POST-TRAUMATIC OSTEODYSTROPHY AT JOINTS.*

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THE communication we have the privilege of making to you is concerned with a curious and obscure disturbance of bone structure, which succeeds an injury in the vicinity of a joint and manifests itself clinically by a syndrome we regard as characteristic. Though Sudeck[17] first directed attention to it as long ago as 1900, and though it has attracted a good deal of attention on the Continent, especially in Leriche's clinic,[3, 11, 12] it has escaped all but the scantiest of notice in this country. This is to be wondered at, for it is not uncommon; it is of distinct importance to the medical practitioner; it is a source of considerable anxiety to the surgeon called upon to treat it; and it forms a chapter of bone pathology that is not without its interest to the pathologist and the radiologist.

Despite many recent observations, it cannot be said that our knowledge of bone growth, structure, and maintenance is yet arranged on an orderly basis. The gross manifestations of trauma, bacterial invasion, and neoplastic activity have long been recognised and understood. Apart from these, bone has been associated in our minds with a permanency of form and a constancy of structure above all other tissues. Now, however, we are forced to regard it as a tissue of extraordinary instability and sensitivity, whose varied response to numberless influences forms one of the most engaging and diverting problems in the whole range of modern surgery. It answers, with rapidity, for example, the slightest changes in its blood supply; its composition and histological structure fluctuate with variations in parathyreoid activity; vitamins exercise a considerable influence over it; age exacts a heavy toll; and general systemic illness leaves its indelible mark on its structure.

Fortunately the problem as it affects the clinician is not so complex as the physiologist and the biochemist would have us believe, for, reduced to its simplest terms, bone can be regarded

* Read at a meeting of the Medico-Chirurgical Society of Edinburgh on 6th December 1933.

as a huge reservoir of mineral salt imprisoned within a framework of connective tissue. The mineral content is by far the most mobile of its constituents, and we know that under certain conditions it shows great and widespread depletion. In the condition under review, however, there occurs a localised depletion of calcium salts which Leriche has called "post-traumatic painful osteoporosis"—a descriptive name that focuses attention on the most characteristic features of the disease, for the pain and the loss of calcium salts are the sequel to injury. The following history is that of a typical case.

Case I.—A joiner (P. R.), aged 43 years, reported at the Out-Patient Department complaining of pain and stiffness of the right wrist. Some two weeks before, he had sustained a slight sprain while pushing a heavy safe door. It was not particularly painful at the beginning, but after a week or two, when we should have expected almost complete recovery, the joint had become swollen and tender, and its range of movement considerably restricted. When first seen, the wrist region was slightly enlarged, and the subcutaneous tissues were the site of a slight localised œdema. The skin was clammy, somewhat cyanosed, and covered with perspiration. Radiological examination at that time was negative (Fig. 1), and a diagnosis of chronic ligamentous sprain was made. The wrist was immobilised on a cock-up splint, but the symptoms were not relieved. His teeth were investigated and cared for; diathermy was applied, but the pain and stiffness persisted. Nine days after his first examination, a second radiogram was taken, and it revealed an arresting state of affairs; the carpal bones now showed a diminished density, there was slight mottling in the region of the triquetrum, and the lamellæ lacked definition. A month later these features were still more in evidence, and the lower end of the radius and the bases of the metacarpals were involved (Fig. 2). Further, the cortex of the carpal bones had become very greatly thinned out, but—and this is important—the joint spaces and the articular surfaces were relatively well preserved. The decalcification increased still further for a time, but his symptoms gradually improved. At present, the wrist is stiff and attempts at movement are painful. The spontaneous pain has disappeared, however, and his last radiograms indicate that recalcification is now proceeding.

