Section of Odontology

President L J Godden OBE FDS RCS

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Relation Between Oral Disease and Acute Bacterial Endocarditis [*Abridged*]

Professor Brian E D Cooke
(*Department of Oral Medicine and Oral Pathology, Dental School, Welsh National School of Medicine, Heath, Cardiff*)

Dental Bacteriæmia

The association between oral disease and bacterial endocarditis will probably never be proved statistically, but I believe there is enough circumstantial evidence to recommend us all to take certain precautions during surgical procedures. Nevertheless, it is not certain that antibiotic cover will prevent bacteriæmia or even bacterial endocarditis.

Obviously, the smaller the number of organisms and the shorter time they circulate in the blood stream, the less likelihood there is of complications. Thus, the dental manipulative procedure is of greater importance than oral disease *per se* in the production of bacteriæmia. The all-embracing term 'oral disease' is in fact only replacing the older and equally vague term 'oral sepsis'. This latter term has befogged our interpretation of the relationship between any specific dental disease, such as gingivitis or periodontitis, and bacteriæmia.

Cates & Christie (1951), reporting an unselected group of 215 cases of bacterial endocarditis, listed dental caries as a focus of infection but no special mention is made of periodontal disease and gingivitis. Presumably dental sepsis is meant to cover the latter, but it would be most interesting to know something of the dental health of the 82 patients in whom a focus of infection was not found.

Just as the dental health of any patient in a series of bacterial endocarditis is rarely referred to in precise fashion, even rarer is any reference to the state of well-being and general fitness of patients presenting for extractions and other dental procedures who were shown at a later date to develop bacterial endocarditis. In Cates & Christie's series, teeth were removed less than three months before the onset of symptoms in 23 cases (10·7%), but in another 8 cases (3·7%) teeth were removed after the onset of symptoms. This lends some support to the suggestion of Feldman & Trace (1938) that dental extractions may have no causal relationship to bacterial endocarditis and that teeth are removed to relieve symptoms of bacterial endocarditis before diagnosis. Furthermore, it appears that there is a complete absence of follow up of those patients at risk, and that the relationship between dental procedures and bacterial endocarditis is generally made in retrospect.

Bacterial endocarditis is a rare disease, while it is very common for a patient to have extraction of teeth, particularly between the ages of 20 and 40. Since it is unlikely that a patient will remember having a tooth out 3–6 months before a serious illness unless there was something about that extraction that the patient remembers, it is unlikely that the patient will spontaneously mention this when presenting at hospital; equally likely is the fact that the question may not even be asked in relation to the extractions, so that the number of patients presenting who have had extractions 3–6 months before an attack of bacterial endocarditis may well be higher than that generally recognized in the literature. Even if this figure or proportion were known, it would be difficult to determine whether or not the relationship is important or just a chance one. Nevertheless, the many case histories in the literature leave no doubt as to the causal relationship between dental extractions and the attack of bacterial endocarditis; Horder (1909) and Rushton (1930) may be quoted as examples.

Patients at Risk

There is no question whatsoever that a number of medical and dental practitioners do not inquire about valvular heart disease before advising or undertaking surgical treatment for patients at risk.

McGowan & Tuohy (1968) ascertained the proportion of patients at risk through careful history-taking in 2,069 patients attending the Oral Surgical Department of the Belfast Dental School in 1967. Of these patients, 113 (5%) were at risk, and of these 76% were presumed to have a cardiac lesion of rheumatic origin, 13% of congenital origin, and 11% of uncertain origin. The 113 patients with cardiac lesions had been exposed to 424 dental surgical procedures covering extractions, scalings or root canal therapy, all of which presumably could have resulted in a transient bacteriæmia. On only 23% of occasions was suitable cover given.

Furthermore, in 64% of these patients no enquiry had been made by the dental practitioner as to whether or not the patient might have been suffering from heart disease of the type which could place him at risk. Only 11% had been warned by their general medical practitioner that any dental or other surgical treatment would require special precautions.

The question which seems unanswerable is the degree of risk in those exposed to 327 dental procedures without appropriate antibiotic cover.

According to Wilkinson (1967), in four recently published series of 428 patients suffering from bacterial endocarditis and presenting shortly after dental treatment, only 12% did not have prophylactic antibiotic cover. He thought that there would be 20% of patients at risk who would not know of any heart lesion, but the majority of those with rheumatic heart disease would be under medical supervision and those with congenital heart disease would also know of their lesion. Wilkinson suggests that every patient at risk should carry a printed card indicating the diagnosis and emphasizing the importance of showing this card to the dentist at every visit.

