Prevention of infective endocarditis

CELIA OAKLEY, WALTER SOMERVILLE

Royal Postgraduate Medical School, Hammersmith Hospital; and The Middlesex Hospital, London

The undiminished incidence of streptococcal endocarditis in this country is a testimony to the failure of prophylaxis.

Ninety per cent or more of medical infective endocarditis in the UK is still caused by the viridans group of streptococci¹ though in the United States the incidence has fallen to 50 per cent in some series probably because of a rise in the relative incidence of cases caused by intravenous drug abuse. Unfortunately, infective endocarditis is not a notifiable disease so the case load is not precisely known and is largely based on personal and published hospital series. An inspired guess suggests that there may be 1500 recognised cases a year. This means only five or six cases a year shared between the medical firms in an average sized district general hospital and perhaps 30 to 50 in a major cardiac unit. On the one hand are the general physician and his registrar who recognise infective endocarditis as a rare disease; on the other is the specialist cardiological team who mainly receive late diagnosed, specially referred, and post-cardiac surgery cases and may see a bacteriologically skewed spectrum with a high mortality.

It should be possible to reason why prophylaxis has failed: (1) Perhaps it is not being given. (2) Perhaps it is not being given to the right people. (3) Perhaps it does not work. (4) Perhaps it is irrelevant.

In 1977 the American Heart Association published its revised recommendations for antibiotic prophylaxis of procedures known to cause bacteraemia.² For dental operations the committee advised intramuscular penicillin followed by oral phenoxymethyl penicillin. With even less practicality they advised intravenous vancomycin followed by oral erythromycin for penicillin sensitive subjects. Only two years earlier Durack, a member of this committee, had carried out a survey in Oxfordshire which showed that dental prophylaxis was administered in the then approved way in only

8 per cent of cases.³ The reason was easy to see. Most teeth are extracted under local anaesthetic outside hospital. Dentists do not prescribe intramuscular penicillin nor do their nurses give intramuscular injections. Most patients prefer to avoid injections anyway and are only too happy to accept oral prophylaxis, but even the most tenacious patient would find it almost impossible to achieve an intramuscular injection half an hour before an extraction in a dental surgery. Usually, therefore, tablets are prescribed, started too early, and carried on too long. Oral prophylaxis started too soon changes the oral flora and may cause infections by penicillin resistant organisms.⁴

We also know that selective prophylaxis misses many of those who most need it. Weinstein emphasised that 40 to 60 per cent of infective endocarditis occurred in patients with no known heart disease.⁵ Such patients are not in line for prophylaxis, effective or not. Congenitally abnormal or "bicuspid" aortic valves are present in 1 to 2 per cent of the male population.⁶

In parentheses one of us notes that more than 90 per cent of urgent valve replacements carried out in our hospital for neglected endocarditis have been in young men with "bicuspid" valves, the existence of which was unknown to them or to their doctors.⁷ Some of those at risk have floppy mitral valves or hypertrophic cardiomyopathy. Many elderly people have degenerative valve disease, "aortic sclerosis", or mitral regurgitation.

It is apparent that if optimal dental prophylaxis is ever going to be achieved out of hospital, it has to be oral and, we suggest, given by the dentist. Only the dentist knows how long the agony in the waiting room will be and can send his nurse to supervise swallowing of the dose at the appropriate time. What should the drug be? Amoxycillin is better absorbed than penicillin V, is independent of recent food intake, and stays in the blood stream longer after a single dose. Recent work has shown that a

single dose of 3 g amoxycillin effectively prevents bacteraemia after dental extraction^{8,9} and this regimen has advantages over four doses of oral penicillin V as suggested by Petersdorf.¹⁰

We suggest that a single dose of 3 g oral amoxycillin is at present the preferred method of dental prophylaxis and one which is likely to be implemented outside hospital, being well tolerated and accepted by patients. Patients with a history of penicillin allergy should be given a single dose of 2 g oral erythromycin instead. These drugs should be stocked and dispensed by the dentist. This, however, will require a change in dental practice. Patients having dental extractions in hospital under general anaesthetic should have intramuscular amoxycillin 1 g as a single dose half an hour before dental extraction under general anaesthetic. Before minor surgery involving the gastrointestinal or genitourinary tracts intramuscular ampicillin or amoxycillin 1 g + gentamicin 80 mg should be given.

The antibiotic protection to give to a patient who is already being treated for infective endocarditis and requires dental extraction is a knotty problem. Unless there is a root abscess it is preferable to defer dental treatment until after completion of the treatment for infective endocarditis and the mouth has been recolonised with streptococci.⁴ Otherwise there is a real risk of introducing *Candida* which is all too commonly present in the mouths of those receiving long-term antibiotics. It is least likely in patients receiving penicillin alone in whom it is probably safe to give intramuscular gentamicin 80 mg or intravenous vancomycin 1 g if dental treatment cannot wait.

