of bacterial endocarditis caused by oral organisms because the cumulative exposure is often up to 10^6 times greater than those occurring following surgical procedures such as extraction (Roberts 1999). Bacteraemia generated during dental procedures usually contain not more than 10^3 CFU mL^{-1} of blood (Everett & Hirschmann 1977). This is in contrast to animal studies linking bacteraemia and IE, where the concentration of organisms is artificially high, typically in the region of 10^5–10^8 CFU mL^{-1} (Glauser & Francioli 1987). This microbial load of bacteraemia has been shown to be an important factor in the genesis of experimental animal endocarditis (Roberts 1999) and thus extrapolation of experimental animal data to the clinical setting is difficult. In a study by Al-Karaawi *et al.* (2001), the cumulative exposure to bacteraemia from dental procedures currently recommended for AP in the American Heart Association (AHA) Guidelines 1997 was compared with the cumulative exposure for dental procedures for which AP was not recommended. High cumulative exposures were obtained for dento-gingival manipulative procedures not currently recommended for AP. This was especially so for rubber dam placement.

Bacteraemia in nonsurgical root canal treatment

A number of studies have been carried out to assess whether root canal treatment produces significant bacteraemia. Many of the early clinical reports of the link between endodontic treatment and bacteraemia are anecdotal, lack the use of an aseptic technique during treatment and do not match the organisms isolated from the bloodstream to those in the root canal (Ross & Rogers 1943, Bender *et al.* 1960, Trivedi 1984, Bender & Montgomery 1986, Green & Haisch 1988). In other studies, the laboratory procedures used were deficient in that samples were cultured only aerobically and in one such study (Robinson *et al.* 1950), no bacteria were detected in the bloodstream following preparation and filling of seven root canals. Endodontic procedures with instrumentation beyond the apex were shown by Bender *et al.* (1963) to produce detectable bacteraemia in 31% of cases, but, when instrumentation was confined within the tooth, blood cultures were negative.

Bacteraemia studies in endodontics with improved techniques

It was not until 1976 that a study applying an aseptic technique and improved anaerobic culture media was published (Baumgartner *et al.* 1976). The authors were able to conclude that no bacteraemia was elicited if instrumentation was kept within the root canal. In a follow-up study, Baumgartner *et al.* (1977) found a detectable bacteraemia in only one of the 30 patients undergoing nonsurgical root canal treatment, with an incidence of 3.3% as opposed to 83.3% following flap retraction, 33.3% following periapical curettage and 100% following dental extraction. In studies where more selective techniques for culturing microorganisms were used, the incidence of bacteraemia has been reported as up to 20% after nonsurgical endodontic treatment, where instrumentation was confined to the root canal (Heimdahl *et al.* 1990). The authors of this study reported that endodontic treatment produced 0.2 CFU mL^{-1} blood compared with 2.19 CFU mL^{-1} during third molar extraction. A series of studies (Debelian *et al.* 1992, 1995, Debelian *et al.* 1996) found no statistically significant difference between the incidence of bacteraemia following instrumentation within and outwith the root canal. Gram-positive anaerobes were the most common isolates from the blood samples whereas Gram-negative anaerobes were

the most likely obtained from the root canal. These studies suggested that a bacteraemia might be produced during root canal treatment. The results were repeated with ribotyping using DNA hybridization method (Debelian *et al.* 1997) and then using both phenotypic and genetic methods (Debelian *et al.* 1998). A bacteraemia was elicited in 31–54% of root canal treatments. The incidence of bacteraemia from root canal treatments was far greater in these studies than previous investigations, as more sensitive culturing and identification techniques were applied. In another recent study, 30 patients receiving nonsurgical root canal treatment were studied (Savarrio *et al.* 2005). A detectable bacteraemia was present in 30% of the patients following conventional culturing, whose preoperative control blood sample was negative. In 23.3% of patients, the same species of organism was identified in both the bloodstream and in the paper point sample from the root canal system. Pulsed field gel electrophoresis was used to identify the genetic homogeneity between the organisms. Blood samples were also analysed for the presence of bacterial DNA by polymerase chain reaction (PCR). This gave a lower detection rate when compared with conventional culture with only 11% of the blood samples displaying bacterial DNA. Poor sensitivity of PCR is consistent with reports from other studies that have highlighted specific problems when using PCR to detect bacteraemia. These include the blood volume analysed by PCR, which is much smaller than that for conventional culturing (Roth *et al.* 1999), blood sample handling and preparation (Hryniewiecki *et al.* 2002) and the fact that where blood specimens are <40 organisms per millilitre PCR is not as sensitive as BACTEC culture (MacGregor *et al.* 1999). Also, methodological variation in blood collection could also contribute. In this study, blood was collected in EDTA tubes, but another method has been described whereby tryptic soya broth was added to the samples producing an equal rate of detection between conventional culturing and PCR (Jordan & Durso 2000). Again, the conclusions from this study were that nonsurgical RCT might invoke a detectable bacteraemia.