This sequence of events is characteristic—the minor trauma, the persistence of symptoms, the progressive interference with joint function, and the pronounced radiological findings. The decalcification is undoubtedly the most striking feature, as indeed Leriche insisted, for its presence and progress are both

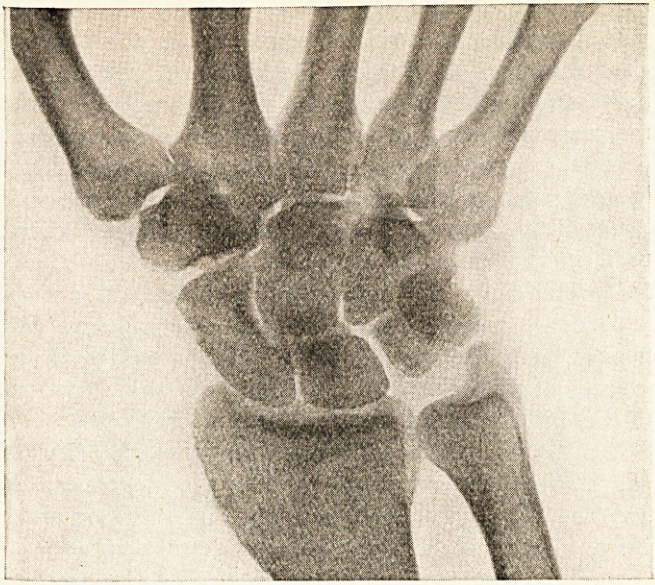

FIG. 1.—Case I. (P. R.). First X-ray of wrist, showing normal structure.

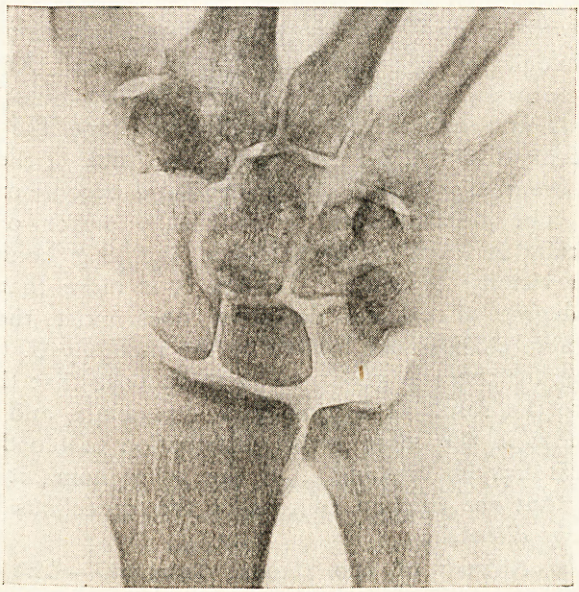

FIG. 2.—Case I. X-ray a month later: Stippled decalcification now present, with general loss of density.

easy to demonstrate, and there is a tendency, on this account, to regard it as the fundamental error and as the precursor of the other changes. It is most likely, however, that the pain and the vasomotor disturbances are equally important, and that all three are expressions of an autonomic nervous lesion. The osteoporosis can be so easily observed, however, that it is usually regarded as the diagnostic and prognostic criterion of the condition, and a good deal of attention is accordingly paid to it in the following pages.

This preliminary communication is based on a series of twenty cases observed during the past year; and on analysis, certain points emerge which demand elaboration. These are: (1) the locus of the disease, (2) the nature of the causal trauma, and (3) the explanation of the clinical phenomena.

(1) **The Locus of the Disease.**—In the majority of our cases, as also in the majority of published accounts of the condition, the polyarticular regions, such as the foot and wrist, have been affected, and the resulting joint disability has in many instances been so severe as to render the part almost completely rigid. The larger joints suffer only occasionally, but our series includes typical osteodystrophy at the knee and ankle.

In the carpal and tarsal regions, the decalcification can be seen radiologically to begin in the short bones. From this primary position, its influence extends to the bases of the metatarsals and metacarpals, and at this stage the related joints become disabled and stiff. The lower ends of the radius and tibia eventually share in the process, and become markedly porotic. The decalcification is practically never observed, radiologically at least, in the shafts of the long bones away from the vicinity of joints. Though it is more than likely that a degree of calcium depletion does exist there, the demonstrable changes are limited to the metaphysis. This is suggestive, for there is good reason to suppose that the bone effects are the result of a prolonged hyperæmia, and it may well be that in the more lavish distribution of blood to the metaphysis lies its undoing. On the other hand, it is also probable that the calcium content of the cancellous bone is more easily dissolved.

