INVITED ARTICLE

ANTIBIOTIC PROPHYLAXIS DURING DENTAL TREATMENT IN PATIENTS WITH PROSTHETIC JOINTS

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Since the early 1960s the replacement of large joints, including hip, knee, elbow and shoulder, has become commonplace. A survey by Hori et al (1978) revealed that some 120 000 total hip and knee replacements were undertaken each year in the United States, and it may be assumed that numbers have increased every year since. In New Zealand, over 4000 total hip replacements are performed annually (NZ Orthopaedic Association–personal communication 1990).

Many orthopaedic surgeons consider that antibiotic prophylaxis is necessary in patients with prosthetic joints before dental treatment which could produce a bacteraemia. A study conducted throughout the United States (Jaspers and Little 1985) revealed that 93% of the orthopaedic surgeons polled believed such prophylaxis was necessary to prevent late infection. Late deep infection is a serious complication sometimes leading to loss of the prosthetic joint and attended by significant morbidity and mortality (Ahlberg, Carlsson and Lindberg 1978; Surin, Sundholm and Bäckman 1983). However, opposing views have also been expressed, mainly by authorities in the dental field. These authors believe that the transient bacteraemias usually associated with dental treatment are unlikely to produce joint infection (Peterson 1980; McGowan and Hendrey 1985; Jacobson et al 1986).

Late joint infections, after three months, are considered to arise either by haematogenous spread or secondary to intra-operative contamination (Ahlberg et al 1978; Andrews et al 1981). Prosthetic joints appear particularly susceptible to infection, probably as a result of a number of factors: venostasis and stagnation, the presence of necrotic bone caused by surgical reaming or the heat generated by methacrylate, and possibly the inhibition of macrophage function by methacrylate (Gristina and Kolk 1983). The incidence of both early and late prosthetic joint infection has been reduced during the last 20 years, by improved operating theatre technique and the use of prophylactic antibiotics. Charnley (1972) demonstrated a reduction in the incidence of deep infection after total hip replacement, from 7% to 0.5% with laminar air flow and use of impermeable operating gowns. Subsequently, Lidwell et al (1984) quoted reports of a reduction of deep infection following total hip or knee replacement from 3.4% to 0.19%, with a combination of ultraclean air, body exhaust suits and prophylactic antibiotics. Further refinement of these operative techniques, surveyed as part of a major multi-centre study of hip and knee replacement, has demonstrated that the overall incidence of joint infection can be reduced to less than 0.1% (Lidwell et al 1987).

Other factors which have been implicated in late deep joint infections are prolonged operating time, repeat procedures, positive culture at operation, and the occurrence of 'superficial' wound infections which subsequently healed (Fitzgerald et al 1977; Surin et al 1983). Studies such as these have lent strong support to the view that most deep infections, including late infections, arise from bacteria implanted at the time of surgery (Charnley 1972; Surin et al 1983). Nevertheless, numerous case reports have appeared detailing examples of apparent haematogenous infection of prosthetic joints, many with convincing bacteriological evidence (D'Ambrosia, Shoji and Heater 1976; Downes 1977; Ahlberg et al 1978; Lattimer et al 1979; Stinchfield et al 1980; Wigren, Karlstrom and Kaufer 1980; Lindqvist and Slätis 1985; Strazzeri and Anzel 1986; Maniloff et al 1987).
The problem has been investigated in an animal model by Blomgren and Lindgren (1981), who used human finger-joint prostheses to replace the knees of rabbits, followed six to eight weeks later by intravenous inoculation of *Staphylococcus aureus*. About one-third of joints became infected. However, the bacterial inoculum used was very large (1.15 × 10⁸ bacteria) and more than half of the animals died either from endotoxin shock or septicaemia. It is therefore difficult to extrapolate these results to the situation in patients where bacteraemia is likely to involve significantly fewer organisms. Even so, this study is cited as the main justification for antibiotic prophylaxis in patients undergoing dental procedures likely to produce transient bacteraemia (Cioffi, Terezhalmy and Taybos 1988).

A review of published literature has identified 21 reported cases of late prosthetic joint infection in which it is suggested the infecting bacteria originated from the oral cavity as the result of dental treatment or infection (see Table I). To accept this association the organism cultured from the joint should be identical to that cultured from the oral cavity. The case would be further strengthened if the same organism was cultured from blood samples obtained concurrently; this is regarded as the minimal acceptable standard by some authors (Ahlberg et al 1978). Furthermore, it would seem reasonable that there be a relationship in time between the dental event, be this treatment or infection, and the initial onset of symptoms of prosthetic joint infection. It should not exceed, for example, three months.

When all these criteria are applied to published reports, not a single case seems to be acceptable, with the possible exception of one (case 7, Table I) in which the infecting organism was cultured from saliva, blood and the infected joint, and was also cultured from urine. Even if the criteria of positive blood culture and a reasonable time relationship are dropped, only one further instance (case 11) seems acceptable. More than half of our collection of cases involved organisms which are normally found on skin (*Staphylococcus aureus, Staphylococcus epidermidis,* and *Propionibacterium*), these also being the organisms most commonly involved in both early and late delayed infection (Charnley 1972; Carlsson, Lidgren and Lindberg 1977; Fitzgerald et al 1977; Andrews et al 1981; Canner et al 1984). Even with the extremely low rates of late infection (0.07%) reported in the Medical Research Council multi-centre series (Lidwell et al 1987) the majority of infecting organisms have been these skin commensals; most of the remainder are common in the intestinal or urinary tracts.