Bacteraemia and rubber dam placement

One other area of difficulty is to assess whether bacteraemia occurs during placement of rubber dam clamps or whether it is related to their movement during treatment. In one study of patients under general anaesthesia, the incidence of bacteraemia reported during rubber dam clamp placement was 29% (Roberts *et al.* 1997). This was higher than the 25% of patients who displayed a bacteraemia during ultrasonic scaling, but lower than the 38.5% who displayed a bacteraemia following tooth brushing. The percentages of positive samples in these groups were statistically significantly greater than that in the baseline group. In another study (Roberts *et al.* 2000), rubber dam placement was again investigated. Statistical calculations showed that the placement of rubber dam caused a percentage prevalence of bacteraemia (31.4%) greater than the baseline (9.3%) or the use of either fast or slow speed drills (12.2% and 32.1%, respectively). However, there were no statistically significant differences in the intensity of bacteraemia between these groups.

Bacteraemia in pulpotomy

When considering pulpotomies, there is a lack of experimental data. If there is no certainty that a bacteraemia is induced during root canal treatment, then it would seem unlikely that a pulpotomy procedure should cause microorganisms to enter the bloodstream. In a study looking at the link between one-step formocresol pulpotomy and bacteraemia, a 4% incidence of bacteraemia was found, although the authors pointed out that this is less than most other dental procedures and roughly equivalent to the reported frequency of spontaneous bacteraemia (Farrington 1973).

Sequelae of bacteraemia

Bacteraemia of dental origin have been linked to many systemic diseases. For review see Murray & Saunders (2000). One of the most serious outcomes of bacteraemia of dental origin is IE. IE is associated with substantial morbidity and mortality (Ramsdale *et al.* 2004), despite improved techniques to aid diagnosis and modern antibiotics and surgical therapies. It affects individuals with structural defects who develop bacteraemia possibly as a result of dental, gastrointestinal, genitourinary, respiratory or cardiac invasive/surgical procedures (Ramsdale *et al.* 2004). The incidence of IE is low and the proportion of cases arising as a result of dental treatment is arguable, estimated to be as low as 4% (Guntheroth 1984, Gendron *et al.* 2000) and as high as 64% of cases of IE (Bennis *et al.* 1995). Although dental procedures are commonly implicated in the aetiology of endocarditis, the number of cases where the sequential relationship can be demonstrated ranges only between 4% and 7.5% of cases (Gendron *et al.* 2000).

Infective endocarditis and dentistry

The relationship between dental procedures and IE has been supported by anecdotal clinical reports since 1908 (Robinson *et al.* 1950, Durack 1985) and by animal experimental data (Bahn *et al.* 1978). There is increasing evidence that spontaneous bacteraemia is more likely to cause IE in 'at-risk' patients than specific dental procedures (Seymour *et al.* 2000). Two recent studies concluded that dental treatment was not a risk factor for IE (Lacassin *et al.* 1995, Strom *et al.* 1998). It is now generally accepted that the majority of cases of IE are not caused by invasive procedures (Durack 1994, Dajani *et al.* 1997). Despite the use of AP in individuals at risk of IE undergoing invasive dental procedures the incidence of the disease has not altered (Durack 1994). Furthermore, this is against a background of a dramatic rise in the numbers of people receiving artificial heart valves (Seymour *et al.* 2000).