Bacterial Endocarditis Following the Extraction of Teeth

Dormer (1958) reported upon 82 patients who had experienced 97 episodes of endocarditis, of whom 11 gave a history that infected teeth had been extracted without antibiotic cover; in 9 of these the causative organism was *Streptococcus viridans*. In 47 of these patients who were infected with *Streptococcus viridans* there was no history of a recent dental extraction, so presumably this organism may reach the blood stream in the absence of dental extraction. In 12 other patients precipitating factors were identified and the series illustrates other foci of infection, apart from the teeth, which become hazards when associated with surgery such as cardiac catheterization and transurethral prostatic resection.

As scientific evidence accumulates concerning the possible relationship between dental manipulative procedures and bacterial endocarditis, a greater dissonance is shown between the theory and practice of the management of patients at risk. The medical and dental adviser who does not see that appropriate precautions are taken during surgical procedures, even though he knows of the hazard and possible consequences to the patient, reasons that the risk is small anyway, and that the patient is subject to a slight bacteriæmia every time he chews. Nevertheless, as I shall point out, the evidence for the latter fact is not at all clear.

The Infective Organism of Bacterial Endocarditis

In the Medical Research Council series (Cates & Christie 1951) the infective organism was identified as *Streptococcus viridans* in 354 patients (87%). The term *Streptococcus viridans* is not a description of a definite species but covers all the α-hæmolytic streptococci. Wilkinson (1967) states that from a study of the literature it appears to be involved in about 65% of cases of bacterial endocarditis, and from this inferred that a dental focus accounts for rather less than two-thirds of the cases.

Leading articles in the *Lancet* (1967) and *British Medical Journal* (1969) stressed the change in the pattern of bacterial endocarditis, and in particular the increase in incidence of involvement in older patients. Since the increase of bacterial endocarditis in this older age group is often caused by *Streptococcus fæcalis*, non-hæmolytic streptococci and *Staphylococcus aureus*, it may well be that disorders of the genito-urinary system, gall-bladder and colon are more likely to be sources of bacteriæmia than dental operations. In other words, if there is a change in pattern in bacterial endocarditis it may well be that the relationship between oral disease and endocarditis is less prevalent, and that other sources of infection in the body are gaining in importance, together with the fact that patients are living longer, when atheroma of the aortic valves alone may predispose to this condition.

The Pathway from the Organisms in the Mouth to the Blood Stream

Burket & Burn (1937) demonstrated that *Serratia marcescens* could be forced into the vascular system from the gingival crevice during extraction. As with later workers, they did not show an association between 'oral sepsis' and the incidence of bacteriæmia, and it was only in

Okell & Elliott's (1935) series that there was such a great variation in degree between patients.

Fish & Maclean (1936) confirmed Burket & Burn's work and at the same time gave a rationale to the findings of Okell & Elliott, for they demonstrated that organisms could be sucked into the open vessels of the periodontal membrane during extractions owing to their anatomical arrangement, and pumped into the blood and lymph channels. They suggested that rocking of the tooth caused alternatively a positive and negative pressure on the vessels of the periodontal membrane.

Dental Bacteriæmia

The work of Okell & Elliott (1935) stands out as a peak in the mountain range of achievement in bacteriology. They clearly demonstrated that there was a bacteriæmia associated with the extraction of teeth, and this fundamental fact has acted as a catalyst to many workers. Nevertheless, the interpretation of their findings is not altogether as straightforward as many would have us believe. First, the extractions were carried out under a general anæsthetic, and there is little doubt that the high proportion of positive blood cultures obtained by these workers was the result of the impact of a gag on the teeth, particularly in those patients with loose and mobile teeth. Furthermore, there was no accurate evaluation of what was meant by marked and moderate gum disease. Patients with marked gum disease might have had easy extractions compared with those with moderate or none, so that the fact that the post-operative blood cultures were 75% positive in patients with marked gum disease and only 34% positive in patients with no gum disease, suggests that the gag may have been an important factor.

After Okell & Elliott, perhaps nobody has done more than Bender *et al.* (1963) in various studies of this problem, and in particular the conditions affecting sensitivity of techniques for detection of bacteriæmia. They evaluated these techniques and concluded without doubt that bacteriæmias occur in all patients following extractions, but detection is dependent on the size of the blood sample and the refinements of technique. Rogosa *et al.* (1960) supported the findings of Bender and his colleagues with the most meticulous studies. Elliott & Dunbar (1968) did not find that the easier extractions of deciduous and young permanent teeth in children diminished the possibility of acquiring a temporary streptococcal bacteriæmia. Furthermore, they showed that penicillin therapy adequate for infectious oral conditions may not be sufficient to reduce materially the incidence of a post-dental extraction bacteriæmia.

