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TEETH, STREPTOCOCCUS VIRIDANS, AND SUBACUTE BACTERIAL ENDOCARDITIS

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The discovery and therapeutic use of antibiotics has completely changed the pattern, course, and prognosis of many familiar diseases, of which subacute bacterial endocarditis (S.B.E.) with its previous 99% mortality (Lichtman, 1943) provides a dramatic example. The literature upon the subject is now considerable, but the many published investigations have been concerned primarily with the broad and obvious problems of the disease—namely, the associated cardiac abnormality, acquired or congenital; the incidence of the disease upon these abnormal hearts; the sex and age incidence; the organism concerned; the choice and dosage of the antibiotic to be employed; the duration of treatment; and the prognosis.

Penicillin in particular (Cates and Christie, 1951) has been outstandingly efficacious and the immediate results of treatment have been good, but in the long-term follow-up the relapse rate has been disappointingly high. It is doubtful whether enough attention has been paid to some of the aetiological factors, in particular to the focus of infection from which the invading organism is discharged into the blood stream to become established upon the structure of a pathological heart. The point is fundamental in the consideration of the therapeutic mastery of the disease and the prevention of relapse. It is the purpose of this communication to demonstrate the importance of the primary focus of infection in the teeth and dental sockets, and its relation to the onset of the disease, to the occasionally unsatisfactory immediate response to therapy, and to the tendency to relapse.

S.B.E. can be divided into two broad categories: (a) cases in which *Streptococcus viridans* is the organism concerned, and (b) those in which other organisms are concerned. In each category the severity and character of the precedent underlying cardiac lesions have much in common, and the presence or absence of cardiac failure will profoundly influence the prognosis in any individual case. There are, however, more striking differences in the severity of the disease, the treatment, the prognosis, the tendency to relapse, and the aetiology.

Category A.—S.B.E. due to *Str. viridans* is generally less severe, its therapy rarely presents major problems, the immediate prognosis is more favourable, but the tendency to relapse is high. The teeth are commonly regarded as the most important focus of infection.

Category B.—S.B.E. due to other organisms is more acute, presents therapeutic problems related to the organism concerned, carries a less favourable immediate prognosis, but is unlikely to relapse if the initial attack is successfully treated. The focus of infection is usually located in a site other than the teeth.

Clinical Material

Our data have been collected from a study of the clinical records of 59 patients who were admitted to the Radcliffe Infirmary, Oxford, in the period 1945–55, and were diagnosed as suffering from S.B.E.

S.B.E. due to Str. viridans (43 patients).—The diagnosis was confirmed by a positive blood culture in 25 patients (58%). Of these, four died while under active treatment, and four subsequently died of heart failure at intervals of one, two, two, and seven years. In 18 patients (42%) with a negative blood culture the diagnosis rested on the clinical picture. Seven patients died while under active treatment, and in five of these the clinical diagnosis was confirmed at necropsy.

S.B.E. due to other Organisms (16 patients).—The responsible organism was as follows: *Anaerobic streptococcus*, two patients; non-haemolytic streptococcus, three; *Staph. aureus*, six; *Staph. albus*, one; and pneumococcus, four. Five of these patients died while under active treatment.

Age Incidence and Aetiology

The age incidence and cardiac pathology are shown in Tables I and II. It will be seen that our small series has one feature in common with several major published series—namely, that S.B.E. is a rare disease in persons under 15 years of age, and thereafter shows a rapid and formidable increase, with a maximal incidence on the

age groups 15-40 years, when acquired cardiac lesions have become established, and that from the age of 40 onwards there is a progressive steady fall in the age groups.

TABLE I.—Age Incidence, Pathology, Mortality

Age		S.B.E.				Died	
		<i>Str. vir.</i> Culture +	<i>Str. vir.</i> Culture -	Other Organisms	Total	Hospital*	Home†
5-9	C	1			1		
10-19	Rh C		1 2 (1)		1 2		1
20-29	Rh C	8 (1)	4 (2)	3 1	15 3	3	
30-39	Rh C	6 (3)	5 (3)	2 (1)	13 2	3	4
40-49	Rh	3 (1)	2 (1)	3 (1)	8	3	
50-59	Rh AS	2		1	2 1		
60-69	Rh AS	2 (2)	2 (2)	2 (2) 3 (1)	6 3	5 1	1
70+	Rh	1 (1)		1	2	1	
Total		25 (8)	18 (9)	16 (5)	59	16	6

Figures in parentheses refer to number of deaths.