(2) **The Nature of the Causal Trauma.** — The second feature of interest is the causal trauma, and this, remarkably enough, is as often minor as major in nature. The injury

may result in a definite fracture, such as a Colles' at the wrist, and a Potts' at the ankle. Frequently, however, the osteodystrophy is the sequel to a slight injury unassociated with fracture: the trauma may then take the form of a torsional strain of the joint associated with ligamentous tearing, or of a simple crush or contusion. Occasionally, too, it follows other than forceful trauma, and we have found it occurring after burns and scalds, and after surgical operation. Examples of these are shown in the following cases.

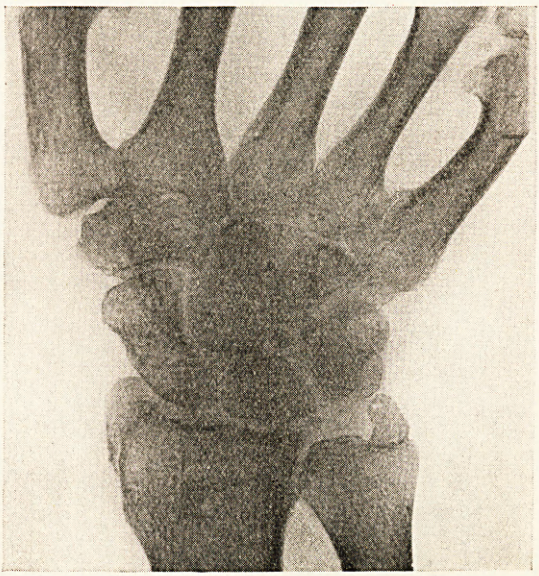

FIG. 3.—Case II. (John B.). Wrist ten weeks after slight fracture, showing commencing osteodystrophy.

CASE II.—*Osteodystrophy following fracture.*—John B., aged 38, sustained a fracture of the anterior margin of the articular surface of the radius. After the usual period and method of immobilisation, the wrist remained stiff and painful, swollen, and bathed in clammy perspiration. Despite further splinting and despite diathermy and massage, the pain and disability persisted, and, ten weeks afterwards, an X-ray examination showed well-marked punctate osteoporosis of the carpus (Fig. 3). In this case, owing to the severity of the symptoms, we sought the help of Professor Fraser, who agreed to perform a periarterial sympathectomy, following the practice of the French school. As a sequel to this, there was dramatic and immediate relief from pain.

Some improvement in function has followed, but it is yet too early to assess the final result.

Case III.—*Osteodystrophy following sprain of a joint.*—Five weeks before admission to hospital, Nurse B., aged 58, had jumped off a step and twisted her ankle. The foot was stiff and painful, and slight generalised œdema was present. The radiogram showed a diffuse and mottled osteoporosis of the tarsus, becoming well marked in the course of the following month (Fig. 4).

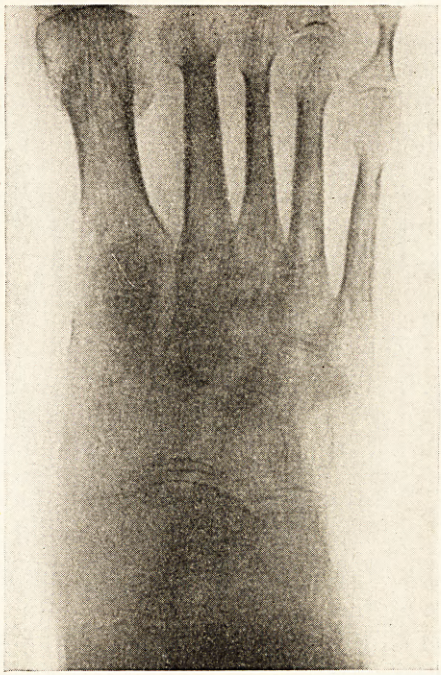

Fig. 4.—Case III. (Nurse B.). Foot nine weeks after injury to ankle-joint. Note the diffuse osteoporosis, and the attenuation of the cortex of the tarsal and metatarsal bones.