Infective endocarditis and nonsurgical endodontics

Up to 1953, no reported cases of IE traceable to root canal therapy had been described (Kolmer 1953). There then emerged several publications recording possible links. In a review of 4281 published cases of IE, in which an alleged source of infection was identified, dental procedures were implicated in 637 (Mc Gowan 1982). Out of these, only seven cases were traced with 'fillings' alone (Harvey & Capone 1961, Eisenbud 1962, Doyle *et al.* 1967, Croxson *et al.* 1971). Only one of these was associated with the development of endocarditis following root canal treatment 2 months previously in a patient not given prophylaxis. Blood cultures revealed *Staphylococcus aureus* with the same antibiotic sensitivity as the strain isolated from the apex of the tooth in question 1 week after admission to hospital (Eisenbud 1962). In a review of 53 cases of IE following dental procedures, seven were attributed to previous RCT. In all cases, there was clear evidence of extracanal instrumentation, mainly through the apical foramen (Martin *et al.* 1997). In a large case–control study (van der Meer *et al.* 1992a,b), three cases of IE were found which were apparently attributed to root canal treatment based on the premise that the infecting organism was consistent with those inhabiting the root canal system and also that the patient had had endodontic treatment in the last 30 days. This was out of a total of 349 patients who had native valve IE, giving the rate of RCT aetiology as 0.86%.

Patients at risk for IE

In the past, the majority of patients who developed IE had a known pre-existing cardiac defect. More recently, this trend has shifted with nearly half of the cases of endocarditis having no known previous cardiac disease (Hoen *et al.* 2002). Some patients with no known heart disease may also develop IE, particularly children up to the age of 2 years and i.v. drug abusers (Durack 1994). Common cardiac conditions at risk include previous endocarditis, prosthetic heart valves, valvular stenosis, ventricular septal defect and valvular damage following rheumatic fever. Some of these conditions have a higher risk of developing endocarditis, namely previous endocarditis and prosthetic heart valves (Durack 1994). The American Heart Association (AHA) and the British Cardiac Society (BCS) Guidelines have stratified the cardiac conditions into high, moderate and negligible risk categories based on the potential if endocarditis develops. The variations between the guidelines with regard to which conditions fall into high and moderate risk categories are shown in Table 3. On the other hand, the latest guidelines from the British Society for Antimicrobial Chemotherapy (BSAC) did not stratify the cardiac conditions into categories and have restricted AP to patients who have a history of previous endocarditis, or who have had cardiac valve replacement surgery, or those with a surgically constructed pulmonary shunt or conduit. They recommend that the current practice of giving patients AP prior to dental treatment be stopped for all patients with cardiac abnormalities, except for the above mentioned conditions. These conditions cause changes in the surface of the endocardium or changes in the blood flow, which may damage the endocardium, thus enabling organisms in the blood to adhere and multiply forming bacterial vegetations. This may lead to a severe systemic illness as well as direct effects on heart function (Oliver *et al.* 2004). Without antibiotic therapy, IE is fatal (Durack 1994). The most common and important complication is heart failure because of the direct effects of the proliferating vegetations on the heart valves, which are eventually destroyed. Embolism of fragments of the vegetation can damage organs and tissues including the brain, lung, coronary arteries, spleen and the extremities of the limbs (Durack 1994).

Table 3 High (H) and moderate (M) risk categories: differences between guidelines

	British Cardiac Society (BCS) Guidelines (Ramsdale *et al.* 2004)	American Heart Association (AHA) Guidelines (Dajani *et al.* 1997)	European Society of Cardiology Guidelines (Horstkotte *et al.* 2004)	BNF (March 2006)
Previous IE	H	H	H	H
Mitral valve prolapse with regurgitation or thickened valve leaflets	**H**	M	M	M
Prosthetic heart valves	H	H	H	**M**
Complex cyanotic heart disease	H	H	H	Not Specified
Surgically constructed pulmonary shunts	H	H	H	Not Specified
Acquired valvular heart disease	M	M	M	M
Noncyanotic congenital heart disease	M	M	M	M
Hypertrophic obstructive cardiomyopathy	M	M	M	Not Specified

Antibiotic prophylaxis

It has long been advised that AP is required before dental procedures likely to produce a bacteraemia because of the high morbidity and mortality related to IE. Two mechanisms are thought to be involved. First, a reduction in the number of organisms in the blood and second, a reduction in the adhesion of organisms to the nonbacterial thrombotic vegetation (Glauser *et al.* 1983). The efficacy of AP is mainly based on animal models and clinical experience (Bor & Himmelstein 1984, Clemens & Ransohoff 1984, Durack 1990). The protective efficiency of antibiotics is only 49% (Pallasch 1989) and many cases of antibiotic failure have been reported (Durack *et al.* 1983, Denning *et al.* 1984, Green & Haisch 1988, van der Meer *et al.* 1992a,b). Strom *et al.* (1998) also concluded that 100% compliance in providing AP would reduce the incidence only marginally and the current policies for prophylaxis should be reconsidered.