Rh=Rheumatic. C=Congenital. AS=Arteriosclerotic.

* Died during course of active treatment.

† Died at home of heart failure some months after discharge.

TABLE II.—Summary of Table I

S.B.E.	Rheumatic	Congenital	Arterio-sclerotic	Died	
				Hospital*	Home†
<i>Str. viridans</i> , culture +	22	3		4	4
" "	14	4		7	2
Other organisms	11	1	4	5	—

* Died in course of active treatment.

† Died at home of heart failure after recovery.

Treatment

The antibiotic used in the treatment of patients with S.B.E. caused by "other organisms" varied according to the nature of the organism concerned. The treatment of these patients is not considered further. Penicillin was the antibiotic used in our series of patients with S.B.E. due to *Str. viridans*. Table III sets out the average amounts of penicillin employed. Readmissions due to relapses are shown under their respective years as separate cases in this table.

TABLE III.—Dosage of Penicillin Given to Patients with S.B.E. Due to *Str. viridans*

Year	Blood Culture Positive				Blood Culture Negative			
	Daily Dose (mega)	Duration	Cases	Died in Hosp.	Daily Dose (mega)	Duration	Cases	Died in Hosp.
1946	0.65	7 weeks	1	—	0.16	2 days	1	1
1947	0.75	8 "	3	2	0.7	8 weeks	1	—
1948	1.6	8.5 "	5	—	0.35	7 "	2	1
1949	1.6	6 "	2	—	0.8	6 "	2	2
1950	2.5	10 "	3	—	1.6	8 "	1	—
1951	0.9	11 "	1*	—	3.0	8 "	2	2
1952	1.5	8 "	2	—	2.5	7 "	3	—
1953	2.3	7 "	4	1	3.5	6 "	3	1
1954	11.0	14 "	3	1	5.0	7 "	4	1
1955	6.6	6 "	4	1	3.0	7 "	2	—

* Child aged 6 years.

The patients have been under the care of different physicians, but the treatment has, nevertheless, been remarkably uniform. During the first five years the penicillin was injected three-hourly, and the amounts employed were gradually increased as the antibiotic became more freely available. From 1951 onwards larger amounts of penicillin were used, given in two, or at the most three, large doses during any 24-hour period, or, alternatively, in divided four-hourly doses. In 1946 three patients were given sulphonamides in addition, and in 1950 one patient received streptomycin for one week. Streptomycin was used concurrently

with penicillin in one case in 1954, and in the same year two patients showed an unsatisfactory response to penicillin (in one instance a total daily dose of 32 million units was used) and streptomycin, chlortetracycline, or oxytetracycline was used for one to several weeks.

From Table III it will be gathered that the immediate mortality was unaffected by the increasingly more intensive therapy over the 10 years: during 1946-50 6 out of 21 patients died (28.7%), and during 1951-5 7 out of 28 died (25%). This is in agreement with the observations of Bunn and Cook (1954), who found no improvement with increased dosage of penicillin in the preceding nine-year period. Two of our patients who relapsed had their initial attack of S.B.E. in 1948, one had his first attack in 1952, and two their first attack in 1954. Since the increased dose of antibiotic has failed to produce any marked improvement in the mortality and the relapse rate there must be some other explanation for the unsatisfactory results.

Dental State

It is of great importance to consider in some detail the relationship between the dental state and the initial attack and relapse of S.B.E. due to *Str. viridans*. We have already seen that there was a rapid and formidable increase of S.B.E. after the age of 15. This increase occurs after the second dentition.

We have studied the dental state of the patients in the groups previously described. We failed to find control figures for the population at large which clearly distinguished patients with complete absence of teeth and with complete dentures from those with very poor teeth or patients who had partial dentures only.

We therefore used as a control group an unselected consecutive series of 315 in- and out-patients from our medical, general surgical, paediatric, and gynaecological and obstetrical departments. "Edentulous" is here used to indicate that the patient had full dentures and no visible teeth. The number of expected edentulous patients in each group of our series has been calculated from the figures obtained in our control group. The two subdivisions of patients with S.B.E. due to *Str. viridans* (culture-positive and culture-negative) have here been fused, for there was nothing to be gained by keeping them separate.