Case IV.—*Osteodystrophy following contusion of a joint.*—Robert B., aged 41, had his foot crushed between a coal-box and an iron bar. He was first examined on the day of the injury, but no bone lesion was discovered clinically or on radiological examination. For three months he attended the Out-Patient Department with persistent pain generalised over the foot, localised sweating, and a diffuse slight oedema—all of which persisted despite support by bandaging, rest in bed, radiant heat, and massage. After four months' absence, he reappeared at hospital with

the story that in the interval the foot had given continuous discomfort. It was still very painful, slightly swollen, and bluish, and the radiologist demonstrated a diffuse osteoporosis of the tarsus and metatarsus (Fig. 5).

Case V.—*Osteodystrophy following scalds.*—Mrs H., aged 51, three months before coming to the Out-Patient Department, upset a pan of boiling water over her left foot. Since then it had been continuously painful and numb. Clinically she showed a rigid flat foot, with a diffuse œdema, and her X-ray shows a typical punctate decalcification readily apparent by contrast with the healthy foot (Fig. 6).

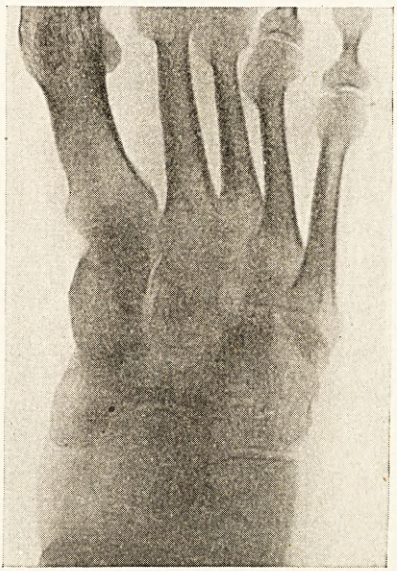

Fig. 5.—Case IV. (Robt. B.). X-ray of foot seven months after crushing accident. Diffuse osteoporosis of tarsus and metatarsus.

Case VI.—*Osteodystrophy following operation on joint.*—George B., aged 38, was thrown off a truck and landed on his out-stretched hands, as a result of which he sustained a fracture of the right wrist, and, on the left side, an anterior dislocation of the lunate. One week after his accident the displaced bone was removed by open operation, and the wrist immobilised for two weeks on a cock-up splint. At the end of this period, the wrist and hand were swollen, blue, moist, and clammy. He was suffering intense pain and this persisted despite treatment. Eight weeks after the original injury some osteoporosis was observed; after a further four weeks it was more marked (Fig. 7), and subsequently it became extreme (Fig. 8). He was so distressed that again Professor

Fraser came to our assistance and admitted him to his ward. He carried out a sympathectomy, again with a striking immediate effect.

(3) **The Clinical Phenomena.**—The clinical evidences of osteodystrophy may be said to form a characteristic syndrome, and all observers have directed attention to this fact. It is always associated with—(*a*) Severe pain, (*b*) stiffness of the

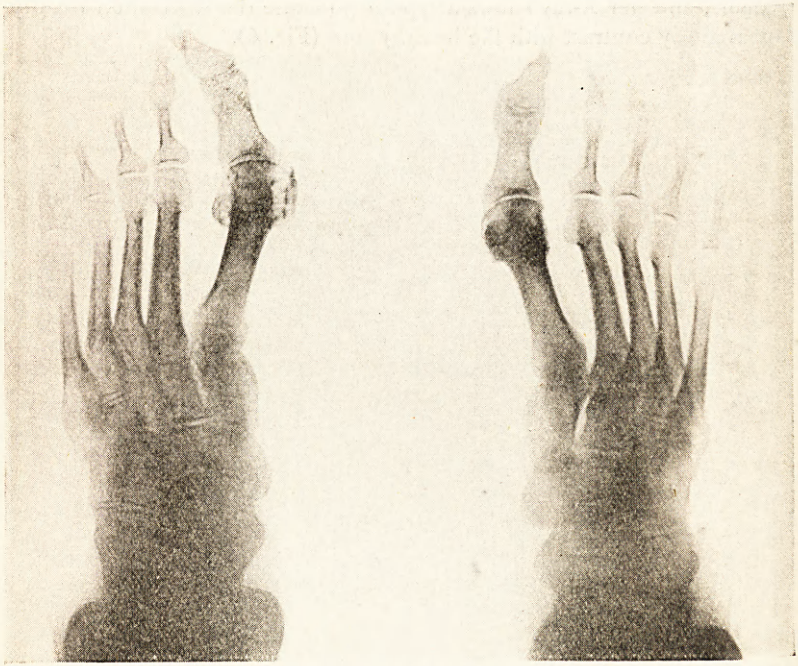

FIG. 6.—Case V. (Mrs H.). X-ray of feet, showing osteodystrophy of left tarsus and metatarsus following scalding three months previously.

affected joint, (*c*) vasomotor disturbances, and (*d*) definite X-ray pathology.

