PUBLISHED VERSION

Scott, J.; Morgan, D.; Avent, M.; Graves, S.; Goss, Alastair Norman

<u>Patients with artificial joints: do they need antibiotic cover for dental treatment?</u> Australian

Dental Journal, 2005; 50(Suppl.2):S45-S53

PERMISSIONS

This document has been archived with permission from the Australian Dental Association, received 18th January, 2007.

Australian Dental Association: http://www.ada.org.au/

http://hdl.handle.net/2440/16810

Patients with artificial joints: do they need antibiotic cover for dental treatment?

JF Scott,* D Morgan,† M Avent,‡ S Graves,§ AN Goss

Abstract

This study reviews whether patients with artificial joints need antibiotic cover for dental treatment. Generally in Australia the practice has developed of giving most patients with artificial joints antibiotic prophylaxis for a wide range of dental procedures. This is partly on anecdotal grounds, partly historical and partly for legal concerns. It has been encouraged by some guidelines. Scientifically, the risk and the benefit of each step in the process needs to be analysed. This review shows that the risk of an artificial joint becoming infected from a bacteraemia of oral origin is exceedingly low whereas the risk of an adverse reaction to the antibiotic prophylaxis is higher than the risk of infection. If all patients with artificial joints receive antibiotic prophylaxis then more will die from anaphylaxis than develop infections. Factors which balance the risk benefit are if the patient is seriously immunocompromised, if the joint prosthesis is failing or chronically inflamed and if the dental procedures, such as from extractions and deep periodontal scaling, produce high level bacteraemias. Recommendations to rationalize antibiotic prophylaxis for patients with artificial joints are presented.

Key words: Artificial joints, antibiotic prophylaxis, dental treatment.

Aust Dent J 2005;50 Suppl 2:S45-S53

INTRODUCTION

'Patients with artificial joints: do they need antibiotic cover for dental treatment?' The traditional orthopaedic answer and, to a lesser extent, dental answer to this question is in the affirmative. The basis to this tradition is partly anecdotal, partly historical and partly legal. Anecdotally there have been isolated cases of joint infection related to dental disease or treatment. Historically, at the turn of the last century, focal infection theory held that many illnesses were caused by foci of infection within localized areas in the body causing systemic illness. One of the key sources of focal infection was the teeth and widespread dental extraction was the main outcome. The basis of this theory was by the process of anachoresis which is the

preferential deposit of bacteria which have localized out of the bloodstream into areas of inflammation.⁴ The legal involvement relates to the warnings which come with most American manufactured orthopaedic implants to the effect that antibiotics should be given for all dental treatment for patients receiving the implant. This broad advice is given partly for commercial but largely for legal concerns.

However, like all traditions in health, these need to be subject to evidence-based risk benefit analysis to test whether in fact they are true. After an overview of the current status of orthopaedic implants, the following steps require analysis (Fig 1): (1) Can bacteria enter the bloodstream from oral pathology or dental treatment? (2) Under what circumstances do oral bacteria settle in distant parts of the body? (3) Can orthopaedic implants become infected by blood-borne oral bacteria? (4) Can oral bacteria be prevented from entering the bloodstream or from infecting orthopaedic implants by the prescription of antibiotics? (5) What are the risks of antibiotic therapy?

The purpose of this review is to perform a risk benefit analysis of the evidence for the indications to prescribe antibiotic cover for dental treatment in patients with artificial joints. On this basis evidencebased management recommendations are made.

Overview of the current status of orthopaedic implants

Joint replacement is a proven cost-effective medical procedure. From small beginnings with hip replacements in the 1950s, it has greatly expanded to include the knee, ankle, shoulder, elbow and finger joints. Generally these joint replacements are successful with an over 90 per cent success rate over a 10-year period.6 The effectiveness of joint replacement coupled with the increasing age of the population has resulted in ever expanding numbers of artificial joints being placed. For example, in the United States in 1995, 243 919 total knee replacements were performed.⁶ In Australia in the financial year 2002-2003, there were 27 833 hip replacements and 28 003 knee replacements. The total number of hip and knee replacements has increased from 32 006 in 1994-1995 to 55 836 in 2002-2003. This represents an increase of 74.5 per cent during the eight-year period.7

Placement of artificial joints requires strict attention to detail with full evaluation of the patient, careful selection of the prosthesis and full informed consent. Patients specifically should be checked for sources of

^{*}Oral and Maxillofacial Surgeon, Private Practice, Adelaide.

[†]Chair, Arthroplasty Group, Australian Orthopaedic Association. ‡Specialist Pharmacist, Antibiotic Utilisation, Pharmacy Department, Royal Adelaide Hospital.