From Table IV emerges the remarkable fact that S.B.E. due to proved or presumptive infection with *Str. viridans* never occurred in an edentulous patient. This is readily seen from the last column of Table IV.

In the group of patients with S.B.E. due to "other organisms" the dental state did not differ greatly from that which would be expected from the control figures.

There was a definite history of dental extraction preceding the onset of the initial attack in 7 of our 43 patients with S.B.E. due to *Str. viridans*. The fact that none of these patients were edentulous strongly suggests that the mere presence of teeth "good" or "bad" was a most formidable potential source of infection with *Str. viridans* probably

TABLE IV.—Dental State According to Age Groups

	5-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	Total
Control group: No. of patients	11	27	50	50	53	42	43	39	315
No. edentulous	0	1	3	10	21	26	33	35	129
%	0.0	4	6	20	40	62	77	90	
S.B.E. due to <i>Str. viridans</i> : No. of patients	1	3	14	13	5	2	4	1	43
No. edentulous	0	0	0	0	0	0	0	0	0
Expected No. edentulous	0	0.12	0.84	2.6	2.0	1.2	3.1	0.9	10.76
S.B.E. due to other organisms: No. of patients	0	0	4	2	4	0	5	1	16
No. edentulous	—	—	1	1	3	—	3	0	8
Expected No. edentulous	—	—	0.24	0.4	1.2	—	3.85	0.9	6.59

responsible for the initial attack of S.B.E. in all cases. In spite of an apparently satisfactory immediate result of treatment the teeth as a probable source of reinfection again enter the picture, for, of our 25 patients with a *Str. viridans* infection with a positive culture, 5 (20%) relapsed after apparent cure from the first attack (Table V). Four relapsed

TABLE V.—Relapses of S.B.E. Due to *Str. viridans*

Case	Sex	Lesion	Age	S.B.E.	1st Relapse	2nd Relapse	3rd Relapse
1*	M	Rheumatic	27	May, 1948	Sept., 1955		
2	F	Congenital	35	Sept., 1948	June, 1949		
3	M	Rheumatic	26	May, 1952	Nov., 1953	June, 1954	
4	M	"	25	April, 1954	Mar., 1955		
5*	M	"	35	Sept., 1954	Jan., 1955	April, 1955	May, 1955

* Blood culture negative on first admission, but positive in relapses.

within 10 days up to 18 months after the initial attack, and one relapsed seven years after the initial attack. Four of these five patients retained "good" teeth, which had been subject to detailed clinical and radiological examination in the course of the initial attack of S.B.E. In Case 5 the teeth were not examined in such detail until the second relapse.

In the belief that teeth, "good" or "bad," present a great danger to the patient with S.B.E. due to *Str. viridans*, we have removed all teeth in seven of our patients who have had at least one attack of S.B.E. This is clearly an inadequate number to prove that our view is correct, but so far none of our seven cases have relapsed after an interval of from fifteen months up to seven years, although the expected relapse rate is 1.4.

The following brief histories of the course of the illness of the patients who relapsed show how intimately the dental history is linked with these relapses.

Case 1

A car delivery driver, born 1921, had rheumatic fever at 13, and tonsillitis followed by rheumatic carditis at 20. He was first admitted to the Radcliffe Infirmary in May, 1948, with malaise, pyrexia, and splenomegaly. Blood cultures were negative. He was treated with penicillin (800,000 units, daily) for 6½ weeks. During this period he had several attacks of unconsciousness (? due to embolic episodes), but his temperature gradually subsided and he was discharged on August 16. His teeth were described as "good" on admission. He remained well.

First Relapse.—In September, 1955, seven years after his initial attack of S.B.E., he was admitted complaining of nausea, faintness, and palpitations. He was found to have splinter haemorrhages and splenomegaly. Blood cultures yielded *Str. viridans*. His teeth remained excellent. He was treated with penicillin, 1 million units four-hourly for six weeks, and he was discharged on January 6, 1956. His teeth remained *in situ*.

Case 2

A housewife, born 1913, had congenital heart disease. She was first admitted in September, 1948, to the Acland Nursing Home, Oxford, with S.B.E. Blood culture yielded *Str. viridans*, sensitive to penicillin. She recovered after a course of antibiotic therapy lasting eight weeks. A number of defective teeth were extracted under adequate penicillin cover.