The pain is ordinarily out of all proportion to the severity of the causal lesion. It is distinctive in that it is practically never relieved by immobilisation, and only slightly by accepted physio-therapeutic means; indeed undue movement and activity seem usually to aggravate it.

The loss of function which results is also severe, and, in many cases affecting the tarsus, the patient has to take to bed. We think the stiffness at least partly due to the spasm which occurs

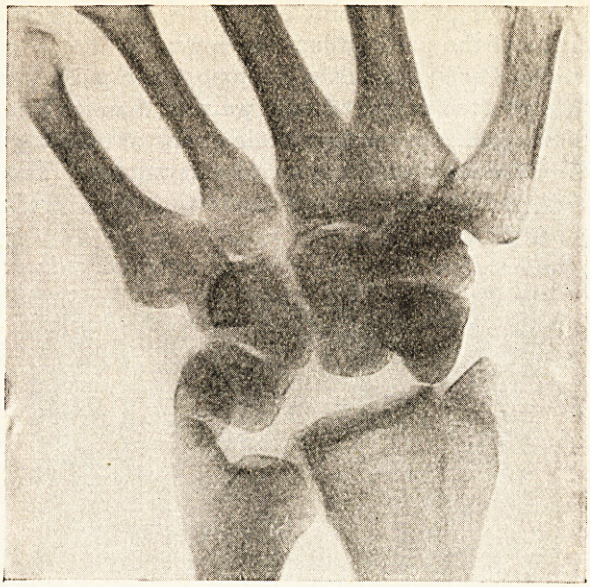

FIG. 7.—Case VI. (Geo. B.). X-ray of wrist, showing commencing osteoporosis, following operation for removal of semilunar.

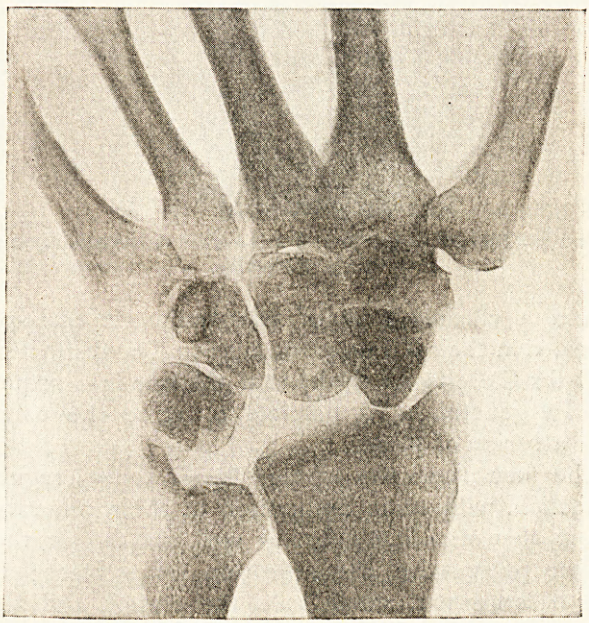

FIG. 8.—Case VI. (Geo. B.). Four weeks later; osteoporosis more marked.

here, as in other painful joint conditions, in order to prevent exaggeration of the pain through movement.

The vasomotor symptoms are amongst the most interesting. At first the affected part is usually warmer than its fellow, but it soon becomes cold. Cyanosis is a fairly constant accompaniment, and there is usually slight generalised œdema. The skin is glossy, and, in the acute stage, covered with a profuse sour-smelling perspiration.

The Radiological Picture.—Sudeck originally described an

FIG. 9.—X-ray of tarsus, showing well-marked osteodystrophy at the stage of stippling.

acute and a chronic type of post-traumatic osteodystrophy which varied in their X-ray appearances. The distinction seems artificial, for there is no doubt that the one is a sequel to the other. In general, it is possible to trace the osteoporosis through a series of stages.

