a strongly positive complement-fixation reaction for typhoid, but his Widal reaction was negative from the start, and remained so for some weeks after. The three natives who persistently showed negative blood reactions and negative findings in both urine and stools were discharged from the isolation hospital after four weeks and returned to the dairy. It was thought that one might rely, if not on their natural immunity, then on the immunity acquired from the Besredka prophylactic treatment. This left five boys, one of them the urinary carrier, and the four others giving positive complement-fixation but negative Widal reactions, and negative findings in stools and urine. After the urine and stools had been examined five times, and it had been noticed that during these weeks their complement-fixation reactions had become weaker and weaker, it was considered that it was safe for them to return to work, and they were allowed to resume work at the dairy. The only one then left over was the urinary carrier; his blood gave a persistently positive complement-fixation reaction, and his urine every week showed typhoid bacilli. It was not to be expected that Besredka treatment could have had any effect on this condition. Treatment was therefore instituted with ordinary typhoid vaccine given under the skin, and large doses of urotropine, but after many weeks, although the typhoid bacilli in the urine showed signs of diminishing in number, they were still to be found. It took four months before they disappeared entirely and the boy could be discharged.

This constitutes the history of the outbreak. The moral of the story is that if the first case of typhoid at the dairy had been recognized as such—whether it was the imaginary stray young native or a member of the regular staff—much suffering and unhappiness would have been prevented. Our dairy by-laws place the obligation on dairy owners to notify the health department of any case of infectious or contagious disease occurring on their premises; it is evident that this is an unsatisfactory arrangement. The onus thus rests on the health department to prove that the dairy owner was aware of the fact that a disease amongst the personnel was infectious or contagious, and that will always be difficult. The whole responsibility should be transferred to the health department, provided that at the same time it receives the powers and facilities to examine all employees upon their being engaged by the dairy, and at regular short intervals thereafter. This examination should include a blood test for typhoid fever, and the foregoing record amply shows that for such work the complement-fixation test is superior to the Widal. If the Widal test had had to be relied on the urinary carrier would not have been detected with the same rapidity, and where two other natives at the time gave a positive Widal reaction the investigation would probably have stopped at that, and the chief culprit would have carried on his work.

It remains to give a brief description of the principles and technique of the complement-fixation test for typhoid. We have for many years insisted on the necessity of abandoning agglutination tests for typhoid in South Africa, as we have proved^{1,2} that the majority of cases cannot be diagnosed by this method. We have also obtained some evidence from animal experiments that this failure to develop agglutinins which we have demonstrated in South Africa is at least partly due to the prevailing excessive sunlight.² The complement-fixation test is positive in over 90 per cent. of cases, is as a rule not positive in persons who have been vaccinated against typhoid but are suffering from some other infectious disease, becomes positive at an early stage of the disease, and is much more helpful in the detection of carriers. Its only drawback is that it requires perfect technique and much more care than agglutination tests. Our present procedure is as follows.

The antigen is prepared by cultivating typhoid bacilli (there is no difference between typhoid strains for this purpose) on ordinary agar slopes for twelve to twenty-four hours and washing them off with saline solution, using about 4 c.cm. per slope. The suspension is heated in the water-bath at 57° for one hour, stored in the ice-chest for forty-eight hours, and filtered through a Berkefeld candle. The filtration must be slow and produce only a few cubic centimetres per hour. Suction may not exceed 16 mm.

of mercury. It is only by strictly observing these rules that a potent and constant antigen can be prepared. Each filtrate must be titrated afresh with each new haemolytic system to be used. This is done by employing diminishing quantities of filtrate, from 0.5 c.cm. to 0.05 c.cm., with 0.5 c.cm. of guinea-pig complement diluted ten times, making up to 1.5 c.cm. with saline solution, incubating for one hour, and then adding 1 c.cm. of haemolytic system. A control tube is required which contains only saline and complement; to this tube also 1 c.cm. of haemolytic system is added. The time is noted when this control tube has cleared, and ten minutes after this the tubes are again inspected. The tube which then shows complete haemolysis and contains the largest quantity of antigen indicates the quantity of antigen to be used in the test proper. The antigen is then diluted so that 0.5 c.cm. contains the suitable dose. As a rule we find that 0.3 c.cm. represents the quantity of antigen to be used.