First Relapse.—In June, 1949, nine months after the initial attack, blood cultures yielded *Str. viridans* sensitive to penicillin. She was admitted to the Radcliffe Infirmary. She had partial dentures and her remaining teeth were in an excellent state of repair. She recovered after a prolonged course of penicillin and the remaining teeth were extracted under penicillin cover. *Str. viridans* was isolated from the roots of the extracted teeth. She has had no further relapses, and in 1953, at the age of 40, she had an uneventful pregnancy and delivery.

Case 3

A chemical engineer, born 1924, had rheumatic fever in 1936 and in 1942, and severe tonsillitis in May, 1951. He was first admitted to the Radcliffe Infirmary on May 28, 1952, following six weeks of pyrexia, rigors, and sweating, which had not responded to treatment with salicylates. He was found to have gross rheumatic valvular disease, Osler's nodes were present, and blood culture yielded a heavy growth of *Str. viridans*. His teeth were regarded by a dentist as clinically healthy; they showed no evidence of apical infection radiologically. He was treated with a prolonged course of penicillin, and recovered.

First Relapse.—In November, 1953, 18 months after the first attack, blood culture again yielded *Str. viridans* sensitive to penicillin, and he recovered after a further course of antibiotic. Frequent careful checks on his teeth had been made during the period following his first attack of S.B.E., including dental x-ray films, which failed to reveal any abnormality. The question of complete dental clearance was raised, but our dentist was reluctant to remove clinically perfect teeth, and as a compromise molars and premolars in both jaws were extracted under penicillin cover. It was argued that these teeth were particularly liable to be subjected to violent movement in the process of mastication. The patient remained well.

Second Relapse.—In June, 1954, seven months after his first relapse, he was readmitted. Penicillin had been started before his admission, and repeated blood cultures were negative, although the clinical evidence for S.B.E. was overwhelming. He responded well to vigorous penicillin therapy, extended over one month. The remaining teeth were extracted under, and followed by, a prolonged period of massive penicillin therapy (8 million units a day in divided doses). He has remained well since.

Case 4

A decorator, born 1929, was rejected from the R.A.F. in 1948 with rheumatic heart disease. Tiredness, night sweats, and pallor occurred in March, 1954. He was admitted to the Radcliffe Infirmary on April 5. Examination revealed haemorrhages in both fundi, scattered petechiae over the neck and shoulders, and gross rheumatic valvular disease. His teeth were excellent, but he habitually ground them at night. Blood cultures yielded *Str. viridans*. He was treated with penicillin (2 million units four-hourly) and streptomycin (1 g. b.d.). The temperature slowly subsided and he was discharged after three months. He remained well, but with gross limitation of his exercise tolerance, and showed occasional splinter haemorrhages.

First Relapse.—In March, 1955, 11 months after his initial attack, he was readmitted and blood cultures again yielded *Str. viridans*, sensitive to penicillin. His teeth were still excellent, also radiologically. He was treated with a prolonged course of penicillin and has remained well since.

He was readmitted in February, 1956, for dental clearance under penicillin cover (2 million units four-hourly). Blood cultures taken before extraction were negative. The teeth were extracted in two sessions, and a heavy growth of *Str. viridans* was obtained from each of eight teeth selected at random. Blood cultures taken five minutes after the second dental extraction yielded a heavy growth of *Str. viridans* in spite of a high penicillin concentration in the blood. Penicillin was continued for a further 10 days, and he has had no relapse since.

Case 5

A band knife cutter, born 1921, had severe tonsillitis in 1947, but no known history of rheumatic fever. He was first admitted to the Radcliffe Infirmary in September, 1954. He presented with general malaise, night sweats, pyrexia, and splenomegaly; his teeth were noted to be carious. Aortic incompetence was found, and, although one blood culture did not yield a positive growth of *Str. viridans*, there was strong clinical evidence of S.B.E. The diagnosis

was uncertain at the time, although it became obvious in retrospect. He was treated with penicillin, 5 million units daily for five days, with a remission of his temperature, and he was discharged on November 30 to his home. He remained afebrile for about one week, after which there was a return of the former symptoms.