They argue that the adverse effects of antibiotics outweigh the potential benefits and indeed one study has stated that patients receiving penicillin may be five times more likely to die from anaphylactic reaction than from endocarditis (Bor & Himmelstein 1984). However, this study only considered people with mitral valve prolapse who are not at increased risk of endocarditis.

Another area of concern is the escalating problem of antimicrobial resistance (Lockhart *et al.* 2002). In a case control study (van der Meer *et al.* 1992a,b), which included all the people in the Netherlands that developed endocarditis following an invasive dental procedure, there was no conclusive evidence as to whether antibiotic prophylaxis was effective or ineffective against IE in high-risk individuals about to undergo an invasive dental procedure (van der Meer *et al.* 1992a,b). The authors also concluded that the majority of cases of IE developed spontaneously and were not associated with a procedure-induced bacteraemia.

In line with these concerns, the Working Party for the BSAC considered that despite the lack of evidence of the benefit for AP to prevent IE associated with dental procedures, many clinicians would be reluctant to accept the radical, but logical, step of withholding AP for dental procedures (Gould *et al.* 2006).

Adjunctive prophylactic measures

In addition to antibiotic prophylaxis of IE, other methods of reducing bacteraemia from an oral origin have been sought. The use of pre-surgical 1% povidone-iodine has been demonstrated to cause significant reduction in bacteraemia from oral sources (Rise *et al.* 1969, Scopp & Orvieto 1971), although routine use may provoke the selection of resistant microorganisms (Park & Hart 1994). In a double-blind study of 60 patients who participated in pre-extraction rinsing with 1% (v/v) chlorhexidine, 1% (v/v) povidone-iodine and a control of NaCl, a significant reduction in bacteraemia between both antimicrobials and the control was shown (MacFarlane *et al.* 1984). However, there was no difference between the two antiseptics. A positive bacteraemia was reported in 40% and 25% of the povidone-iodine and chlorhexidine rinsers, respectively. A further study (Lockhart 1996), however, showed no difference in the rates of bacteraemia with 0.2% topical chlorhexidine treatment. The AHA recommends chlorhexidine or povidone-iodine mouthrinses, whilst the BSAC recommends the administration of chlorhexidine gluconate mouthwash 0.2% held for 1 min in the mouth, before dental treatment in patients who are susceptible to IE.

Guidelines

Various guidelines have been proposed for AP, although it has not been possible to perform controlled clinical trials in human beings to establish their effectiveness, because of ethical issues of withholding AP from patients. The recommendations for AP are modified periodically on the basis of experimental models, pharmacokinetic studies, bacterial susceptibility tests, retrospective clinical series, studies on procedure related bacteraemia and studies on the efficacy of AP. Current guidelines from the British Cardiac Society (BCS) (Ramsdale *et al.* 2004), the AHA (Dajani *et al.* 1997) and the BSAC (Gould *et al.* 2006) differ with regard to which antibiotic regimens should be prescribed and for which dental procedures.

Which nonsurgical endodontic procedures require AP?

Both the AHA and the BCS have established a precise description of the dental procedures requiring AP prior to treatment. On the other hand, the BSAC recommends AP for all dental procedures involving dentogingival manipulation or endodontics. With regard to nonsurgical endodontics, the AHA and the BCS only recommend AP if root canal instrumentation is beyond the apex, whilst the BSAC guidelines stipulate endodontic procedures in general. When it comes to rubber dam, matrix band and wedge placement and nonvital pulpotomy of the primary molar, the AHA, BCS and BSAC guidelines disagree. These procedures have been included in the recommended prophylaxis procedures in the last updated guidelines of the BCS 2004 and BSAC 2006, (although it could be argued that not all rubber dam application would require dentogingival manipulation), but not AHA guidelines. The variations between these guidelines for nonsurgical endodontic procedures are shown in Table 4.

Recommended AP regimen for nonsurgical endodontic treatment under local anaesthesia

All the above-mentioned guidelines agree that the first choice antibiotic in adult patients not allergic to penicillin is Amoxicillin. Clindamycin 600 mg is the antibiotic recommended in patients allergic to penicillin. The AHA also recommends Cephalexin, which is a first generation cephalosporin as an alternative agent to amoxicillin even though 5–10% of patients that are penicillin allergic are also allergic to cephalosporins. Azithromycin 500 mg oral suspension is recommended as an alternative in patients that are unable to take oral medication or patients that are allergic to penicillin. The available evidence from animal models on IE supports the efficacy of this drug as a prophylactic agent against oral streptococci (Girard *et al.* 1993, Rouse *et al.* 1997, Tsitasika *et al.* 2000).