[§]Director, National Joint Replacement Registry, Australian Orthopaedic Association.

^{||}Oral and Maxillofacial Surgeon, The University of Adelaide; Director, Oral and Maxillofacial Surgery Unit, Royal Adelaide Hospital and Adelaide Dental Hospital.

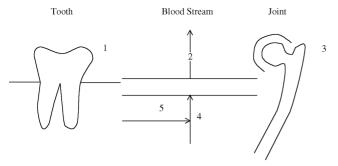


Fig 1. Schematic representation of the steps involved in an analysis of the risk and benefits of antibiotic prophylaxis to prevent infection of artificial joint replacements by oral bacteria.

potential bacteria or contamination of skin, oral, dental, bowel or urinary origin and, where possible, treated pre-operatively. Once the decision has been made to proceed, patients are usually admitted on the day of surgery. In theatre considerable effort is made to reduce the likelihood of operative infection. All patients have full surgical site antimicrobial preparation and are given high doses of intravenous prophylactic antibiotics at the commencement of the operation. Intravenous antibiotic prophylaxis with cephazolin is administered at induction of the procedure and continued for 24



Fig 2a. Total joint operating theatre.



Fig 2b. Intra-operative picture of a total knee replacement.



Fig 3a. Total knee replacement - lateral view.

hours. If the patients have a type I allergy to penicillin or cephalosporins, or if they are at high risk for MRSA colonization or infection, vancomycin should be used instead of cephazolin.^{8,9} The operation should be performed by experienced surgical personnel in a dedicated operative suite (Figs 2a and 2b). The theatre should be equipped with vertical laminar flow units or at the very least be a high flow theatre. It is also common for operating staff to wear body exhaust suits, although the evidence for the advantage of this when all other techniques aimed at reducing infection are utilized remains uncertain. In addition, in procedures where prothesis requires bone cement (methylmethacrylate) fixation, it is common to use antibiotic containing bone cement.

Placement of artificial joints involves hard and soft tissues reactions to the surgery and to the implant. In broad terms the bone reaction follows the same stages of healing as for bone fractures, namely, initial haematoma formation, an inflammatory reaction, callus formation, bone remodelling and new bone formation.¹⁰ In general terms and in the absence of complications the process is largely complete within



Fig 3b. Total knee replacement - AP view.

two to three months although subtle bone remodelling continues for 12-24 months (Figs 3a and 3b).

Joint replacements may fail for a multiplicity of reasons which include: infection, both early and late; loosening of the joint components; fracture of the joint components; wear of the prosthetic articular surface; excessive inflammatory reaction to wear particles; and bone fracture and iatrogenic factors relating to the surgical technique.¹¹ All of these factors have been exhaustively analysed but apart from infection will not be considered further in this study.

A number of comorbidities have been identified that increase the risk of implant failure due to infection. Key ones are immunosuppressive therapy, immunosuppressive conditions such as diabetes mellitus, rheumatoid arthritis, cancer, general poor medical condition, obesity and smoking. Also, the younger the patient at the time of first implantation, the greater the chance it will fail, probably because of increased activity before the patient's death of other causes. When a joint replacement fails it will have significant consequences to the patient in terms of pain, disability, greater risks associated with any subsequent surgery and increased costs both to the patient and the health



Fig 4a. Failed septic knee replacement – lateral view.

care system (Figs 4a and 4b). Failed joints require careful analysis of the reason for failure. Treatment options will depend on the reason for failure, including removal of the joint, revision and replacement of part or all of the joint, joint fusion for failed knee replacement or rarely amputation of the limb. In general there are significantly greater and more complex problems associated with the management of failed artificial joints than with the primary placement. 8,9 Revision surgery has a much higher risk of complication including infection and an increased mortality risk.

Interestingly to the dental profession, attempts at replacing the temporomandibular joint with its complex anatomy, small size but high load, have been much less successful than replacement of limb joints.



Fig 4b. Failed septic knee replacement - AP view.

Currently there is no routinely available temporomandibular joint for implantation although a number are under research evaluation.

Can bacteria enter the bloodstream from oral pathology and dental treatment?

