The teeth and infective endocarditis

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SUMMARY During 1981 and 1982 544 cases of infective endocarditis were investigated retrospectively by means of a questionnaire. Only 13.7% had undergone any dental procedure within three months of the onset of the illness, and in 42.5% there was no known cardiac abnormality before the onset of the disease. Furthermore, the number of cases occurring annually was about the same as or more than it was before the introduction of penicillin. The mouth and nasopharynx were the most likely sources of the commonest organism, *Streptococcus viridans*, and it is suggested that it is not dental extractions themselves which are of importance but good dental hygiene. In most patients with infective endocarditis the portal of entry of the organism whatever its nature cannot be identified. If this is so antibiotics are being given to only a small proportion of those at risk, and this would explain why the number of cases is much the same as it was before the introduction of penicillin. Furthermore, the large proportion of patients with no known previous cardiac abnormality adds to the difficulty of providing effective prophylaxis.

The evidence suggests that antibiotic prophylaxis should still be given before dental procedures, and a schedule is appended. Much more importance should be given, however, to encouraging people to seek better routine dental care. We also believe that doctors and dentists should appreciate that the pattern of the disease has changed considerably in the past 50 years and that the information given here warrants a revised approach to the problem.

For more than 50 years doctors and dentists have believed that dental procedures carried out on patients with rheumatic or congenital heart disease are the most common cause of infective endocarditis. Sixty years ago Lewis and Grant¹ suggested that transient bacteraemia might cause infective endocarditis in those with abnormal heart valves, and in 1930 Rush-ton² described endocarditis after dental extraction. Five years later Okell and Elliott³ reported a high incidence of streptococcal bacteraemia after dental extraction. Burket and Burn⁴ added confirmation by painting gingival crevices with *Serratia marcescens* (then thought to be non-pathogenic but now known as a potential cause of infective endocarditis) before dental extraction and thereafter isolating the organism from the blood in 20% of 90 patients. Taran⁵ in 1944 reported four children with negative blood cultures before tooth extraction who died from streptococcal infective endocarditis after the procedure. Scaling,⁶ gingivectomy, dental irrigation,⁷ brushing the teeth,⁸ and even chewing⁸ may induce bacteraemia.

There is therefore abundant evidence to suggest

that dental procedures may cause bacteraemia and that this may progress to infective endocarditis. While proof is lacking and probably impossible to obtain, however, doctors and dentists have accepted this relation and have endeavoured to protect their patients.

The proportion of patients with infective endocarditis in whom a dental procedure or sepsis has been considered responsible has varied. Cates and Christie⁹ in their study of 442 cases over 30 years ago found that 23 (5%) had had a dental extraction within three months of the onset of illness and 72 (16%) had dental sepsis or caries. In the 1956-65 series from St Bartholomew's Hospital¹⁰ 34% of 93 patients had received or needed dental treatment, but in the next decade only 11 (18%) of 60 patients at this hospital gave a history of recent dental work or gum infection¹¹; in a later series from Birmingham¹² only four of 70 patients had had recent dental treatment. In 93 episodes of culture positive endocarditis at St Thomas's Hospital,¹³ however, 31 (33%) were thought to be of dental origin, and Oakley¹⁴ indicated that in as many as 40% of patients a history of a dental procedure during the previous three months could be

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elicited. By contrast, a possible dental cause was present in only 9% of a series from the University of Washington Hospitals.¹⁵ Pogrel and Welsby¹⁶ reported a recent dental extraction in only 5% of 83 cases, and Cherubin and Neu¹⁷ found a decreasing incidence of preceding dental extraction from 10.3% in 1938-47 to 6.3% in 1958-67 and commented on the uncertain relevance of the dental history.

These reports indicate that certain cases of infective endocarditis are causally related to previous dental procedures or dental sepsis. It is not known which patients are at risk, whether antibiotic prophylaxis is necessary before dental procedures, and, if so, which antibiotic is indicated and how it should be used. Current practice of antibiotic prophylaxis certainly appears unsatisfactory.^{14 18}

Two senior members of the British Cardiac Society suggested that it would be useful to investigate the part played by dental sepsis and dental procedures and the efficacy of antibiotic prophylaxis carried out at present. To this end the British Cardiac Society in association with the Medical Services Study Group of the Royal College of Physicians has studied patients in the British Isles with infective endocarditis during 1981 or 1982.

Patients and methods

Members of the British Cardiac Society promised support for the investigation and enlisted the help of colleagues who were not members of the society. The regional advisers of the Royal College of Physicians encouraged all physicians in their region to participate and the *British Heart Journal*, *British Medical Journal*, and *Lancet* publicised the project. Dr Spence Galbraith and Dr Susan Young of the Communicable Disease Surveillance Centre were helpful in asking microbiologists to notify the investigators of any patients coming to their notice.