First Relapse.—He was readmitted on February 4, 1955, with pyrexia, malaise, and multiple embolic phenomena. S.B.E. was clinically certain, and in spite of negative blood cultures he was treated from November 24, 1954, with 4 million units of penicillin daily. The fever subsided immediately and the embolic phenomena gradually disappeared; the penicillin was discontinued on April 19, 1955.

Second Relapse.—On April 29, 1955, 10 days after the discontinuance of the penicillin, his fever relapsed, and he was put on a further course of large doses of penicillin for five days. During this period the worst carious teeth were extracted. He remained well until the third relapse.

Third Relapse.—On May 27, 1955, 14 days after discontinuance of penicillin, he relapsed again. On this occasion blood cultures yielded a heavy growth of *Str. viridans*. His teeth were again reviewed, and he was found to have gross osteitis and apical infection. He was put on to large doses of penicillin and a complete dental clearance was carried out. He remained on penicillin for a further 10 weeks and was discharged asymptomatic on August 15. He has remained well since.

Discussion

Does S.B.E. due to *Str. viridans* Ever Occur in an Edentulous Patient?

We cannot answer this question, but so far as we are aware there is no specific reference in any published paper to an edentulous patient with this disease, nor has inquiry of many physicians of great experience yielded a confidently affirmative reply that they have met with such a patient. It is perhaps relevant to note that in clinical experience an apparently edentulous patient may not infrequently be shown by radiological examination to retain buried roots and dental remnants. This seems to us to be a fundamental point worthy of further detailed examination in an adequate series of patients.

Cates and Christie (1951) note that of a representative group of 215 patients in whom a focus of infection was sought none was found in 82 (38%), but no record is available of whether these patients were edentulous (personal communication). They comment that in 103 (48%) "there were signs of dental sepsis or tooth extraction," and that in their view dental sepsis is even more common than as reported in either the M.R.C. series (422 cases) or the American series (457 cases) reported by Anderson and Keefer (1948). There is, however, no actual record of the dental state of the individual patients in either of these two series.

Dental Extractions as the *Causa Causans* of the Initial Attack of S.B.E.

Okell and Elliott (1935) demonstrated beyond any doubt the significance of dental extractions in the production of a bacteraemia due to *Str. viridans*, and Elliott (1939) showed that the rocking movements employed before dental extraction, and even the minor movements of a tooth in the course of mastication in patients with pyorrhoea, were sufficient to produce a transient bacteraemia. This was 20 years ago, and yet we question whether this fundamental observation has ever received the attention it deserves in spite of the fact that since about 1937 it has been freely recognized that *Str. viridans* was sensitive to (a) the very considerable range of sulphonamides which are efficient in subduing a transient or persistent bacteraemia associated with dental extraction or dental sepsis, but relatively ineffective in reaching the organism deep in the vegetations which developed in a diseased heart; and (b) the ever-increasing and more efficient range of antibiotics which have been shown to be capable of not only subduing the bacteraemia but also

penetrating into these vegetations and destroying the organism, so that an apparent cure of S.B.E. can be achieved in a high proportion of these desperately sick patients.

According to Gilchrist and Matthew (1948-9) "oral sepsis is a well-worn theme, but in subacute bacterial endocarditis the evidence for incrimination of the teeth is overwhelming." In a varying proportion of cases in all published series a dental extraction has been specifically identified as the *causa causans* (Rushton, 1930; Abrahamson, 1931; Okell and Elliott, 1935; Feldman and Trace, 1938; Ward *et al.*, 1946; Anderson and Keefer, 1948; Cates and Christie, 1951). That such cases should occur in *specific relation to dental extractions* is clearly a reflection upon both medical and dental practitioners, in that these patients are in most cases known to have acquired or congenital cardiac lesions, and that for such patients dental extractions are significantly hazardous. There is no logical or general uniformity of practice in the provision of "cover" for dental extractions, and in general it can be said that the provision of such "cover" is casual and inconsequent, and is frequently ignored. We refer to this later.