If the filtrate is to be kept for more than a few days it is advisable to add 0.25 per cent. carbolic acid to the filtrate previous to titration. A good filtrate has considerable keeping qualities, and although we do not use it after it is fourteen days old we have found that it will keep its antigenic properties for at least thirty days.

We prepare haemolytic system by determining the minimum quantity of a sheep's cells-rabbit serum which, in the presence of 0.5 c.cm. of ten times diluted guinea-pig serum, will completely dissolve in two hours 0.5 c.cm. of a 5 per cent. suspension of sheep's cells, the total volume in each tube being made up to 2.5 c.cm. Four times this minimum quantity of serum is then used in the test proper. The serum is suitably diluted for this purpose with saline, and equal quantities of diluted serum and 5 per cent. sheep's cells suspension are mixed.

The patient's serum is inactivated, and 0.1 c.cm. is put up with 0.5 c.cm. antigen, 0.5 c.cm. complement 1 in 10, and 0.4 c.cm. saline. This is incubated for one hour, and then 1 c.cm. of haemolytic system is added. At the back of every patient's tube is a serum control, containing 0.2 c.cm. of the patient's serum, no antigen, 0.5 c.cm. complement, and 0.8 c.cm. saline. In addition an antigen control, containing 0.5 c.cm. antigen, 0.5 c.cm. complement, and 0.5 c.cm. saline, is put up.

Readings are taken when the antigen control has cleared, and then for every individual patient when his serum control tube has similarly cleared. Serums from the later stages of the disease show complete and lasting fixation; those from earlier stages show partial fixation, which is often not permanent. It is useful to include a known positive serum in the series, either from a previously tested typhoid patient or from an injected rabbit.

Human serum which produces complete fixation under these conditions will retain that quality when kept at room temperature for a considerable time.

By following this technique we have been able to make a positive serological diagnosis of typhoid fever in cases where the presence of the disease was established beyond doubt by positive blood cultures, but where Widal's agglutination test was persistently negative.

We have found the method particularly valuable in the detection of carriers. Here we also often found a negative Widal and a positive complement-fixation reaction, as illustrated by the case recorded in this paper.

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THE ROLE OF DEAD AND INFECTED TEETH IN AUTOGENOUS INFECTIONS.

BY

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WHILE the literature teems with works on the evil effects of pyorrhoea alveolaris—and there can be no doubt as to its baneful effect on health—there is another dental condition which is but now receiving the attention it deserves—namely, the tooth which has died before or after filling, and which has subsequently become infected. That death may occur without pain makes the condition all the more unsuspected.

The bacteria in pyorrhoea pass into the mouth, where the majority are ingested by the salivary corpuscles; the rest pass on to the stomach and are destroyed, if the gastric juice is normal. In the case of the filled tooth the bacteria cannot regain the mouth by way of the original decay, but must enter the bone, from which they or their toxins may be disseminated by the blood stream to all parts of the body, when those tissues which are "below par" fall a prey to them. By the irony of fate it is those people who take care of their teeth, having no untreated caries, who

are often the sufferers. The following references to clinical cases will illustrate these points.

Case 1.—A man, aged 27, had thrombosis of the posterior tibial venae comites. Five years later he developed the first of a series of eight or nine attacks of the lymphangitis which precedes elephantiasis. The whole course of the lymphatics accompanying the saphenous vein and the superficial inguinal glands became inflamed for a period of about a week. Though there were no obvious caries one tooth was bluish, and x-ray examination showed an area of rarefaction at its root. On opening the pulp pus welled up, and the tooth was removed. Another attack, differing somewhat from the others, occurred a year later, and two days after its commencement a tooth crowned by the old method without root treatment became slightly tender for a day. An attack of fibromyositis occurred; this had been frequent before the first extraction, but had not been experienced since. X rays showed no abnormality. The tooth was removed, however, and a granuloma was found at the root which yielded *Streptococcus salivarius* and diphtheroid bacilli. The pulp cavity was found to be full of greenish pus from which was obtained a scanty growth of *Staphylococcus albus*. There has been no return of the fibromyositis or lymphangitis in the nine months which have elapsed, and the oedema has been less marked.¹