There is substantial soundly-based scientific literature on this. 12,13 Clinical research protocols involve the placement of intravenous access with microbiological cultures at fixed times following physiological or treatment events. Oral bacteria clearly

do enter the bloodstream during chewing, teeth clenching and toothbrushing although the amounts are small and transient.¹⁴ Quantitatively, the bacteraemia is greater if the patient has periodontal disease.¹⁵ The greatest amount of bacteraemia occurs following extraction of erupted, periodontally involved teeth. There is less bacteraemia from unerupted teeth or from removal of asymptomatic mandibular fracture fixation plates.¹⁶

Generally, the bacteraemia can be demonstrated within one minute of the manipulation and is usually greatest five minutes following the procedure. Bacteria usually cannot be demonstrated in the bloodstream at 30 minutes after manipulation. Most studies show aerobic bacteria in the bloodstream, the most common types being viridans group *streptococci* although most of the common oral commensals can be demonstrated. If a thorough and an extensive culture for anaerobic bacteria is performed then anaerobic bacteria will be demonstrated. These are dominated by Actinomyces, campylobacter bacteria and lactobacillus species. Commonly, with careful culture, the anaerobic bacteria will form up to 85 per cent of all the bacteria in the bloodstream.¹⁷

General dental treatment such as local anaesthetic injection, fillings, impressions and dentures do not cause significant bacteraemia above those which occur in normal chewing or toothbrushing. ¹⁴ The most significant bacteraemias are produced by extraction of standing teeth, particularly if there is associated periodontal disease. Similar levels of bacteraemia are generated by deep periodontal scaling in the treatment of periodontal disease. ¹⁸

There is a group of intermediate dental procedures e.g., deep matrix band placement with interdental wedging, rubber dam clamps and endodontic manipulation, beyond the apex where there are significant periapical areas.¹⁹ In general the longer the dental manipulations continue the greater the bacteraemia (Table 1).

It needs to be understood that the oral cavity is not the only site of bacteraemia. Skin, bladder and gut all have resident flora and all have been demonstrated to result in transient bacteraemia. A considerable bacteraemia will result from colonoscopy for patients with inflammatory bowel disease such as diverticulitis.⁹

Under what circumstances do oral bacteria settle in distant parts of the body?

Although there is lengthy anecdotal history that a dental disease and treatment will result in bacterial colonization at different sites of the body, there is little clinical or experimental evidence to scientifically show that this is true.

Bacteria within the bloodstream pass into areas of acute inflammation as there is increased vascularity. Dependent on the nature of inflammatory reaction this may increase or decrease the destruction of bacteria. With the specific types of inflammatory response relating

Table 1. The relative incidence of bacteremia, distant bacterial colonization and adverse response to antibiotics

Event – Dental treatment	Risk of occurrence	
Bacteraemia during chewing	<70%	But small numbers and transient
Bacteraemia during non-surgical dentistry	<70%	But small number and transient
Bacteraemia during dental extraction in patient with periodontal disease	100%	Large number of bacteria both aerobic and anaerobes
Bacteraemia during deep periodontal scaling	100%	Large number of bacteria both aerobic and anaerobes
• Distant colonization from oral bacteria	0%	Not recorded in healthy individuals
Brain abscess	Occasional	Individual reports
• Infected joint prosthesis	0.03 to 0.07%	More likely in immunocompromised patient
Allergic to antibiotics		Usually known
Urticaria Anaphylaxis	3-5% 1 in 2500 to 5000	Allergic history
 Death from anaphylaxis 	1 in 25 000 to 50 000	

to rheumatoid arthritis or from a reaction to wear particles it would appear that they may make a failing implant more prone to infection. In areas of chronic inflammation, where there are concurrent areas of inflammation and necrotic tissue, there is a possibility of colonization into the less vascular areas of necrosis and thus a greater chance of bacterial colonization.

In this context it should be noted that in bacterial endocarditis where bacteria from the bloodstream colonize damaged heart valves, the key issue is abnormal blood flow with turbulence about the valves. This allows the bacteria to settle and attach to the valves. Heart valves are relatively avascular and thus the bacteria have the opportunity to colonize in the absence of an inflammatory response. Heart valves may also be damaged by bacteria in an immunological response. The classic example is in rheumatic fever where *streptococci* in the oropharynx provoke an immunologic response with secondary damage to the heart valves as there are similarities in structure between the bacterial walls and the heart valve. This, however, is not a bacterial infection.

The probable basis for most foci of infection type disease is where the presence of foci of bacteria relate to general systemic unhealthiness. Thus once the focus of infection is removed and as the patients recover, they feel better. Again this is not through distant bacterial colonization (Table 1).

Cerebral abscess is an example of where oral bacteria may lodge at distant sites.²¹ There are a few cases in which the organisms are demonstrably oral bacteria from the patient's mouth. The only reasonable portal of entry would be via the bloodstream. Usually it is considered there has been some form of haemodynamic variation in the brain or some form of prior brain injury to act as the site for the bacterial colonization.