Relationship of Relapse to the Dental State

Even with efficient antibiotic therapy and the obviously complete recovery from the disease in a high proportion of patients, the relapse rate within a few months or years of apparent cure is disturbingly high (18% of 354 patients (Cates and Christie, 1951) and 25% of our present small series). Several factors can influence the relapse rate, such as failure to treat adequately, failure of the therapeutic agent to penetrate to the organism established in cardiac vegetations, or development of resistance by the organism. *We believe that failure to eliminate the primary dental focus from which the organism derives is the major cause of relapse.*

Five relapses occurred in the group of patients with S.B.E. due to *Str. viridans*; each of these patients retained teeth. It is also clear that it was the retention of teeth in these patients which accounted for the relapses.

So long as teeth remain in a jaw there is ever present an ideal mechanism for the infliction of minor trauma upon the alveolar bone or mucous membranes. Mastication, teeth-grinding, and particularly the major trauma associated with extraction of teeth will inevitably expose the patient to recurrent bacteraemic showers. We believe that in recognition of the high relapse rate, the complete removal of all teeth should be carried out. If the gravity of the initial disease be accepted a relapse may be even more disastrous. Is it justifiable to evade the responsibility of advising such a patient to have all the teeth extracted under antibiotic cover because on clinical and radiological evidence they appear to be "good"?

The removal of all "bad" teeth and a thorough toilet of the mouth is usual in the course of the active treatment of S.B.E. as soon as the fever and major clinical manifestations of the disease have disappeared, and while the patient is under full antibiotic therapy.

In four of our relapsed patients (Cases 1, 2, 3, and 4) every possible care had been taken to ensure that all "bad" or questionable teeth had been removed, and that only "good" teeth were retained. Nevertheless one relapse occurred in Cases 1, 2, and 4, and two relapses in Case 3. There had been no dental extractions to account for the relapses.

Dental Extractions and Antibiotic Cover

Dental extractions are followed by a bacteraemia due to *Str. viridans* (Rushton, 1930; Okell and Elliott, 1935; Elliott, 1939) which is more severe and prolonged when the dental sepsis is gross, but is dealt with by the normal defence mechanisms of the body in most patients undergoing dental extractions. Secondary manifestations of this episodic bacteraemia may be apparent in any of the cavities lined by serous or synovial membranes, or as metastatic lesions in any organ of the body, and it is a general and

desirable practice to advise all patients with acquired or congenital cardiac lesions to consult their dentist regularly, and in the event of a dental extraction to ensure that this is carried out under antibiotic "cover."

The diagnostic criteria employed to determine whether dental sepsis is significant are (1) the clinical assessment by an experienced dentist, and (2) the radiological evidence of the presence of a root abscess, of alveolar destruction, or of periostitis. When such criteria are unfavourable the "bad" teeth are removed under antibiotic "cover," if favourable the teeth are judged to be "good." We question whether such criteria are adequate, whether "good" teeth do not leave an ever-present source of danger in a patient who under no circumstances can afford the risk of relapse. How long do teeth remain "good"?

The details of the "cover" seem to have received but scanty attention. What should be considered an adequate prophylactic "cover"?

The American Heart Association recommend that 30 minutes before an extraction 600,000 units of procaine penicillin oil containing 2% aluminium monostearate plus 600,000 units of soluble penicillin should be given by intramuscular injection, but we question whether this is entirely sufficient to provide adequate protection for an invasive process which may continue from the site of trauma for a period of several days. At most this could be expected to give cover for 24 to 48 hours. Under full cover with soluble penicillin (8-12 mega units a day, given four- to six-hourly) three of our patients yielded a *positive blood culture* following dental extractions, although this bacteraemia was immediately controlled. Under similar cover six of our patients yielded a very heavy growth of *Str. viridans* within 12 hours, from extracted teeth, both "bad" and "good." Five of these patients (including Cases 1, 2, and 3) had "good" teeth as judged by clinical and radiological examination, but all three had agreed to a complete dental clearance.

It is therefore abundantly clear that the "cover" did not prevent a bacteraemic shower, although this was immediately controlled, and that this "cover" by the systemic route does not penetrate into the tooth socket and mucous membrane of the mouth. In Case 4 we attempted to eliminate the possibility of contamination by the buccal flora with penicillin lozenges for 24 hours before dental extraction, but this failed to eradicate the *Str. viridans* and the lozenges were discontinued on account of the danger of producing a radical change in the buccal flora.

It has been our practice freely to admit to hospital for dental extraction any patient with an acquired or congenital heart lesion to ensure that the hazard to which they must be exposed shall be minimal.