A proforma was devised which was kept as short as possible so as to encourage completion. It required name, age, and sex of the patient; hospital in which treated and name of consultant; results of blood cul-

ture; details of any dental or other procedure during the three months before the onset of illness; and the interval between the procedure and the first symptoms of infective endocarditis. In addition, details of the pre-existing cardiac abnormality, if any, and information as to whether it was previously known to the doctor, dentist, or patient were requested.

Results

A total of 544 proformas were received concerning 541 patients, three of whom had two attacks of infective endocarditis within the two years. Patients' ages ranged from 2 to 87 years (mean 51.6 years) and there was a greater proportion of males (ratio 2:1).

A dental procedure had been carried out within three months of the onset of the illness in only 74 (13.7%) of the 544 cases notified, 67 without and seven with antibiotic prophylaxis. Of the 67 who were not given antibiotic prophylaxis 37 had known pre-existing cardiac abnormalities. Fifty-three of the 74 (71.6%) were due to *S viridans*, the apparent incubation period being one month or less in 20 (37.7%) of these 53. In addition, 48 of those who had not received dental treatment had dental sepsis, and in 33 of the 48 the organism responsible was *S viridans*. In another 176 patients without overt dental sepsis and who had not undergone any dental procedure within three months the causal organism was also *S viridans* (Table 1).

Table 2 gives brief details of 10 of the 67 patients who had undergone a dental procedure without antibiotic cover but in whom the organism isolated on blood culture or other factors suggested that the portal of entry of infection was not dental. Table 3 gives similar details of nine of the 48 patients with dental sepsis in whom the infective endocarditis was unlikely to have been of dental origin. Table 4 gives details of the seven patients who despite antibiotic prophylaxis developed infective endocarditis after dental treatment.

In 230 of the 541 patients the doctor, the dentist, and the patient were unaware of the presence of any

Table 1 Cases of infective endocarditis in relation to dental procedures and dental disease

	All cases of infective endocarditis		Cases of infective endocarditis due to <i>S viridans</i>	
	No	% of total	No	% of total
Dental procedure before onset of disease				
Within 1 month			20	3.7
Within 3 months	74*	13.7	33	6.1
Dental sepsis	48	8.8	33	6.1
No dental procedure and dental state healthy or edentulous	422	77.5	176	32.4
Total	544	100	262	48.3

*Seven with and 67 without antibiotic prophylaxis.

Table 2 Patients who underwent a dental procedure without antibiotic cover within three months of the onset of infective endocarditis but in whom the portal of entry was probably not dental

Case No	Sex	Age	Organism	Dental procedure	Latent interval (months)*	Other features
8	M	32	<i>Staphylococcus</i>	Filling	2	Drug addict. Also received dog bite to hand within four weeks of onset. Bicuspid aortic valve found at aortic valve replacement
59	M	18	<i>Enterococcus</i>	Filling	3	Previously normal heart valve found at aortic valve replacement
121	M	44	<i>Staph albus</i>	Scaling and filling	2	Known aortic incompetence
122	M	38	<i>Staph epidermidis</i>	Scaling	1	Previous infective endocarditis
141	M	37	<i>Staph aureus</i>	Not stated	1	Chronic glomerulonephritis. Renal failure
157	F	47	<i>Str faecalis</i>	Scaling	3	Previously undiagnosed rheumatic heart disease. Illness followed an operation for piles
173	M	38	<i>Str viridans</i>	Extraction	—	Infective endocarditis started four weeks before extraction. Known "floppy" mitral valve
256	F	39	<i>Str pneumoniae</i>	Scaling and filling	3	No known heart disease before onset. Followed aspiration of a pleural effusion from which <i>Str pneumoniae</i> was cultured
273	M	63	<i>Str bovis</i> biotype 1	Scaling	2	No known previous cardiac abnormality
374	M	73	<i>Str viridans</i>	Scaling and filling	—	Had been ill for several months before dental procedure. Known congenital heart disease

*Approximate time between dental procedure and onset of symptoms.