Can orthopaedic implants become infected by blood-borne bacteria?

There is extensive soundly-based scientific literature on this. 1,6,8-10 It is important that all papers which set out to document joint infections have meticulous methodology as it is easy for the source of the infection to be based on anecdote. Ideally, to confirm that an

implant has been infected from an oral treatment, one requires a coincidence history and an accurate and simultaneous typing of the oral flora bacteraemia and joint organisms.¹ These steps have not usually been taken in most investigations in the literature and some papers are based solely on history.

Infection following a joint replacement is a devastating complication. 8.9 All require intensive and prolonged treatment. In one study of a consecutive series of infected hip replacements, 58 per cent were successfully re-implanted with a further artificial joint after a resolution of the infection, 34 per cent had implant removal but no replacement but local tissue flaps; 4 per cent had leg amputation at the level of the hip and 5 per cent of the patients died.9

Infections in joint replacements are divided into early or late occurring.^{8,9} Early infections, that is within the first three months following implantation, primarily relate to infection introduced at the time of the operation, either sourced from the patient or the surgical staff. The incidence of this is low and of the order of 0.39 per cent.⁹

Later infections, more than three months after primary implantation, are usually secondary to bacteraemia. The incidence is low and of the order of 0.97 per cent.²² The prevalent bacteria are Staphylococcus aureus (35 per cent) and Staphylococcus epidermidis (15 per cent). These are of skin origin. Some or most of these may even have been introduced at the time of surgery but have a delayed presentation. Group A streptococci, which are mainly of oropharyngeal origin, occurred in about 8 per cent of cases. Escherichia coli, which is the classic alimentary tract bacteria, were involved in about 4 per cent of cases. Thus bacteraemic-related joint infections may occur but generally at a low incidence. Skin organisms are the predominant group. The risk of oral-related infections is very low with figures in the range of 0.04-0.07 per cent^{23,24} (Table 1).

There are a number of publications on the risk of joint infections. Laporte *et al.* reviewed 2973 cases of joint replacements and found that 52 had late infections.² They were of the opinion that three of these were 'strongly associated' with a dental procedure. Two of

these three patients had additional risk factors, namely, diabetes and rheumatoid arthritis. None of the three patients had received prophylactic antibiotics and all of the dental procedures had lasted more than 45 minutes.

Waldman, in an extensive review of 3490 patients with knee arthroplasties, found 62 cases of late infection of which only seven had a dental association but over half of these had systemic risk factors.²⁵ Little, in a prospective study of 1000 patients, had three patients develop late joint infections. None of these were related to dental treatment.

Maderazo, in a review of 100 late joint infections, found that 34 of the patients had invasive health procedures performed coincident with the infection.²⁴ Five of these patients had dental treatment and all received prophylaxis. Ainscow and Denham followed 1000 patients with 1112 total joint replacements for up to six years. Of these, 224 had received dental treatment. However, not all the patients were administered prophylactic antibiotics and none had documented joint infection.²⁶ Jacobson investigated 2693 patients with whom 30 developed late joint infections. Of these, nine of the 30 were type II diabetics or on long-term immunosuppression.²⁷

Besides dental treatment there is also the matter of untreated dental disease. Ching *et al.* described four cases with late infections with *Streptococcus viridans* in patients who had poor oral health.²⁸ A further four cases with haematogenous *Streptococcus sanguis* joint infection without dental treatment but oral sepsis demonstrated the need for good oral hygiene.²⁹

Can oral bacteria be prevented from entering the bloodstream or from infecting orthopaedic implants by antibiotic treatment?

This question has been demonstrated in clinical studies where blood cultures are performed following dental treatment with appropriate prophylactic antibiotic coverage. 30-32 The incidence and magnitude of bacteraemia after an extraction does not decrease. However, the antibiotics inhibit bacterial growth at the localized site of bacterial colonization but there is no certainty that antibiotics will prevent the infection as evidenced in the Maderazo study where all five patients with apparently oral-related bacteria as the source of the infection of joint replacements received prophylactic antibiotic cover.24 Conversely, dental treatment with or without prophylactic antibiotic therapy resulted in 224 out of 1000 consecutive joint replacement patients not having joint infection. However, three of the remaining 776 patients who did not have dental treatment or receive prophylactic antibiotics had late joint infections of orally-related bacteria.²³ The authors did not specify what the patients' oral health state was at the time of the infection.

What are the risks of antibiotic therapy?