In the first phase the affected bones show irregular areas of rarefaction, with some diminution in their general density. In Fig. 9 this stippling effect is well marked in the tarsus. Thereafter progressive decalcification takes place; the bone lamellæ become attenuated, then the cortex, and the entire bone acquires a "glassy" appearance from its almost complete

permeability to the X-rays. The transition may be rapid or slow, and it has an important effect when it is located in the carpus and the tarsus, for the contours of the individual bones become indistinct, and the entire area may be converted into a poorly calcified but almost homogeneous mass. These stages of stippled rarefaction and cortico-lamellar attenuation are followed after a variable time by a phase of recalcification which appears spontaneously, and quietly progresses over a

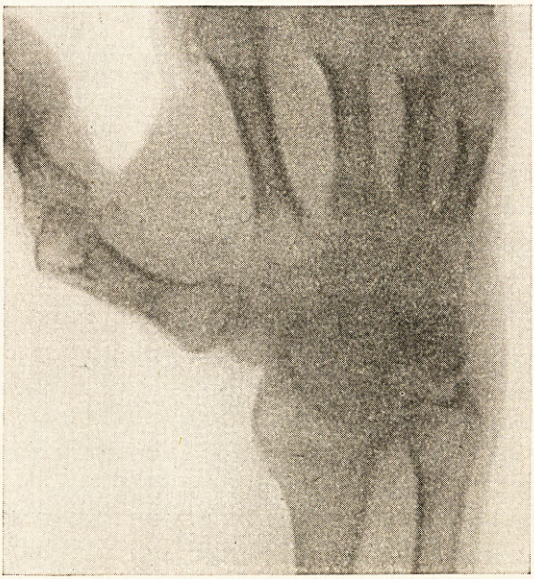

FIG. 10.—Case VII. (Mrs C.). X-ray of wrist nine months after Colles' fracture. A diagnosis of carpal arthritis had been made; but the osteoporosis is evident, and on close inspection, the joint surfaces are quite regular and intact.

period of years. It is probable, however, that complete reconstruction seldom occurs, the old density is never acquired, and unfortunately, the process of repair is often associated with ankylosis. This is the result of a dual process. In the first place, the individual bones become fused; in the second place, the calcium extracted from the bones is apparently deposited in the capsule (Fig. 11). Fortunately, the majority of the other features eventually subside, but despite this, the residual disability may be great and lasting. This was so in the next case, where we were able to study the eventual clinical and radiological appearances.

Case VII.—Mrs C., aged 53, sustained a Colles' fracture of the right wrist in May 1930. Five weeks later the wrist-joint was manipulated because of pain and stiffness. Seven weeks later, the wrist being still swollen, tender, and rigid, a further manipulation was carried out, and a splint applied. The symptoms still persisted for about nine months, however. At the end of this time the joint was completely functionless, but, with care, the usefulness of the fingers was conserved, and she was able to use the hand quite well. She was accordingly discharged, a

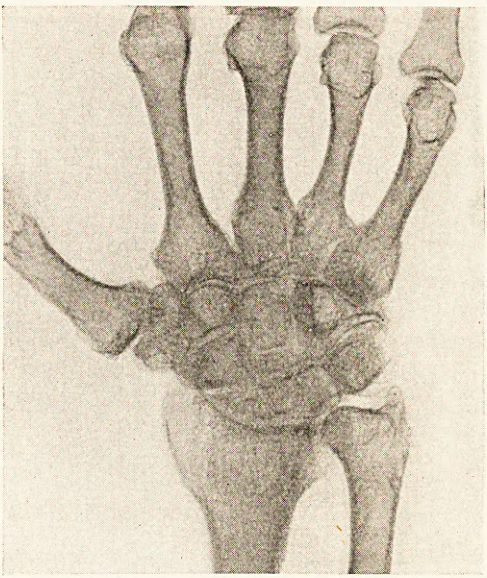

Fig. 11.—Case VII. (Mrs C.). X-ray of same wrist three years after original injury. The osteodystrophy is now healed, but the joint is ankylosed. Note deposits of calcium in capsule bridging the gap between the navicular and lunate and the lower end of the radius.