Variations between guidelines

The AHA no longer makes distinctions between high and moderate risk patients when recommending a

Table 4 Variation between current guidelines with regard to dental procedures that require chemoprophylaxis in patients at risk. (Key: recommended AP: Yes; not recommended: no; not specified: NS)

Procedure	BCS Guidelines (Ramsdale *et al.* 2004)	AHA Guidelines (Dajani *et al.* 1997)	British Society for Antimicrobial Chemotherapy (BSAC) (Gould *et al.* 2006)	European Society of Cardiology (Horstkotte *et al.* 2004)
Rubber dam placement	Recommended (YES)	Not recommended (NO)	If dento-gingival manipulation	Not specified (NS)
Matrix band and wedge placement	YES	NO	YES	NS
Gingival retraction cord placement	YES	NO	YES	NS
Root canal instrumentation beyond apex	YES	YES	YES	NS
Avulsed tooth reimplantation	YES	YES	YES	YES
Nonvital pulpotomy of primary molar	YES	NO	YES	NS
Mucoperiosteal flap to gain access to tooth or lesion	YES	YES	YES	YES
Vital pulpotomy of primary molar	NO	NO	YES	NS
Pulpotomy of permanent tooth	NO	NO	YES	NS

treatment regime. Irrespective of the type of underlying cardiac condition, the AHA recommends that oral amoxicillin be taken 1 h before the procedure. This move towards oral administration may result in a reduction in both cost and risk of anaphylactic reaction whilst increasing compliance and efficacy (Sullivan 1982, van der Meer *et al.* 1992a,b). The Working Party for the British Society for Antimicrobial Chemotherapy (BSAC) has also agreed that a single oral dose will achieve adequate serum levels where AP is required (April 2006). In an attempt to reduce the adverse gastrointestinal effects of high-dose amoxicillin whilst still maintaining effective plasma levels, the AHA has revised its recommended oral dose of amoxicillin from 3 to 2 g. Following this change, members of the BSAC Endocarditis Working Party claimed that there is likely to be little difference in efficacy between the 2 and 3 g amoxicillin doses (Littler *et al.* 1997). However, they also stated that a theoretical advantage of the 3 g dose is that higher serum concentrations of amoxicillin might be expected in some individuals after 10–12 h. This extended period of cover may be useful if additional risk factors are present, such as a strain of viridans *streptococcus* with a reduced susceptibility to amoxicillin. Also, in a study by Littner *et al.* (1986), the 3 g Amoxicillin sachet had a good pharmacodynamic profile and patient acceptability, and this influenced the BSAC Endocarditis Working Party to recommend the 3 g dose. Thus, the BSAC and the BCS still call for 3 g. As a single dose provides plasma levels above the minimum inhibitory concentrations of most oral streptococci and prolonged inhibitory activity against such strains, since 1997, the AHA has eliminated a second dose of amoxicillin, given 6 h postoperatively. The BSAC protocol has advocated the use of a single dose since 1982. This change will reduce cost, the risk of allergic reaction and the potential of microbial resistance associated with the administration of a second dose (Doern *et al.* 1996). The AHA recommends i.v. AP only in patients who are unable to take oral medication. The BSAC Guidelines recommend antibiotic admistration via an i.v. route only where it is logicially easier. On the other hand, the BCS still recommend i.v. AP for patients that fall into high-risk categories, for example, patients with a history of IE, but the recommended dose of amoxicillin pre- and post-surgically is different. The current recommendations of these guidelines are shown in Tables 5 and 6.