Antibiotics are not harmless medications and all are associated with side effects and adverse reactions. For the penicillin group the risk of an urticaria type reaction is between 3-5 per cent.³³ Though this is not life-threatening it is a temporary but significantly disabling condition. Anaphylaxis is a life-threatening condition and without immediate treatment may result in death. The risk of anaphylaxis to penicillin type antibiotics is quoted at 1 in 2500 to 5000.³⁴ The risk of a fatal anaphylaxis is 10 per cent of all cases of anaphylaxis. There are also the associated risks of increased bacterial resistance to the commonly prescribed antibiotics and the development of multiresistant strains.³⁵

Risk benefit analysis

Although bacteraemia is likely to occur following dental procedures the bacteria are transient and the incidence of joint infections related to the haematogenous route is low. In addition, the literature does not support prophylactic antibiotic therapy for dental treatment and it is inappropriate to expose patients to the adverse effects of antibiotics.

There is a small, recent and thorough scientific literature which supports this risk benefit analysis. Seymour calculated that if 100 000 patients had a joint replacement then 30 would have a late infection about the prosthesis which would require treatment. If all were given penicillin antibiotics then there would be 40 cases of anaphylaxis and four deaths.³⁶

Jacobson calculated that the risk of death from penicillin outweighs the benefit of prescribing it.²⁷ The adverse effects are more common and harmful than the occasional case of joint infection that it may prevent. Besides anaphylaxis and other allergic reaction he also highlighted the problems of gastro-intestinal disturbance and the development of resistant strains.

Analysis of health costs leads to a similar conclusion. It is cheaper to treat the occasional case of joint infection than to fund antibiotics and the cost of adverse sequelae. Jacobson calculated that, on 1990 costs, the cost of preventing one case of joint infection by widespread antibiotic prescription was US\$480 000.²⁷

However, there is a small group of patients with current infection who require prompt treatment with appropriate antibiotics. There is a greater risk of joint replacement infection when there is untreated oral sepsis, or dental procedures involving extraction, or deep periodontal curettage for patients with significant periodontal inflammation, for artificial joints with pre-existing local inflammation from either initial placement or from prosthetic failure. When these factors are present the benefits of antibiotic therapy exceed the risks.

DISCUSSION

This review shows that the traditional management practice of providing antibiotic cover for all dental treatments for patients with artificial joints is not necessarily justified and, indeed, could be potentially

dangerous. Altering traditional beliefs and health conventions, however, is never easy. The current classic example is the misuse of antibiotics in medical and dental practice.³⁷ All medical practitioners know that an upper respiratory tract infection is viral but over 50 per cent will prescribe antibiotics. Most dentists know that toothache in the absence of localizing clinical and radiographic signs is not bacterial but many will still prescribe antibiotics 'just in case'. Many medical and dental practitioners will agree that in such circumstances antibiotics are inappropriate but prescribed anyway as the patient 'demanded antibiotics'. Intensive public and health professional education programmes are now just showing evidence of working. Some patients understand that antibiotics are not the be all and end all and there has been a plateauing of the antibiotic prescription rate. This, however, has taken much time and effort and there is still a long way to go.37

There are a number of antibiotic prophylaxis guidelines which have been produced by national bodies. In July 2003, the American Dental Association published an advisory statement in association with the American Academy of Orthopaedic Surgeons 'Antibiotic prophylaxis is not routinely indicated for most dental patients with total joint prosthesis but it may be advisable to consider pre-medication in a small number of patients who may be at potential risk of experiencing haematogenous joint infections'. This is a major change in attitude by the Americans. The article contained a table which indicated risk groups. These related mainly to patients who were immunocompromised.

The British National Formulary has a similar review which looks at the rationale behind the guidelines and examines the issues from a risk management point of view.³⁹ It emphasizes that the risk of antibiotic prophylaxis outweighs the risk of joint infection. It also emphasizes the need for communication between orthopaedic surgeons and dentists. It confirms that within broad guidelines all patient situations are individual and this requires individual communication, co-operation and treatment plans.

The Australian guidelines for the use of drugs in dentistry state that there is no evidence of the infection of implants associated with dental treatment.⁴⁰ It then goes on to state that if patients are immunosuppressed, if extensive surgery is performed or if established infection is present, antibiotic prophylaxis should be provided.⁴¹ This fairly strong statement has been widely interpreted by many dental practitioners to provide antibiotic cover for most patients with artificial joints.

The corresponding author's institution guidelines for antibiotic prophylaxis, across the Royal Adelaide Hospital campus, were formulated in 1999 and revised in 2003.⁴² This was under the leadership of the Antibiotic Working Party, a sub-committee of the Drug Committee and it involved extensive consultation. This guideline has to a degree regulated what had been a

Table 2. Recommendation for dental treatment of patient with artificial joint replacement

Prior to placement of the first artificial joint

Referral to a dental practitioner for comprehensive dental examinations including radiographs.

