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.

REFERENCES.

- 1 A. P. Bertwistle: *British Medical Journal*, September 19th, 1926, p. 363.
- 2 R. Lawford Knaggs: *Inflammatory and Toxic Diseases of Bone*, p. 300.
- 3 The Filled Dead Tooth as a Source of Streptococcal Infection, *Lancet*, November 6th, 1926, p. 953.
- 4 J. Nesbitt: *Journ. Royal Naval Medical Service*, August, 1927, p. 142; *Clin. Journ.*, 1927, p. 504.
- 5 Sir William Willcox: *Practitioner*, August, 1927.

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

of fracture may also be elicited by Vaughan's knuckle percussion test, which is described by Todd.

"The metacarpal joints are all flexed to a right angle, or if possible the patient is made to clench the fist; he is then told to keep his eyes shut, whilst the surgeon taps the knuckles smartly, one after another. If the fracture of the scaphoid is present sharp pain will be elicited when the head of the second metacarpal is struck, but not when any of the others is struck."

I have not found this sign of great value in diagnosis. In some cases it is not present, and in others it would appear to be present although no fracture is seen on x-ray examination.

The movements of the wrist, both in flexion and extension, are markedly limited, and the latter movement causes pain referred to the dorsal aspect of the navicular. Pain on flexion is rarely localized to a definite point.

The clinical features may be summarized as follows:

1. Swelling on the dorso-radial aspect of the wrist.
2. Obliteration of the tendons bounding the anatomical snuff-box.
3. Tenderness over the dorsum of the navicular.
4. Tenderness at the base of the snuff-box.
5. Limitation of flexion and extension of the wrist, the latter causing pain over the site of fracture.

While these symptoms are always prominent in recent cases they persist in untreated cases for long periods. I have recently seen a patient who had had an untreated fracture of the navicular for two years, and at the end of that period, when he came to hospital, he still showed all the characteristic signs which have been described.

I would stress the fact that diagnosis can be established without an x-ray examination, but, when available, plates should be taken to confirm the diagnosis. It is of the utmost importance that the skiagram be taken with the hand in marked ulnar deviation, for only in this position will all fractures of the navicular be detected.

FIG. 3.

FIG. 3.—Note the swelling in the anatomical snuff-box with masking of the extensor and abductor tendons.

FIG. 4.

FIG. 4.—Photograph taken five weeks after a fracture of the navicular, which had been treated in a plaster case. The patient was entirely free from pain, and full dorsiflexion was possible.

TREATMENT.

Immobilization is the keynote to success in the treatment of fracture of the navicular. The value of active movement in upper limb fractures is widely recognized, but the number of patients who suffer from a chronic disability following what has been erroneously diagnosed "sprained wrist," and treated by active movement, is sufficient proof that in this type of fracture, at least, it is not to be recommended. When the patient is seen

early the following treatment has given excellent results. A plaster case is applied to the forearm, wrist, and hand, the wrist being dorsiflexed and the fingers and thumb maintained in the position of normal grip. In most cases it is necessary to give an anaesthetic to enable sufficient dorsiflexion to be obtained. The parts are immobilized in this

plaster for a period of three weeks, but at the end of a fortnight the upper half of the plaster is removed to allow of massage. At the end of the third week the plaster is discarded and free movement encouraged. All the patients who have been treated in this way have obtained perfect results, both as regards movement and freedom from pain (Fig. 4).

When the patient is not seen until a month or two have elapsed immobilization has not proved of value. In some of these cases the proximal fragment has been removed by operation, but the results are not very satisfactory, and equally good results have been obtained by a prolonged course of massage, together with exercises for the wrist and hot and cold douching of the part. In my experience, however, the vast majority of these patients are left with a chronic disability, pain and stiffness in the wrist, and with a marked tendency for osteo-arthritic changes to occur (Fig. 1).

REFERENCE.

¹ *British Journal of Surgery*, vol. 9.

TWO CASES OF ACUTE YELLOW ATROPHY OF THE LIVER FOLLOWING ADMINISTRATION OF ATOPHAN.

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IN 1922 Schroeder¹ first drew attention to the toxic properties of the drug variously known as phenylcinchoninic acid, cincofen, quinophan, atophan, agotan, and phenoquin. His seventeen cases—chiefly of mild poisoning—consisted of headache, gastro-intestinal disturbances, and transient jaundice; while Worcester-Drought² a year later reported a similar case. Following a communication from Langdon-Brown³ in 1926, a dozen fatalities were reported by Evans,⁴ Glover,⁵ Willcox,⁶ and Wells.⁷

In his presidential address to the Section of Therapeutics of the Royal Society of Medicine Graham⁸ emphasized the fact that every fatal case recorded was the result of uninterrupted use of this compound for a more or less lengthy period. Individual susceptibility plays a certain

part in determining the production of poisoning, as may also previous hepatic derangement, but it is noteworthy that there is no record, except in our second case, of any untoward result of this form of therapy where the drug is given in the correct manner. By this is meant its administration for periods of not longer than four days with intervals of not less than four days, as advised by Graham. This method brings about the maximum excretion of uric acid; the extension of the drug beyond these bounds, therefore, is not only therapeutically useless, but may be positively harmful.

The following are notes of the authors' two cases:

CASE I.

Read by L. J. A. L. before the Liverpool Medical Institution on February 23rd, 1928.

E. H., aged 55, had suffered from rheumatoid arthritis for many years. In July, 1927, on the advice of a neighbour, she began taking atophan at the rate of three 7½-grain tablets daily and without intermission. During the second week of January, 1928, she developed mild jaundice, apparently without any other symptoms. The final catastrophe occurred suddenly. The jaundice became more pronounced, on January 20th coma followed delirium, and she died on the following day.

The faeces had retained their normal colour throughout, and on this account a diagnosis of "acute yellow atrophy of the liver" was made. In view of the self-administration of atophan a coroner's inquest was held and a *post-mortem* examination performed.