Table 3 Patients with dental sepsis thought to be unrelated to infective endocarditis

Case No	Sex	Age	Organism	Other features
16	F	61	<i>Str bovis</i>	Had congenital heart disease but this was not known
34	M	50	<i>Staph aureus</i>	Had a previously normal heart. Required aortic valve replacement
103	M	60	<i>Enterococcus</i>	Previous aortic valve replacement for calcific aortic stenosis. Suture of lacerated thumb without antibiotic cover shortly before onset
105	F	54	<i>Str bovis</i>	May have had congenital heart disease but this was not known
150	M	44	<i>Str bovis</i> biotype I	No known previous heart disease
175	M	77	<i>Str bovis</i> biotype I	Gastric polyp. Known mitral incompetence of uncertain origin
264	F	74	<i>Staph aureus</i>	Known rheumatic heart disease
434	F	63	Negative	Followed gastroscopy. Previous mitral and tricuspid valve replacement
506	M	59	<i>Str faecalis</i>	Known previous heart disease presumed degenerative. Sudden death while awaiting aortic valve replacement

Table 4 Details of patients developing endocarditis after a dental procedure despite antibiotic prophylaxis

Case No	Sex	Age	Organism	Dental procedure	Latent interval (months)*	Antibiotic prophylaxis	Other features
30	M	56	<i>Str viridans</i>	Scaling	1	Oral erythromycin	Previous aortic valve replacement and pulmonary autograft for congenital heart disease
184	M	6	<i>Str viridans</i>	Extraction of four teeth	1	Oral penicillin three days before and seven days after	Previous Blalock-Taussig shunt for univentricular heart and pulmonary stenosis
375	M	57	<i>Str mutans</i>	Extraction	2	Erythromycin and rifampicin (penicillin allergy)	Mitral incompetence of unknown aetiology. Previous infective endocarditis
382	F	58	Blood culture negative	Extraction	1	Oral penicillin started 48 h before extraction	Rheumatic heart disease
429	M	49	Two different micrococci	Scaling	3	Amoxycillin	Rheumatic heart disease. Previous mitral valve replacement. Severe gardening laceration shortly before onset
449	M	31	<i>Str viridans</i>	Filling	2	Oral penicillin	Congenital heart disease
479	M	70	<i>Str viridans</i>	Scaling	1	Ceporex 500 mg three times daily 24 hours before and five days after	Previous aortic valve replacement

*Approximate time between dental procedure and onset of symptoms.

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cardiac abnormality before the onset of infective endocarditis (Table 5). In some it was clear at necropsy or at valve replacement that no pre-existing cardiac abnormality had been present, and it also appeared that patients who were drug addicts or immunosuppressed or those on chronic dialysis usually had normal hearts before their infective endocarditis developed. In a number of patients a bicuspid aortic valve was present and unrecognised, and in others it was uncertain whether the valve damage was due to infective endocarditis on a normal valve or infective endocarditis on a valvular or other cardiac abnormality not previously diagnosed. It appears, however, that many more than half of these 230 patients had normal hearts before infective endocarditis developed. Table 6 shows similar details for

those with dental sepsis and those who had had a dental procedure within three months of the development of infective endocarditis, and this table attempts to quantify the number without previous cardiac abnormality, although this estimate may well be inaccurate.

In most of the 544 episodes no portal of entry for the infection was apparent but in a number of cases infection may have occurred through the skin or alimentary, urinary, or respiratory tracts. Patients who were immunosuppressed, diabetic, or dependent on alcohol and, more particularly, those who had had valve replacement or previous infective endocarditis seemed especially susceptible (Table 7).

Discussion

As we expected the number of proformas returned fell far short of the total incidence of infective endocarditis in the British Isles during 1981 and 1982. This was evident from the Offices of Population Censuses and Surveys mortality statistics¹⁹ and from the weekly Communicable Disease Reports.²⁰ Incomplete reporting is also indicated by the fact that only 74 fatalities (13.7%) were notified, well below the predicted figure. Nevertheless, we do not believe that this shortfall introduces any bias.

Table 1 shows that in 74 of the 544 cases a dental procedure was carried out with or without antibiotic prophylaxis within three months of the onset of infective endocarditis and that in another 48 overt dental sepsis was present. Tables 2 and 3 identify those patients in whom a more probable, non-dental portal of entry for the infection was present or in whom the organism grown on blood culture was unlikely to have been of dental origin and two patients in whom the illness occurred before the dental procedure. Subtracting these reduces the total to 64 (11.7%) for dental procedures and 39 (7.1%) for dental sepsis. Of these 103, 84 were due to *S. viridans*.

Cates and Christie⁹ suggested that the incubation period might be up to three months and this has been believed and quoted ever since. Certainly, in staphylococcal infections it is often shorter as it is in many cases of viridans infection (Table 1).