Appropriate treatments as indicated to make the patient orally fit. Dentist if requested give a written opinion that the patient is orally fit with no evidence of oral infection.

Arrangements made for regular dental review.

Dental problem in the first 3 months following artificial joint placement

Infection with abscess formation: Urgent and aggressive treatment of the abscess. Remove the cause (exodontic or endodontic) under antibiotic prophylaxis.

Pain: Provide emergency dental treatment for pain. Antibiotics are indicated if a high- or medium-risk dental procedure performed. Noninfective dental problem without pain: Defer nonemergency dental treatment until 3 to 6 months after prosthesis replacement. Dental treatment after 3 months in a patient with a normally

functioning artificial joint Routine dental treatment including extraction. No antibiotic prophylaxis required.

No antibiotic prophylaxis required.

Regular dental review desirable.

Dental treatment for patients with significant risk factors for artificial joint infection

Immunocompromised patients include:

- those with insulin-dependent diabetes
- those taking immunosuppressive treatment for organ transplants or malignancy
- · those with systemic rheumatoid arthritis
- those taking systemic steroids (e.g., patients with severe asthma, dermatological problems)

Consultation with the patient's treating physician is recommended. *Failing, particularly chronically inflamed, artificial joints:* Consultation with the patient's treating orthopaedic surgeon is recommended.

Defer non-essential dental treatment until orthopaedic problem has resolved.

Previous history of infected artificial joints:

Routine non-surgical dental treatment – no prophylaxis indicated. Antibiotic prophylaxis recommended for:

- all extractions
- deep periodontal scaling

Regular dental reviews mandatory.

Established infection by oral organisms on an artificial joint Urgent referral to dentist to determine and eliminate any oral cause. Aggressive treatment by removal of the cause, extraction or endodontic under antibiotic prophylaxis.

highly individualistic approach to common problems. For patients with joint replacements, they agree that antibiotic prophylaxis was only indicated for high-risk dental procedures in immunocompromised patients with joint problems.

A guideline based on this review is presented in Table 2. Logically, the first step should be that all patients undergoing joint replacement should be dentally fit. This should be determined by a dentist after full oral examination and radiographs. The common situation of the orthopaedic surgeon asking the patients if their teeth are 'OK' is not enough. Interestingly, the corresponding author in 35 years of public and private practice cannot recall ever having been asked to check a patient's mouth for fitness or otherwise prior to joint replacement. Such referral, however, has been made a few times for patients presenting with an established joint infection. Cardiology and organ transplant candidates in South Australia are routinely referred for an oral health review.

Table 3. Recommended antibiotic regimens

- 1. Dental clinic LA extractions or deep curettage Amoxycillin 2-3g orally 1 hour prior to procedure
- Theatre procedures
 Amoxycillin 1g I/V at induction
 Followed by 500mg amoxicillin I/V or orally 6 hours later.
- Penicillin hypersensitivity, long term penicillin, recent penicillin/other B-lactam.
 Clindamycin 600mg 1 hour prior to procedure or Vancomycin 1g I/V 1 hour to finish 2 hours or Lincomycin 600mg just prior to the procedure
- 4. High risk case (i.e., Gross oral sepsis/severely immunocompromised/previous joint infection.) Gentamicin 2mg/kg I/V just before procedure (can be administered 3mg/kg provided there is no concomitant renal disease) PLUS Amoxycillin 1g I/V just before procedure followed by

PLUS Amoxycillin 1g I/V just before procedure followed by 500mg I/V or orally 6 hours later.

If hypersensitive to penicillin replace amoxicillin with Vancomycin 1g I/V over 1 hour to finish just before procedure.

Dental treatment in the pre-implantation phase should be aggressive to eliminate current foci of infection. If the condition cannot be rapidly resolved by restorative, endodontic or periodontal treatment the involved teeth should be extracted. Antibiotic prophylaxis would not usually be required for such pre-implantation treatment.

In the initial phase following placement of joint prosthesis dental treatment would not normally be required if the patients have been made dentally fit prior to the procedure. The patients in this first threemonth phase after receiving a prosthetic joint are usually in some orthopaedic discomfort and usually¹⁵ not sufficiently mobile for routine dental treatment. If a dental infection arises it should be treated aggressively by endodontics or extraction with appropriate therapeutic antibiotics. Once a joint prosthesis is stabilized and functioning well, routine dental treatment including extractions in the absence of gross periodontal disease do not require antibiotic prophylaxis. However, antibiotic prophylaxis should be prescribed for patients who have already had an episode of replacement for an infected prosthesis as this may indicate that they are at increased risk of developing an infection. Standard prophylactic regimens are presented in Table 3.