Case 2.—A man with arthritis of the left knee was treated by rest for two years, followed by manipulation, which caused a "flare-up." For two more years walking was only possible with the aid of a stick. Attention was accidentally drawn to a tooth which had been stopped, and an x-ray examination showed apical rarefaction of this and of an adjacent tooth. These teeth were extracted, and a course of antistreptococcal vaccine instituted. The joint gave no further trouble for five years, when two more teeth showed apical rarefaction. After their removal the joint cleared up at once.

Case 3.—A man, aged 62, had been afflicted with osteitis deformans for twenty years; the other tibia, right humerus, clavicle, and head became involved later. The teeth were very foul, and much pyorrhoea alveolaris was present; this condition must have existed for many years, and probably dated from the time of onset of the osteitis. All the septic teeth were extracted, large masses of granulation tissue were removed, and portions of bone from the upper alveolus, including a local enlargement, in order to lessen the deformity. The skin over the tibiae, which had become oedematous and inflamed, speedily cleared up, and the bone lost its tenderness.²

Case 4.—A married woman, aged 35, came for dental treatment seven years ago, at which time she was suffering from tenosynovitis of the wrist. There were three dead teeth, and the gums were badly infected, streptococci being recovered from them. On removal of the dead teeth, six months later, the synovitis disappeared, though streptococci were still present in the gums.³

Case 5.—A woman, aged 65, had fibromyositis of the neck muscles and cervical spondylitis deformans, leading to much limitation of movement. The removal of two dead teeth caused considerable improvement, but she refused to have other dead ones out.³

Case 6.—A man, aged 45, had for two years suffered from sciatica, which failed to respond to vaccines or sunlight. He had two dead teeth, which were extracted, one yielding a pure culture of *Streptococcus longus*. The socket did not heal readily, and whenever pus accumulated in it he had an exacerbation of his sciatica, which was promptly relieved by opening up the socket.³

Case 7.—A young woman with dyspepsia and anaemia had fleeting joint pains accompanied by slight swelling. The appendix was removed and found to have an abscess at its tip, but her condition did not improve. Two years later she began to have an evening rise of temperature, associated with severe nasal catarrh. A radiogram showed a rarefied area at the tip of a filled dead tooth, which was extracted. After three rigors, during each of which the socket became painful and inflamed, her health improved greatly, and the "rheumatic" pains and pyrexia disappeared.⁴

Case 8.—A man, aged 45, suffered from myocarditis and emphysema; removal of eleven grossly septic teeth was followed by great improvement in health. Later six more were removed, and now, nine months after the extractions, he is able to walk two to three miles without discomfort, whereas formerly great discomfort followed after a mile. (Dr. Keith Tucker, Margate.)

Case 9.—Eight years ago a man began to have pain and swelling of the right hand, threatening later to incapacitate him from work. One tooth was removed, causing an exacerbation. X rays showed many apical abscesses. The infected teeth were removed, with the result that the condition cleared up entirely. (Mr. Fowler Ward, Ipswich.)

Signs of Infection.

The determination as to whether a tooth is dead is usually not difficult, as the tooth generally takes on a bluish-black colour. The diagnosis of infection, on the other hand, may be very difficult; the view that the live tooth may be infected further complicates matters. The most important clinical sign is tenderness on clinching the jaws and on percussion. This tenderness is well recognized by the patient, and is quite unlike that which results from "cold." The presence of tenderness in a dead tooth should

be regarded as sufficient reason for extraction; a radiograph sometimes shows nothing abnormal. In some cases the tongue will be found furred opposite an infected tooth.

A radiogram is invaluable in most cases. The first evidence of infection is a thickening of the periodontal membrane, visible in the film as an increase in the dark line round the tooth. As the condition proceeds one of two things happens—either (a) the pus may escape by passing between the tooth and the bone, causing a further increase in thickness of this line, usually on one side; or (b) if exit is impossible, a crescentic area of rarefaction appears, the tip of the root forming the concavity of the crescent. In either of these cases, but especially in the latter, actual absorption of the root may occur if the organisms are very numerous or virulent; in other cases the root appears bulbous owing to deposit of cementum, the organisms being of lower virulence.