The control of the incidental antibiotic therapy has been in our hands, and our practice is: (a) for the removal of one or two teeth or stumps, 1 mega unit of penicillin 30 minutes before operation, followed by 1 mega unit the same night, and repeated the morning and evening of the subsequent day. (b) For more extensive extractions or a "clean sweep" the same practice is followed, but is maintained for not less than three days post-operatively, and, when a patient is under treatment for S.B.E. or has had a previous attack of S.B.E., to increase the daily dosage to 1 mega unit every four to six hours for at least five post-operative days.

Under this regime it has been striking how satisfactorily and rapidly even extensively traumatized gum margins will heal.

Summary

In a series of 43 patients admitted to hospital with subacute bacterial endocarditis (S.B.E.) due to *Streptococcus viridans* no patient was edentulous. The diagnosis was confirmed by a positive blood culture in 25 patients (58%); in 18 patients (42%) the diagnosis rested on the clinical picture, but was confirmed by post-

mortem examination in five cases. In seven patients (16%) the initial attack of S.B.E. was specifically related to dental extractions 10-42 days before admission to hospital. Relapses occurred only in five patients who retained some teeth after the initial attack of S.B.E. had been subdued. All five patients had positive blood cultures. In four patients such teeth as they retained had been judged as "good" after clinical and radiological examination, such teeth as were considered "bad" having been removed under antibiotic cover during the initial attack of S.B.E. In the fifth patient dental extractions were not carried out until the second relapse.

Antibiotic "cover" for dental extractions in patients in the course of active therapy did not prevent a bacteraemic shower, although this was immediately controlled, nor was this "cover" effective in sterilizing the tooth sockets or mucous membranes of the mouth.

We raise the question of whether failure to prevent a significantly high relapse rate has been due not to ignorance of fundamental facts but to the uncritical acceptance of the view that the organism concerned and the antibiotic to be used will determine the final success or failure of treatment.

Our small series of cases can be regarded as representative of the cases of S.B.E. admitted to any large general hospital. In S.B.E. due to *Str. viridans* the evidence overwhelmingly incriminates the teeth as the all-important source of infection. So long as teeth or fragments of teeth remain in the jaws a formidable, but preventable, hazard is ever present in the lives of these patients, and we postulate that in all patients who develop S.B.E. due to *Str. viridans* scrupulous complete dental clearance should be carried out as an essential item of the therapeutic attack upon the disease.

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REFERENCES

- Abrahamson, L. (1931). *British Medical Journal*, 2, 8.
 Anderson, D. G., and Keefer, C. S. (1948). *The Therapeutic Value of Penicillin*. Ann Arbor, Michigan.
 Burn, P. A., and Cook, E. T. (1954). *Ann. intern. Med.*, 41, 487.
 Cates, J. E., and Christie, R. V. (1951). *Quart. J. Med.*, 20, 93.
 Elliott, S. D. (1939). *Proc. roy. Soc. Med.*, 32, 747.
 Feldman, L., and Trace, I. M. (1938). *Ann. intern. Med.*, 11, 2124.
 Gilchrist, A. R., and Matthew, H. (1948-9). *Trans. med.-chir. Soc. Edinb.*, 128, 25.
 Lichtman, S. S. (1943). *Ann. intern. Med.*, 19, 787.
 Okell, C. C., and Elliott, S. D. (1935). *Lancet*, 2, 869.
 Rushton, M. A. (1930). *Guy's Hosp. Rep.*, 80, 39.
 Ward, G. E. S., Meanock, R. I., Selbie, F. R., and Simon, R. D. (1946). *British Medical Journal*, 1, 383.

"This is a new and important development in hospital dentistry," said the Minister of Health, Mr. R. H. TURTON, at the opening of a new dental unit at Sunderland General Hospital on December 14. The unit, consisting of an out-patient department and a laboratory, forms the largest part of the building programme for a pilot scheme for providing a complete hospital dental service for the Sunderland and Cherry Knowle hospital management committee groups. The intention is to provide complete dental treatment for long-stay patients, emergency treatment for short-stay patients and such other dental treatment as is necessary as part of their medical care, specialist diagnosis and treatment for out-patients, routine dental treatment for patients with open tuberculosis, and routine dental treatment for hospital staff. The full cost of the buildings is estimated at about £20,000.