Bender & Pressman (1956) demonstrated that extensive rupture of capillaries in or around the gingival sulcus in extractions demanding severe trauma produced bacteriæmia in 93·4%, but in mild trauma, 68·7%. This is consistent with the results of other studies. Nevertheless, Robinson *et al.* (1950) found that there is no statistical relationship between bacteriæmia and type of anæsthesia, or the age, sex, tooth vitality and mobility, pocket formation or gingival inflammation or radiographic apical areas. Perhaps most important of all, they found there was no individual predisposition to bacteriæmia in patients who have bacteriæmia on the first extraction.

Frequency of Bacteriæmia Following Chewing Food or Toothbrushing

The following sentence, taken from Elliott's paper (1939) entitled 'Bacteriæmia and Oral Sepsis' and presented to this Section 30 years ago, has probably caused more confusion in people's minds than any other factor:

> 'Since we know that in severe pyorrhœa slight degrees of dental trauma, such as might be occasioned by biting on a loose tooth, commonly lead to a bacterial shower in the blood-stream, it is apparent that under those conditions there is probably an almost constant intermittent leakage of organisms from the mouth into the blood.'

This reasoning was based on the former findings of Okell & Elliott that in 12 of their 138 cases of extraction under general anæsthetic streptococci were recovered from a specimen of blood taken before operation, and on one further case of his own, in another series, which had a positive blood culture before extraction. These patients were all thought to have severe gum infection. My own thoughts are that these were in fact probably teeth traumatized through the application of gags preparatory to the nitrous oxide and oxygen anæsthesia given in those days when the teeth had to be extracted fairly quickly under difficult conditions. On this evidence Elliott said it was thus easy to imagine that a very slight degree of trauma in a mouth the subject of marked gum infection was sufficient to produce blood invasion. Elliott quoted Round *et al.* (1936) for further evidence to support his thoughts on this occasional bacteriæmia. These workers took 10 ml of blood from 10 patients who had chewed mint lumps for 10 minutes and found a bacteriæmia following this, one with *Staphylococcus aureus* and the other with *Streptococcus viridans*. Furthermore, these workers said they were going to do this on 100 patients, but I cannot find any evidence or record in the literature of this having been done, and

certainly this work has not been confirmed by other workers; Lazansky *et al.* (1949) gave up taking pre-operative blood samples after the first 161 cases proved sterile.

In order to support his thought that mastication or even brushing of the gums might produce temporary bacteriæmia, Elliott (1939) showed that rocking of a tooth in the presence of marked gum disease before an extraction caused a streptococcal bacteriæmia in 18 out of 21 cases, and in 5 out of 20 cases in those patients with clean mouths. But, once again, these extractions were all done under a general anæsthetic, and I believe it was caused by gag pressure on teeth that were not necessarily going to be extracted in patients with marked gum disease.

Thus, in summary I can find no evidence that mild trauma such as mastication or brushing the teeth can cause a transitory bacteriæmia from any mouth.

Endodontic Treatment

Over the years Bender *et al.* (1963) have strongly advocated endodontic treatment as the least likely to produce bacteriæmia compared with exodontia and periodontia. They demonstrated that when manipulations in 50 patients were restricted to the root canal there was no bacteriæmia, whereas when the reamer reached the periapical area in 48 patients the incidence of bacteriæmia was 31·2%. Nevertheless, this is small compared with that resulting from extractions which in some series can be as high as 93·4%. Thus, antibiotic cover is still important for patients at risk undergoing endodontic treatment.

Fillings

Dormer (1967) suggests that fillings as well as extractions may precipitate bacterial endocarditis. Nevertheless, there appears to be no evidence that bacteriæmia may accompany or follow the filling of a tooth.

Periodontal Treatment

Bender *et al.* (1963) demonstrated that periodontal treatment in 10 of 12 patients had a positive culture following gingivectomy, and of 15 patients with deep scaling 8 had positive bacteriæmias and only 6 of 20 after superficial scaling.

The Edentulous Patient

Streptococcus viridans endocarditis is infrequent in the edentulous patient, although it has been occasionally reported and thought to result from the nipping of cheeks by ill-fitting dentures or even ulcers on the alveolar ridge. The increasing incidence of deaths due to bacterial endocarditis in the age group when the patients are often edentulous is, of course, quite unrelated. I would have thought there is now enough evidence to suggest that elective surgery with appropriate precautions resulting in removal of the patient's remaining teeth was probably the best course to be taken after an attack of bacterial endocarditis.