It is of the utmost importance to examine the whole length of the jaws when a dental focus of infection is suspected, as only in this way can remnants, cysts, and abscesses be revealed. One cause of persistence of symptoms is residual infection of the socket; in such cases the socket outline may be apparent years after clinical healing has occurred. It must be clearly understood that a radiogram only shows involvement of the alveolus with any certainty; a tooth may be dead and infected and show nothing on the skiagram, as in Case 1.

Causes of Death and Infection of Teeth.³

1. Failure to remove all carious dentine at the time of filling allows caries to go on until finally the pulp is reached and death of the tooth occurs.

2. Synthetic fillings such as are used for front teeth may kill the pulp, owing to their phosphoric acid content, unless they have a cement lining.

3. Paraform and other disinfectant dressings may kill the pulp, which subsequently may become infected by the blood stream.

4. "Capping" the pulp is a frequent cause of ultimate death of a tooth.

5. Trauma to a tooth or the alveolus in the vicinity may interfere with the circulation of the pulp.

6. Operations on the antrum may implicate the superior dental nerves and vessels.

7. Local anaesthetics containing adrenaline render the tooth painless, so giving no warning of pulp exposure. Pain in this case develops immediately the effect of the drug has worn off.

8. Large metal fillings near the pulp may cause irritation and death of teeth, but these again are nearly always painful from the time of filling.

A devitalized tooth, in which the pulp has been replaced by gutta-percha, may function perfectly for many years. It is advisable to have it x-rayed at intervals for apical rarefaction; if this is present the tooth must be redressed until sterile and then refilled, or, better, extracted on the first evidence of infection.

Bacteriology.³

Examination for the micro-organism present at the apex of the tooth is difficult owing to the flooding of the socket with saliva due to the partial vacuum created by extraction. Before this is done the gum should be painted with absolute alcohol; iodine is contraindicated, since traces tend to adhere to the tooth and inhibit growth in culture. Immediate culture of the blood as it wells up is the surest way of discovering the bacteria responsible. The commonest organism found is the *Streptococcus salivarius*, but in acute abscess *Streptococcus haemolyticus* is frequent. If immediate culture is impossible, it is futile to place the tooth loose in a test tube, since contamination is inevitable. It should be embedded in the stopper of a wide-mouthed vessel. To examine the pulp the tooth is washed with alcohol, dried, then fractured, and its contents cultured.

Pathology.

In an infected tooth the necrotic nervous and vascular tissue forms an ideal pabulum for micro-organisms. In untreated caries the toxins and pus may pass into the mouth by way of the decay. In a filled tooth this exit is closed, so they proceed down the apical canals into the

bone, from which they may then pass into the mouth between the tooth and the bone, but in the greater number of cases the pus lies pent up in the apex, forming an abscess. Here in the sinusoids of the marrow it is free to exert a toxic influence on the whole body, as is shown by the anaemia which is so common in these cases. Apparently the relatively avascular tissues, such as fibrous tissue and synovial membrane, are the usual sites of election for local manifestations. The pulp chamber contains a bead of pus; any increase in the number or virulence of the bacteria causes increased pressure, which results in toxins and pus being squirted down into the bone. Natural resolution, which can occur in bone, cannot take place here, owing to the fact that the walls cannot collapse and no new tissue is available to fill the pulp cavity. Rarely, however, calcification occurs.

As with other streptococcal infections, once a patient has been attacked recurrences are very liable to occur, either from residual infection or from the subsequent infection of other teeth (Cases 1, 2, 6, and 8).

It is not yet possible to give a complete list of the diseases caused by infected teeth. Among those given are fibromyositis, arthritis, and lymphangitis. To these may be added others of a "rheumatic" nature, and possibly Dupuytren's contracture. It must not be thought that every case will clear up on removal of the infective focus; the condition may only be diagnosed when the disease is advanced, and then even its arrest may be problematical.