diagnosis of carpal arthritis having been reached (Fig. 10). On looking over the case recently, however, it became obvious that both symptomatically and radiologically she had been an example of post-traumatic osteodystrophy, and we asked her to report for examination. Her latest radiograms (three years after the original injury) show the usual picture of a healed decalcification—the longitudinal lamellæ, though weaker, are relatively well arranged, but the cortex is thin, the whole wrist is fragile-looking and poorly calcified, the individual carpal bones have fused, and the capsule has become infiltrated with calcium salts (Fig. 11).

The Diagnosis of Post-Traumatic Osteodystrophy.

Concerning the diagnosis of post-traumatic osteodystrophy, it is necessary to add little to what has already been said. Pain of gradually increasing severity, stiffness, and vasomotor phenomena, especially occurring after a trivial injury to a polyarticular region such as the ankle or the wrist, should suggest its onset. So, too, in the case of a fracture, the late occurrence of painful rigidity and œdema, despite adequate reduction and appropriate immobilisation, should be the indication for further radiological examination.

The X-ray picture is to be regarded as characteristic, and there are perhaps two points which offer more help than others —(1) the stippled effect of the decalcification, and (2) the intact nature of the articular surfaces, at least in the beginning of the disease.

There are several conditions, however, all associated with localised calcium depletion, in which confusion may arise. In each, the osteoporosis is apt to be preceded by injury, but the sequence of events is different, and the related clinical phenomena diverse. Despite this, it is certain that previous to its recognition —indeed up to quite recent years—many examples of the lesion have been erroneously regarded and treated as tuberculous or rheumatoid, a diagnosis apparently supported by the long-drawn-out clinical course and the amount of residual disability.

In the main there are three types of osteoporosis which must be distinguished. The atrophy of disuse is the first. It usually follows a period of prolonged immobilisation, and though associated with some degree of stiffness, is painless and without vasomotor accompaniments. In the accompanying radiogram, where it is seen following months in plaster in a successful attempt to induce healing in an old-standing fracture of the navicular, it lacks the mottled character of the true post-traumatic form; the uniform lamellar fragility presents quite a different appearance (Fig. 12).

The atrophy of chronic joint-disease must also be excluded, in particular that form which accompanies tuberculous arthritis and that associated with rheumatoid disease. Tuberculous infection of joints is often preceded by injury, but it usually occurs in the larger joints. The radiological features of the decalcification are very similar to the simple post-traumatic porosis, and this is not surprising, since the all-important factor

of hyperæmia is present in both; nevertheless there is one useful point of distinction, for, in tuberculosis, the articular surfaces are eroded and irregular in consequence of the destruction, and this appearance occurs particularly early when the disease occurs amongst the small and closely-set joints of the tarsus and carpus (Fig. 13).

The atrophy of rheumatoid arthritis is easy to separate, for the rheumatoid disease, though beginning in the polyarticular joints, is seldom preceded by trauma, and is almost invariably

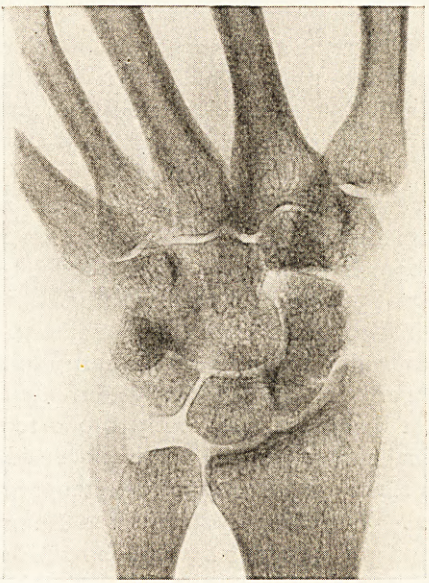

FIG. 12.—X-ray of wrist, showing atrophy of disuse. Taken after four months' immobilisation in plaster for ununited fracture of the navicular.

polyarticular. Further, it never presents the punctate appearance of post-traumatic osteodystrophy.