Conclusion

The following pattern of case management varies only slightly from that proposed by Bender *et al.* (1963):

(1) A careful history is taken with particular reference to rheumatic fever, chorea or congenital heart disease.
(2) Every patient known to be at risk should carry a card for presentation to the dental surgeon at every visit.
(3) Patients are advised to attend for regular dental treatment so that all procedures can be elective.
(4) Bacteria in the gingival crevice are tested against antibiotics.
(5) Appropriate antibiotics should cover all dental procedures that might facilitate entry of bacteria into the blood stream.
(6) A local anæsthetic is used in preference to a general anæsthetic.
(7) Ideally, only one tooth should be extracted at a time with the minimum of trauma – or 20 minutes allowed between extractions if the patient is under antibiotic cover, for under such conditions bacteriæmia lasts for only 10–15 minutes.
(8) A light scaling to one or more teeth is preferable to heavy scaling, and electrosurgery to gingivectomy.
(9) Patients are advised to report to their doctor immediately should they feel unwell during the weeks following any operative procedure.
(10) After the patient has recovered from the first attack of bacterial endocarditis it is probably best for all the remaining teeth to be extracted, with the provision of full dentures.

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Professor G R F Hilson
(Department of Medical Microbiology, St George's Hospital Medical School, London SW1)

Is Chemoprophylaxis Necessary?

In considering the connexion between oral disease and endocarditis a statement in fairly crude form of the theory and practice involved, generally accepted at present, is as follows: it is assumed that episodes of bacteriæmia with organisms of oral origin are relatively common in the majority of individuals, especially after dental extraction (Okell & Elliott 1935), and that in a minority there exist abnormalities of cardiac structure which render them liable to localize organisms in the circulation on the heart valves or other parts of the endocardium, with subsequent development of vegetations and other pathological features so well known in this condition; on this basis it is thought reasonable to give chemotherapeutic cover during dental procedures so as to intercept the organisms before they can be localized on the valve, or even to carry out dental procedures, again under suitable cover, aimed at reducing or eliminating the risk of bacteriæmias of dental origin. This approach may be a valid one: but it is just as well to examine the various sources of evidence for its justification and to evaluate the practical measures involved. For this purpose we need to consider the nature and incidence of bacterial endocarditis, the relationship to it of the oral flora and the mechanisms involved in its pathogenesis, and some of the problems of chemotherapy; the principles of diagnosis and treatment of the individual case, however, are of interest only in so far as they shed light on aspects of bacteriæmia and chemotherapy.

It is the custom to divide bacterial endocarditis into two categories: the acute form, mainly due to highly pathogenic bacteria, and the subacute form caused by organisms of low intrinsic pathogenicity. It is widely accepted that the mouth is the main source of the commensal streptococci found in subacute bacterial endocarditis, and that this is the condition, rather than the acute infection, which is connected with oral disease and dental manœuvres. In the rest of this paper, therefore, I shall concentrate on the problems which appear to arise out of this concept of the causation of subacute bacterial endocarditis in susceptible individuals by transient bacteriæmias of streptococci of oral origin, mainly exemplified by *Streptococcus viridans*; however, some reference to the more general problem of bacterial endocarditis as a whole will be needed from time to time.

Incidence of Subacute Bacterial Endocarditis (SBE)

Most of the data available refer to bacterial endocarditis of all kinds and show in general that the death rate from such infections began to fall in the 1930s, coinciding with the use of sulphonamides, and declined steeply until about 1954, since when the annual rate has fallen only slightly. In this country, from the Registrar-General's returns, the annual number of deaths has fallen from about 1,000 to just under 300 in recent years (Dormer 1966). The fall in the number of deaths is attributable to the introduction of penicillin and other antimicrobial agents; these drugs have, however, influenced the incidence of cases to a much lower degree. In the earlier records, since the fatality rate was 100%, incidence and death rates were numerically equal; now, however, the lives of about 3 in every 4 patients can be saved (Lerner & Weinstein 1966, Hampton & Harrison 1967). Since the death rate from bacterial endocarditis is now about one-quarter of what it was in the 1930s, and the case fatality rate is also down to 1 in 4, it can easily be seen that the case incidence of bacterial endocarditis has remained essentially unaltered despite the widespread use of chemotherapeutic agents active against the causative organisms – uses which may have been with the intention of prophylaxis, or merely coincidental. A trend towards the involvement of older age groups, especially males, is evident. One can make some attempt to study the incidence of SBE as such, assuming an overall unaltered incidence of bacterial endocarditis. In a series of 221 cases of bacterial endocarditis at the University of Minnesota Hospitals (Pankey 1961, 1962) between 1939 and 1959, 75% were SBE; in the more recent series of Lerner & Weinstein (1966) 57% were SBE, with a case fatality rate of 12%. It is likely, therefore, that there has been a slight reduction in the incidence of SBE, and that there is a significant reduction