Sir William Willcox⁵ states that the disabilities responsible for one-sixth of the days lost in the industrial world come under the category "rheumatic." In 100 consecutive cases of this nature he found the teeth to be responsible in 72. He writes:

"The eradication of the primary focus in early cases of arthritis has, in my experience, been followed by cessation of active inflammatory changes of the infected joints. Where bony changes have occurred, or other destruction of the joint, it cannot be expected that removal of the focus of infection will rectify these. When active inflammation of the joint has ceased treatment for restoration of movement will be followed by real improvement."

Much has been written on the areas of rarefaction seen in skiagrams of the teeth, and Weston Price has brought forward evidence that there are granulomata shutting off the toxins and bacteria from the general circulation. This may be so; I have heard of a man with six such areas who is in perfect health. Surely, however, there must be a grave potential danger of the resistance of this granulation tissue becoming lowered from time to time and allowing toxins and bacteria to escape.

Important advances might be made if all suspicious teeth found at necropsies of patients suffering from the diseases mentioned in this article, and others of obscure origin, were extracted and cultures made of their contents.

Summary.

1. A tooth may die painlessly before or after filling; infection readily occurs, and such a tooth may be the focus of autogenous infection.
2. The condition may be very much more serious than pyorrhoea alveolaris.
3. A positive radiogram indicates apical infection, but a negative one does not signify absence of disease of the pulp.
4. Continuance of symptoms after extraction, or in edentulous jaws, may be due to tooth remnants such as fragments of roots, abscesses, and cysts.
5. Special precautions are necessary at the time of extraction if bacteriological investigation is attempted.
6. The commonest diseases caused by dead and infected teeth are those termed rheumatic—namely, fibromyositis, synovitis, and arthritis.

It is a pleasure to acknowledge the help I have received from a symposium on this subject held at the Post-Graduate Hostel. Especially would I like to mention the contributions of Mr. Doubleday, Mr. Sprawson, and Dr. Human on the dental aspect, and of Dr. Canti on the bacteriological investigations.

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FRACTURE OF THE NAVICULAR.

BY

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So many patients are seen suffering from a persistent painful disability of the wrist following an unrecognized fracture of the navicular (Fig. 1) that it is important that the symptoms of this lesion be more widely known. These symptoms are characteristic, the treatment is simple, and the results extremely satisfactory if the patient is dealt with soon after the fracture occurs.

FIG. 1.—Radiogram of the carpus taken two years after the injury, showing, A, the two ununited fragments of the navicular. There are definite osteoarthritic changes present.

No attempt is made in this paper to consider every aspect of the subject—this has been done already by Todd.¹ My purpose is rather to accentuate the clinical features of the condition which are of diagnostic importance.

Fracture of the scaphoid bone (os naviculare) is by no means an uncommon lesion. In the past two years I have seen forty-six examples of the condition, but unfortunately the majority were late cases. These patients come to hospital complaining of persistent pain and stiffness in the wrist and inability to carry out their work efficiently. The history usually is that months or years previously the patient had fallen and injured the wrist. Either it was treated as a simple sprain or he paid no attention to it at the time, but the wrist never fully recovered, and was a continual source of trouble. If patients are to be spared a disability such as this it is essential that the fracture from which they suffer be diagnosed at once, and as the symptoms are so definite and characteristic, provided that this lesion be kept in mind, there should be little reason for its escaping notice.

The mechanism of injury is usually a fall on the outstretched hand, very similar to that which produces a

FIG. 2.—Case of recent fracture of the navicular, showing the dorso-radial swelling. Note that the swelling does not extend to the ulnar side of the wrist.

Colles's fracture. Less commonly the fracture results from the back-fire of a motor car. On examination of the wrist no gross deformity is noticed, but a very definite oedematous swelling is seen, situated on its dorso-radial aspect and extending into the anatomical snuff-box, with the result that the tendons bounding it no longer stand out prominently on extension of the thumb (Figs. 2 and 3). The swelling does not extend to the ulnar side of the wrist. Palpation reveals normal bony points, but there are two points of marked tenderness—one over the dorsum of the navicular, immediately below the line of the wrist-joint, and the other in the anatomical snuff-box, immediately below the styloid process of the radius. Pain at the site