When considering the importance of these apparent dental risks the incidence of dental procedures and the proportion of subjects with dental sepsis in the general population should be taken into account. A recent publication²¹ shows that 60% of the school roll in maintained schools are inspected annually by the Community Dental Service or General Dental Service, although Todd six years earlier suggested a lower proportion.²² Among adults²³⁻²⁶ about half go to their dentists for a regular check up and about another one sixth attend occasionally. Many publica-

Table 5 Proportion of the 541 patients with known pre-existing cardiac abnormality and with unknown or no cardiac abnormality

	No patients	% of total
Previously known cardiac abnormality	307	56.7
No known pre-existing cardiac abnormality	230	42.5
Not stated	4	0.8
Total	541	100

Table 6 Proportion of the 122 patients whose infective endocarditis may have been of dental origin with known, unknown, and no cardiac abnormality before the onset of illness

	No patients	% of total
Previously known cardiac abnormality	73	60.0
Cardiac abnormality not known before onset of illness but probably present	22	18.0
Probably no cardiac abnormality before onset of illness	27	22.1
Total	122	100

Table 7 Sources of infection other than dental

	No of patients
Alimentary tract	22
Genitourinary tract	23
Respiratory tract	17
Skin	14
Drug addiction	6
Fractures	4
Occurring during pregnancy (2) or after parturition (1)	3
Related to non-cardiac surgical vascular procedure (1), cardiac catheterisation (5), venogram (1), blood donation (1), anticoagulant treatment (1), and chronic haemodialysis (6)	15
Within two months of valve replacement or other cardiac surgery	13

tions do not state exactly what intervals are meant by the term "regular check ups", doubtless because the variable pattern makes it impossible, but the general concept of "regular" seems to be at least twice a year. Whether these reports are valid for the whole population is questionable, but if they are it is obvious that about a quarter of all people—whether ill or not—when asked if they have had any dental procedure within the previous three months will say that they have, though in most this will have been only scaling and polishing. This proportion is more than double that found in the patients with infective endocarditis in this study. For periodontal disease the figures quoted vary,^{24 27-29} but the incidence increases with age and it is more common in those who do not attend their dentist regularly. Published work suggests that periodontal disease may be present in more than one third of adults, which may reflect the fact that in these surveys it is being searched for particularly thoroughly. The finding of possibly relevant dental sepsis in only 7.0% of our 541 patients—a little over one fifth of that found in the general population—may be due partly to failure to record its presence in hospital notes even though a high proportion of the patients had been reviewed by consultant dental surgeons.

It seems likely that infective endocarditis is sometimes of dental origin, and the high proportion (84 of 103) of viridans infections in those who had undergone dental procedure within the previous three months and those with dental sepsis supports this hypothesis. Moreover, Table 1 shows that in only 176 (42%) of 422 patients without dental sepsis and who had not undergone any dental procedure was the causal organism of the viridans species; though it is unrealistic to think that many of these 176 infections did not arise from the mouth how the organism entered the bloodstream is uncertain. In those whose dental state is considered healthy brushing the teeth and chewing⁸ may be responsible, and doubtless some may have minor and unrecognised periodontal infections. *S. viridans* abound in the mouth and nasopharynx and were responsible for nearly half of the 544 cases of infective endocarditis. On the evidence we have antibiotic cover of dental procedures seems to contribute little to reducing the incidence of the disease, but of course we do not know of those given such prophylaxis successfully nor those with cardiac abnormalities who came to no harm from a dental procedure without antibiotic cover. If we consider the enormous number of dental procedures carried out the risk of subsequent endocarditis is clearly small and it is certainly not confined to those with cardiac abnormalities. The magnitude of the bacteraemia, the virulence of the organism, and perhaps most of all the resistance of the host are probably determining factors. Older people, diabetics, those

dependent on alcohol, those who are immunosuppressed, and those with cancer are especially vulnerable, and foreign bodies such as prosthetic valves and previous infective endocarditis also increase susceptibility to infective endocarditis.

The dental prophylaxis of infective endocarditis was made more difficult by the fact that pre-existing heart disease was not known to be present in more than two fifths of the patients. Indeed, in a substantial proportion the heart appeared normal on clinical examination (Tables 5 and 6). Those with no known cardiac abnormality cannot be protected from dental hazards unless everyone undergoing a dental procedure and perhaps everyone brushing their teeth or chewing receives antibiotic cover. This would be impossible to implement and would be unacceptable to most patients, who regard a visit to the dentist as having a lower priority than going to the bank and are aware of the not infrequent adverse effects of antibiotics.