In the reported cases of late infection due to streptococci thought to have originated from the oral cavity, the organisms involved have usually been those with a wider distribution than the mouth. For example, group A β-haemolytic streptococci are predominant in the nasopharynx and throat, group B β-haemolytic streptococci are widespread on the body, and group C streptococci are mainly found in the nasopharynx despite being isolated sometimes from the saliva and gingivae. *Streptococcus viridans* would, however, be likely to have originated from the oral cavity, and it is of interest that, in the only three reported cases where this organism produced late infection, the patients concerned had severe periodontal disease. This is a setting in which bacteraemia would frequently occur, with *Streptococcus viridans* as a significant organism. It must be conceded that in such cases with late joint infection, there is a possible cause-and-effect relationship.

Late infection of prosthetic joints appears to be comparable with that of infective endocarditis developing in association with cardiac valvular homografts or prosthetic valves. However, the role of haematogenous bacterial seeding in endocarditis has been established, both in patients and in experimental animals (Kaye 1985; Sullam, Drake and Sande 1985). In cases of endocarditis caused by *Streptococcus viridans* originating from the oral cavity, the pathogenicity of this normally innocuous organism results from cell-surface dextran production which confers an unusual ability to adhere to platelets (Baddour et al 1989). There is a widespread consensus for antibiotic prophylaxis against infective endocarditis when certain dental procedures are undertaken (Kaye 1986).

The issue is further complicated by considering the circumstances under which delayed prosthetic joint infection, caused by organisms implanted at the time of surgery, is likely to manifest. This will usually occur during a period when immunological responses are depressed, perhaps in association with an unrelated, sometimes viral infection, allowing activation of subclinical bacterial infection. This is the same background to the common conversion of a chronic dental infection, as might surround a long-dead tooth, into an active clinical infection. It is thus easy to ascribe a cause-and-effect relationship to the simultaneous manifestation of a dental infection and a late prosthetic joint infection.

Prophylaxis against the possibility of prosthetic joint infection from a distant focus requires the choice of an antibiotic which will cover the organisms likely to originate from that site. The organisms of the bacteraemia which may result from dental procedures or infections have been studied with reference to infective endocarditis, and it has been established that penicillins provide the best cover (Kaye 1986). However, in most joint infections, bacteria are found which are not susceptible to penicillins. Therefore the use of penicillins would be logical only if there was a strong likelihood of bacteraemia of oral origin. Cephalosporins are the most popular antibiotics used by orthopaedic surgeons for prophylaxis during joint replacement operations (Gristina and Kolkin 1983; Maderazo, Judson and Pasternak 1988), and are often recommended for prophylaxis against oral organisms (Cioffi et al 1988; Maderazo et al 1988; Shay and Lloyd 1988). However, these antibiotics have been shown
to be ineffective against a haematogenously seeded *Streptococcus viridans* endocarditis in an animal model (Durack and Petersdorf 1973).

If it were accepted that a very small number of late prosthetic joint infections could result from haematogenously seeded from the mouth, then a risk-benefit analysis of antibiotic prophylaxis could be made. Jacobson et al (1988) have compared the documented incidence of allergic reactions to both penicillin and the cephalosporins with the likelihood of late prosthetic joint infections, based upon an assumed incidence of 29 to 68 cases of joint infection per 10,000 dental visits. This would be a high incidence of infection in the light of the evidence so far discussed, but the risk of allergic reactions, including severe or fatal ones, is much higher for both groups of antibiotics.

The role of antibiotic prophylaxis has also been investigated prospectively by Ainscow and Denham (1984) who followed 1000 patients with 1112 total joint replacements for an average of six years. The patients were not advised to take antibiotics prophylactically to cover subsequent dental or surgical procedures; during follow-up only three patients developed joint infections, all of which were associated with documented skin infections.

Although the weight of evidence suggests that late prosthetic joint infection is an exceedingly unlikely consequence of bacteraemia originating from the mouth, it is recognised first, that certain groups of patients are more at risk, and secondly, that massive or continuing bacteraemia may compound this risk. Patients in high-risk groups are those with rheumatoid arthritis (D’Ambrosia et al 1976; Stinchfield et al 1980; Ainscow and Denham 1984), diabetes mellitus (Jacobson et al 1986), on steroids (Wilson, Salvati and Blumenfeld 1975), and those with re-operated joints, particularly hips (Fitzgerald et al 1977; Canner et al 1984). To these groups it would seem sensible to add patients who are immunosuppressed. It could thus reasonably be argued that, in such patients, antibiotic prophylaxis to cover dental procedures which are known to cause significant bacteraemia is justifiable. Large and continuing episodes of bacteraemia from the
oral cavity occur mainly in patients with advanced periodontal disease or generalised dental sepsis. These patients should be identified prior to joint surgery and made free of dental disease.

The present state of knowledge may therefore be summarised as follows:
1) There is no convincing published evidence to support a general policy of antibiotic prophylaxis to cover dental procedures in patients with prosthetic joints.
2) Patients with rheumatoid arthritis, re-operated hips, diabetes mellitus, and those on steroids or immuno-suppression, constitute a group at higher risk. In these patients antibiotic prophylaxis may be justified to cover procedures likely to produce a significant bacteraemia.

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REFERENCES