It has been argued that dental prophylaxis may be largely irrelevant and edentulous subjects are not immune from streptococcal endocarditis.¹¹ The evidence linking dental extraction with infective endocarditis is tenuous but in one series as many as 40 per cent of patients with streptococcal endocarditis had had dental procedures within the preceding three months.¹¹ Dental extraction usually produces a brisk but transient streptococcal bacteraemia. Similar but presumably lesser bacteraemia may recur several times each day during chewing. Bacteraemias are common and may follow squeezing a boil or even defecation.¹² Which bacteraemias precede endocarditis? The development of streptococcal bacterial endocarditis probably needs heightened host susceptibility at the time and bacteraemia alone may not be enough unless perhaps it is of massive size. It is a common observation that some patients develop recurrent attacks of infective bacterial endocarditis despite every precaution while others with gross periodontal disease, no

prophylaxis, and susceptible valve disease never get infected at all.

Despite the foregoing, it is logical to argue that though low grade bacteraemia is more common, heavy bacteraemia is more dangerous. Normal hearts are also more common but detectably abnormal hearts are more at risk. If effective prophylaxis could be organised to cover predictable heavy bacteraemia in the most susceptible subjects there could be a pronounced fall in the incidence of infective endocarditis.

Because of the evident difficulty in identifying susceptible subjects, as shown by Weinstein's figures, then it would be logical to cover all predictable heavy bacteraemia. If all dental extractions are covered routinely, as seems desirable,¹³ it would be essential for dentists to dispense the antibiotics.

Some members of the American Heart Association working party on prevention of infective endocarditis have been soliciting cases of failed prophylaxis.¹⁴ With respect, it seems more important first to ensure that prophylaxis is given. It is a sobering thought that a rise in the standard of oral hygiene throughout the country would probably help more than any other measure to reduce the incidence of streptococcal infective endocarditis. If we can accomplish this and prophylaxis too then endocarditis should become a rare disease. And, even more important, if we could achieve earlier diagnosis and treatment it need not still be a fatal one.

References

- 1 Hayward GW. Infective endocarditis: a changing disease. *Br Med J* 1973; **ii**: 706-9 & 764-6.
- 2 American Heart Association. Committee report: prevention of bacterial endocarditis. *Circulation* 1977; **56**: 139A-43A.
- 3 Durack DT. Current practice in prevention of bacterial endocarditis. *Br Heart J* 1975; **37**: 478-81.
- 4 Garrod LP, Waterworth PM. The risks of dental extraction during penicillin treatment. *Br Heart J* 1962; **24**: 39-46.
- 5 Weinstein L. Infective endocarditis: past, present and future. *J R Coll Physicians Lond* 1971-1972; **6**: 161-74.
- 6 Roberts WC. The congenitally bicuspid aortic valve. *Am J Cardiol* 1970; **26**: 72-83.
- 7 Wise JR Jr, Bentall HH, Cleland WP, Goodwin JF, Hallidie-Smith KA, Oakley CM. Urgent aortic-valve replacement for acute aortic regurgitation in infective endocarditis. *Lancet* 1971; **ii**: 115-21.
- 8 Shanson DC, Ashford RFU, Singh J. High-dose oral amoxycillin for preventing endocarditis. *Br Med J* 1980; **280**: 446.
- 9 Perez G, Darrell JA, Oakley CM. To be published.
- 10 Petersdorf RG. Antimicrobial prophylaxis of bac-

- terial endocarditis. Prudent caution or bacterial overkill. *Am J Med* 1978; **65**: 220-3.
- 11 Croxson MS, Altmann MM, O'Brien KP. Dental status and recurrence of *Streptococcus viridans* endocarditis. *Lancet* 1971; **i**: 1205-7.
 - 12 Rogosa M, Hampp EG, Nevin TA, Wagner HN Jr, Driscoll EJ, Baer PN. Blood sampling and cultural studies in the detection of postoperative bacteremias. *J Am Dent Assoc* 1960; **60**: 172-80.
 - 13 Oakley CM. Prevention of infective endocarditis. *Thorax* 1979; **34**: 711-2.
 - 14 Bisno AL, Durack DT, Fraser DW, Kaplan EL, Oliveria MA. Failure of prophylaxis for bacterial endocarditis: American Heart Association registry (letter). *Br Heart J* 1980; **43**: 244.

Requests for reprints to Dr Celia M Oakley, Royal Postgraduate Medical School, Hammersmith Hospital, Du Cane Road, London W12 0HS.