Regular and careful dental care is clearly important for those with known heart disease and they should certainly have antibiotic cover when dental procedures are undertaken. As already indicated there are considerable difficulties in identifying those for whom such measures are important and there are other problems. Usually consultation between doctors and dentists is infrequent, and many physicians and general practitioners giving advice and instituting prophylactic measures are unaware of what should be offered. This is evident from Table 4. Furthermore, among those who had dental procedures without antibiotic cover were a consultant surgeon with congenital heart disease, a doctor with a systolic murmur since youth, and two patients who were instructed in the importance of dental prophylaxis but told that it was necessary only for extractions. There was also the dental surgeon (case 429, Table 4) who had had a valve replacement but was not given antibiotic treatment when he lacerated his hand while gardening.

The recommendations of the American Heart Association on dental prophylaxis³⁰ are complicated and mostly entail the use of injected antibiotics; they are totally unrealistic for guidance of doctors and dentists in the British Isles. The measures recommended by the British Society for Antimicrobial Chemotherapy³¹ (Appendix) are more appropriate and should be widely publicised and implemented in patients with known cardiac disease. The *Dental Formulary* should be suitably amended. In this matter the work of Shanson *et al.* regarding tolerance to oral erythromycin³² and amoxycillin³³ is important. They suggest 1.5 g erythromycin or 3.0 g amoxycillin as the most effective prophylactic dosage and that least likely to provoke gastrointestinal or other symptoms, which is the same as that recommended by the British

Society for Antimicrobial Chemotherapy.³¹

Looking back to Horder's³⁴ description of 150 cases in 1909, to Cates and Christie's⁹ large series in 1951, and to subsequent publications¹⁰⁻¹³ it is clear that the disease has changed considerably. Firstly, the advent of antibiotics has reduced mortality from 100% to about 30%; secondly, the fall in the incidence of rheumatic heart disease and increased longevity have raised the mean age of those attacked and made the aortic valve more vulnerable than the mitral valve, which, when affected, is usually due to the presence of mitral valve prolapse; thirdly, normal hearts are now often attacked; and, fourthly, emergency valve replacement has made it possible to save many who would otherwise die. Certain features are not, however, changing. Most cases are still streptococcal; in England and Wales the number of cases, apart from a peak after the first world war, appears much the same or a little more than it was over 70 years ago.³⁴ From 1911 to 1918 there was an average of 573 deaths from infective endocarditis each year in England and Wales. During 1919-21 this figure rose to 1203, doubtless due to the influenza epidemic and its streptococcal complications. From 1931 to 1944, before antibiotics became available, the average was 964 each year, and during the past 25 years the number of deaths due to infective endocarditis have been 318 in 1958, 321 in 1963, 282 in 1968, 259 in 1973, and 215 in 1978. With a mortality of 30% this means that there are now as many or more cases than there were in Horder's day, but the actual number of deaths is gradually declining, even more than the figures suggest as the population has increased over this period. This unchanged incidence and only slightly decreasing mortality suggest either that people's teeth are not putting them at great risk or that dental prophylaxis is not being given when it should be and that when it is applied is not always being carried out effectively. With regard to dental prophylaxis our findings substantiate both these criticisms and also indicate that although infective endocarditis is sometimes of dental origin such causation is currently less common than previously believed.

Infections and lesions of the alimentary, genitourinary, and respiratory tracts and of the skin and procedures for their investigation and treatment are assuming increasing importance in the causation of infective endocarditis. Patients who are immunosuppressed, diabetic, dependent on alcohol, and particularly those with prosthetic valves or previous infective endocarditis are at especial risk (Table 7). The proportion with prosthetic valves may, however, be overrepresented in this series.

A separate publication will give details of the pathogenesis and microbiology of the 544 episodes of infective endocarditis reported here.

We thank members of the British Cardiac Society and the fellows and members of the Royal College of Physicians who participated in this survey and Dr Susan Young of the Communicable Disease Surveillance Centre.

Appendix

Summary of recommendations of the British Society for Antimicrobial Chemotherapy for the dental prophylaxis of subacute bacterial endocarditis

- (1) Patients not allergic to penicillin
 - (a) Adults—3 g amoxycillin orally one hour before dental procedure
 - (b) Children under 10—half the adult dose
 - (c) Children under 5—one quarter of the adult dose
- (2) Patients allergic to penicillin and those who have recently had penicillin treatment
 - (a) Adults—1.5 g erythromycin orally 1-2 hours before the dental procedure and 0.5 g six hours later
 - (b) Children under 10—half the adult dose
 - (c) Children under 5—one quarter of the adult dose
- (3) Patients having a general anaesthetic
 - (a) Adults—1 g amoxycillin intramuscularly before induction and 0.5 g amoxycillin by mouth 6 hours later
 - (b) Children under 10—half the adult dose

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