The teeth and infective endocarditis

RICHARD BAYLISS, CYRIL CLARKE, CELIA OAKLEY, WALTER SOMERVILLE, A G W WHITFIELD

From the British Cardiac Society and the Medical Services Study Group of the Royal College of Physicians

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 Grant suggested that transient bacteraemia might cause infective endocarditis in those with abnormal heart valves, and in 1930 Rushton described endocarditis after dental extraction. Five years later Okell and Elliott reported a high incidence of streptococcal bacteraemia after dental extraction. Burket and Burn 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. Taran in 1944 reported four children with negative blood cultures before tooth extraction who died from streptococcal infective endocarditis after the procedure. Scaling, gingivectomy, dental irrigation, brushing the teeth, and even chewing 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 Christie 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 Hospital 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 infection; in a later series from Birmingham only four of 70 patients had had recent dental treatment. In 93 episodes of culture positive endocarditis at St Thomas’s Hospital, however, 31 (33%) were thought to be of dental origin, and Oakley and others indicated that in as many as 40% of patients a history of a dental procedure during the previous three months could be
Elicited. By contrast, a possible dental cause was present in only 9% of a series from the University of Washington Hospitals. 15 Fogrel and Welsby 16 reported a recent dental extraction in only 5% of 83 cases, and Cherubin and Neu 17 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 culture; 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>
<thead>
<tr>
<th>Table 1 Cases of infective endocarditis in relation to dental procedures and dental disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>All cases of infective endocarditis</strong></td>
</tr>
<tr>
<td>---------------------------------------------</td>
</tr>
<tr>
<td>Dental procedure before onset of disease</td>
</tr>
<tr>
<td>---------------------------------------------</td>
</tr>
<tr>
<td>Within 1 month</td>
</tr>
<tr>
<td>Within 3 months</td>
</tr>
<tr>
<td>Dental sepsis</td>
</tr>
<tr>
<td>No dental procedure and dental state healthy or edentulous</td>
</tr>
</tbody>
</table>

*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

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

*Approximate time between dental procedure and onset of symptoms.

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

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

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

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

*Approximate time between dental procedure and onset of symptoms.
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 statistics and from the weekly Communicable Disease Reports. 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 Christie suggested that the incubation period might be up to three months and this has been believed and requested 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 publication 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. Among adults about half go to their dentists for a regular check up and about another one sixth attend occasionally. Many publica-
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 popula-
tion 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, 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 thor-
oughly. 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 some-
times of dental origin, and the high proportion (84 of
103) of viridans infections in those who had under-
gone 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 infec-
tions. S viridans abound in the mouth and nasopharynx
and were responsible for nearly half of the 544 cases of infective endocarditis. On the evi-
dence 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 con-
sider the enormous number of dental procedures car-
rried out the risk of subsequent endocarditis is clearly
small and it is certainly not confined to those with
cardiac abnormalities. The magnitude of the bacte-
riaemia, 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 immunosuppres-
sed, and those with cancer are especially vulnerable,
and foreign bodies such as prosthetic valves and pre-
vious infective endocarditis also increase susceptibil-
ity 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 proce-
dure 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 antibiot-
ics.

Regular and careful dental care is clearly important
for those with known heart disease and they should
certainly have antibiotic cover when dental proce-
dures are undertaken. As already indicated there are
considerable difficulties in identifying those for whom
such measures are important and there are other prob-
lems. Usually consultation between doctors and
dentists is infrequent, and many physicians and gen-
eral 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 anti-
biotic 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 cover
when he lacerated his hand while gardening.

The recommendations of the American Heart
Association on dental prophylaxis 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 recom-
bended by the British Society for Antimicrobial
Chemotherapy (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
erthyromycin32 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
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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 publications16-13 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

References

1 Lewis T, Grant RT. Observations relating to subacute infective endocarditis. Heart 1923; 10: 21-99.

2 Rushton MA. Subacute bacterial-endocarditis following the extraction of teeth. Gey's Hospital Reports 1930; 80: 39-44.


BAYLIS, CLARKE, OAKLEY, SOMERVILLE, WHITFIELD


Requests for reprints to Medical Services Study Group, Royal College of Physicians, 11 St Andrew’s Place, London NW1 4LE.
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R Bayliss, C Clarke, C Oakley, W Somerville and A G Whitfield

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