Conclusion

There are currently insufficient primary data to know whether AP is effective or ineffective against IE, other serious illness or death in people 'at-risk' who are about to undergo a dental procedure such as root canal treatment. Some authors claim that the risk of

Table 5 Variation between current guidelines with regard to antibiotic regimens that should be prescribed for at risk adult patients undergoing dental procedures under local anaesthesia

Clinical situation	AHA Guidelines (Dajani *et al.* 1997)	British Cardiac Society Guidelines (Ramsdale *et al.* 2004)	BSAC (Gould *et al.* 2006)
1. Standard general prophylaxis for high and moderate risk patient	Amoxicillin 2 g orally 1 h pre-procedure	Amoxicillin 3 g orally 1 h pre-procedure except patients with a history of infective endocarditis (IE)	Amoxicillin 3 g orally 1 h pre-procedure
2. Patient is unable to take oral medications	Ampicillin 2 g i.m. or i.v. 30 min pre-procedure	Azithromycin 500 mg oral suspension1 h pre-procedure	Azithromycin 500 mg oral suspension 1 h pre-procedure
3. Patient is allergic to penicillin	Clindamycin 600 mg orally 1 h pre-procedure Or Cefadroxil or Cephalexin 2 g orally 1 h pre-procedure Or Azithromycin oral suspension or Clarithromycin 500 mg 1 h pre-procedure	Clindamycin 600 mg 1 h pre-procedure	Clindamycin 600 mg 1 h pre-procedure
4. Patient is allergic to penicillin and is unable to take oral medication	Clindamycin 600 mg i.v. 30 min pre-procedure Or Cefazolin 1 g i.m. or iv 30 min pre-procedure	NS	Azithromycin 500 mg oral suspension 1 h pre-procedure

Table 6 Variation between current guidelines with regard to antibiotic regimens that should be prescribed for high-risk adult patients undergoing dental procedures under local anaesthesia

Clinical situation	AHA Guidelines (Dajani *et al.* 1997)	BCS Guidelines (Ramsdale *et al.* 2004)	BSAC (Gould *et al.* 2006)
1. Patients with previous IE	Amoxicillin 2 g orally 1 h pre-procedure	Amoxicillin 2 g i.v. + Gentamicin 1.5 mg/kg iv < 30 min pre-procedure + Amoxicillin 1 g iv or orally 6 h post procedure	Amoxicillin 3 g orally 1 h pre-procedure
2. If allergic to penicillin	Clindamycin 600 mg iv 30 min pre-procedure Or Cefazolin 1 g im or iv 30 min pre-procedure	Vancomycin 1 g i.v. over 2 hr, 1–2 h pre- procedure + Gentamicin 1.5 mg/kg iv < 30 min pre-procedure Or Clindamycin 300 mg iv < 30 min pre-procedure then iv clindamycin 150 mg 6 h later	Clindamycin 600 mg orally 1 h pre-procedure Or Azithromycin 500 mg oral suspension

inappropriate use of antibiotics and widespread antibiotic resistance appear to be far more important than any possible perceived benefit (Tong & Rothwell 2000). There is still significant debate as to who is 'at-risk' from dental-induced bacteraemia and which procedures require chemoprophylaxis. The literature shows that nonsurgical endodontics might invoke a detectable bacteraemia (Baumgartner *et al.* 1977, Heimdahl *et al.* 1990, Debelian *et al.* 1997, 1998, Savarrio *et al.* 2005), with a 0.86% risk of causing native valve IE (van der Meer *et al.* 1992a,b). On the other hand, placement of rubber dam has been shown to cause a percentage prevalence of bacteraemia statistically significantly greater than the baseline or the use of fast or slow drills (Roberts *et al.* 2000). Conversely, in a recent case–control study, dental treatment was not found to be a risk factor for IE (Strom *et al.* 1998).

Prophylaxis against IE should primarily be concerned with the maintenance of good oral hygiene and prevention of oral disease to reduce the magnitude and frequency of spontaneous bacteraemia (Longman *et al.* 1993). Because of the increasing evidence that spontaneous bacteraemia is more likely to cause IE than dental procedures (Seymour *et al.* 2000), the importance of soft tissue health as a prophylactic measure for IE cannot be overstated (Lavelle 1996). Ethically, practitioners need to discuss the potential benefits and harms of antibiotic prophylaxis with their patients before a decision is made about antibiotic administration (Oliver *et al.* 2004). There is a problem in that practitioners feel that they are bound by current guidelines and medico-legal considerations, to provide antibiotic prophylaxis, rather than to make decisions based on best evidence. The profession does require clear, uniform guidelines that are evidence-based (Al-Karaawi *et al.* 2001) and in this regard new BSAC guidelines have made a welcome move away from blanket antimicrobial prophylaxis for anyone vaguely at risk from dental bacteraemia. They hint that further reductions in recommended prophylaxis may be inevitable. In spite of this, further research is required in order to inform decision-making regarding whether AP should be prescribed prior to dental treatment as well as to ascertain its effectiveness.

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