Finally, there remains the currently not uncommon scenario where patients demand that they have antibiotic treatment for all dental treatment because their orthopaedic surgeon has advised them that they should. In that case one does need to carefully evaluate the precise nature of the dental problem and the treatment needs. One then needs to communicate with the patient's orthopaedic surgeon so that the actual risk benefit situation can be determined. If there is no indication for antibiotic prophylaxis in accordance with these guidelines but the orthopaedic surgeon is insistent that antibiotics are given, then an informed consent decision needs to be made. If antibiotics are requested, it needs to be recorded that the orthopaedic

surgeon requested prophylaxis be given. Hence the orthopaedic surgeon would bear the responsibility for any adverse outcome related to the administration of the antibiotic therapy.

REFERENCES

- 1. Sandhu SS, Lowry JC, Reuben SF, Morton ME. Who decides on the need for antibiotic prophylaxis in patients with major arthroplasties requiring dental treatment: Is it a joint responsibility? Ann R Coll Surg Engl 1997;79:143-147.
- Seymour RA, Whitworth JM, Martin M. Antibiotic prophylaxis for patient with joint prostheses – still a dilemma for dental practitioners. Br Dent J 2003;194:649-653.
- 3. LaPorte DM, Waldman BJ, Mont MA, Hungerford DS. Infections associated with dental procedures in total hip arthroplasty. J Bone Joint Surg Br 1999;81:56-59.
- 4. Chronic oral sepsis and its relation to systemic disease. Focal infection. In: Stones HH, ed. Oral and dental diseases. 4th edn. Edinburgh & London: E&S Livingstone, 1962:679-690.
- Cohen RG, Forrest CJ, Benjamin JB. Safety and efficacy of bilateral total knee arthroplasty. J Arthroplasty 1997;12:497-502
- American Academy of Orthopaedic Surgeons: National Centre of Health Statistics Rosemont. Illinois. American Academy of Orthopaedic Surgeons 14. 1999.
- 7. Australian Orthopaedic Association National Joint Replacement Registry Annual Report. Adelaide: AOA, 2004. ISSN 1455-366. http://www.dmac.adelaide.edu.au/aoanjrr/aoanjrr.jsp. Accessed November 2005.
- 8. McPherson EJ, Woodson C, Holtom P, Roidis N, Schufelt C, Patzakis M. Periprosthetic total hip infection. Clin Orthop Relat Res 2002;403:8-15.
- 9. Peersman G, Laskin R, Davis J, Peterson M. Infection in total knee replacements: a retrospective view of 6489 total knee replacements. Clin Orthop Relat Res 2001;392:15-23.
- Rhinelander FW. Physiologic responses of bone to implants. In: Biocompatibility of orthopaedic implants. Boca Raton: CRC Press, 1982:51-74.
- Cameron HU, Hunter GA. Failure in total knee arthroplasty: mechanisms, revisions and results. Clin Orthop Relat Res 1982;170:141-146.
- 12. Heimdahl A, Hall G, Hederberg M, et al. Detection and quantitation by lysis filtration at bacteremia after different oral surgical procedures. J Clin Microbiol 1990;28:2205-2209.
- 13. Okabe K, Nagagawa K, Yamomoto E. Factors affecting the occurrence of bacteremia associated with tooth extraction. Int J Oral Maxillofac Surg 1995;24:239-242.
- 14. Guneroth WG. How important are dental procedures as a cause of infective endocarditis. Am J Cardiol 1984;54:797-801.
- 15. Malinverni R, Overholser CD, Bille J, Glauser MP. Antibiotic prophylaxis of experimental endocarditis after dental extractions. Circulation 1988;77:182-187.
- Rajasuo A, Nyfors S, Kanervo A, Jousimies-Somer H, Lindqvist C, Suuronen R. Bacteremia after plate removal and tooth extraction. Int J Oral Maxillofac Surg 2004;33:356-360.
- Otten JE, Pelz K, Christmann G. Anaerobic bacteremia following tooth extraction and removal of osteosynthesis plates. J Oral Maxillofac Surg 1987;45:47.
- 18. Bender IB, Naidorf IJ, Garvey GJ. Bacterial endocarditis: a consideration for physician and dentist. J Am Dent Assoc 1984;109:415-420.
- 19. Debelian GJ, Olsen I, Tronstad L. Anaerobic bacteremia and fungemia in patients undergoing endodontic therapy: an overview. Ann Periodontal 1998;3:281-287.
- 20. Singh J, Straznicky I, Avent M, Goss AN. Antibiotic prophylaxis for endocarditis: time to reconsider. Aust Dent J 2005;50 Suppl 2:S60-S68.

- Molavi A, DiNuble MJ. Brain abcess. In: Harris AA, ed. Microbial Disease: Handbook of Clinical Neurology. New York: Elsevier Science, 1998:143.
- 22. Saleh KJ, Macaulay A, Radosevich DM, et al. The Knee Society Index of Severity for failed total knee arthroplasty: Development and validation. Clin Orthop Relat Res 2001;392:153-165.
- 23. Little JW. Dental treatment in patients with joint replacements. Oral Surgery 1983;55:20-23.
- Maderazo EG, Judson S, Pasternak H. Late infections of total joint prostheses. A review and recommendations for prevention. Clin Orthop Relat Res 1988;229:131-142.
- Waldman BJ, Mont MA, Hungerford DS. Total knee arthroplasty infections associated with dental procedures. Clin Orthop Relat Res 1997;343:164-172.
- Ainscow DA, Denham RA. The risk of haematogenous infection on total joint replacements. J Bone Joint Surg Br 1984;66:580-582.
- Jacobson JJ, Schweitzer S, DePorter DJ, Lee JJ. Antibiotic prophylaxis for dental patients with joint prostheses: a decision analysis. Int J Technol Assess Health Care 1990;6:569-587.
- 28. Ching DW, Gould IM, Rennie JA, Gibson PH. Prevention of late haematogenous infection in major prosthetic joints. J Antimicrob Chemother 1989;23:676-680.
- Bartzokas CA, Johnson R, Jane M, Martin MV, Pearce PK, Saw Y. Relation between mouth and haematogenous infections in total joint replacement. BMJ 1994;309:506-508.
- Hall G, Hedstrom SA, Heimdahl A, Nord CE. Prophylactic administration of penicillins for endocarditis does not reduce the incidence of postextraction bacteremia. Clin Infect Dis 1993;17:188-184.
- Hall G, Heimdahl A, Nord CE. Effects of prophylactic administration of cefaclor on transient bacteremia after dental extraction. Eur J Clin Microbiol Infect Dis 1996;15:646-649.
- Kaneko A, Sasaki J, Yamazaki J, Kobayashi I. Intravenous administration of vancomycin is ineffective against bacteremia following tooth extraction. Tokai J Exp Clin Med 1995;20:65-66.
- Idsoe O, Guthe T, Willcox RR, de Weck AL. Nature and extent of penicillin side-reactions, with particular reference to fatalities from anaphylactic shock. Bull World Health Organ 1968;38:159-188.

- 34. Parker CW. Allergic reactions in man. Pharmacol Rev 1982;34:85-104.
- Molavi A, Dinubile MJ, Brain abcess. Handbook of Clinical Neurology, Microbiological Diseases. New York: Elsevier Science, 1988:149
- Jacobson JJ, Schweitzer S, Kowalski CJ. Chemoprophylaxis of prosthetic joint patients during dental treatment: a decisionutility analysis. Oral Surg Oral Med Oral Pathol 1991;72:167-177.
- Jaunay T, Sambrook P, Goss A. Antibiotic prescribing practices by South Australian general dental practitioners. Aust Dent J 2000;45:179-186.
- 38. Position Statement. American Dental Association; American Academy of Orthopedic Surgeons. Antibiotic prophylaxis for dental patients with total joint replacements. J Am Dent Assoc 2003;134:895-899.
- British National Formulary. BNF 48 (September 2004). http://www.bnf.org. Accessed November 2005.
- 40. Woods R. A guide to the use of drugs in dentistry. 12th edn. Sydney: Australian Dental Association Inc, 1996.
- 41. Victorian Drug Usage Advisory Committee. Antibiotic Guidelines. 9th edn. Melbourne: Victorian Medical Postgraduate Foundation Therapeutics Committee, 1996:92-95.
- Prevention of endocarditis on infection of prosthetic implants or grafts. Royal Adelaide Hospital – Antibiotic prophylaxis guidelines. 1999. Revised 2003.

Address for correspondence/reprints:
Professor Alastair N Goss
Oral and Maxillofacial Surgery Unit
Faculty of Health Sciences
The University of Adelaide
Adelaide, South Australia 5005
Email: oral.surgery@adelaide.edu.au