Neurotrophic atrophy following nerve lesions, or spinal lesions such as spina bifida, is the last condition to which reference need be made. It is practically never an acute process, though it often is apparently started off by a simple injury. Thereafter it progresses slowly over a period of years, until a truly remarkable degree of bone destruction is present, indeed until the bones are converted into cylinders of fibrous tissue, completely devoid of mineral salts. There are almost invariably present certain tropic changes which give additional help in the diagnosis (Fig. 14).

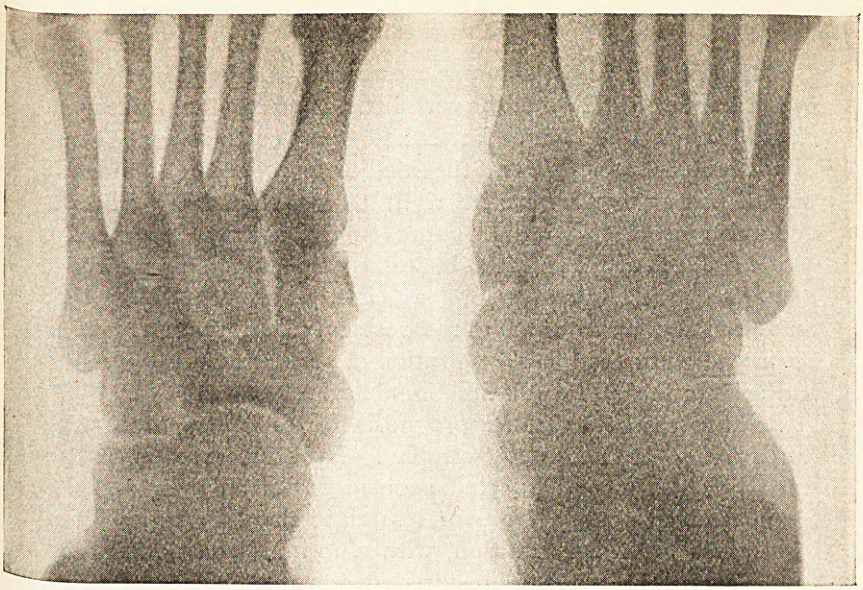

FIG. 13.—Tuberculous arthritis of the right tarsus. Note particularly the destruction of the joint surfaces, especially of the navicular and the cuneiform—the feature of diagnostic importance.

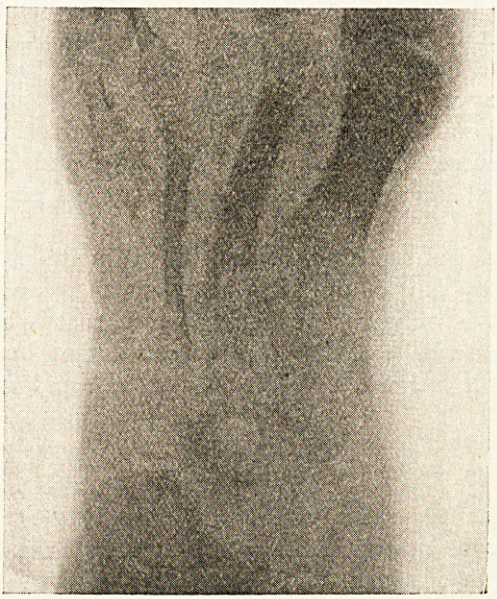

FIG. 14.—Neurotrophic atrophy. Shows gradual disappearance of metatarsal bones.

Prognosis.

Mild cases of post-traumatic osteodystrophy may clear up in the course of a few months with little or no residual functional disability, but this cannot be looked upon as the usual course of untreated cases. From the medico-legal point of view it would, in our experience, be right to say that there exists a general tendency to gradual improvement, and that, eventually, recalcification of the affected bones takes place. This improvement frequently takes as long as eighteen months to become manifest, and unfortunately, the recalcification, though usually accompanied by distinct amelioration of pain, is seldom complete and seldom initiates a full functional recovery.

Within these limits, the prognosis varies considerably with the situation of the osteodystrophy. Eventual recovery is the rule when the foot is affected, save in so far as the patient is frequently left with a more or less rigid flat foot, or even, in some cases under observation, with a form of spastic flat foot. In the wrist the ultimate result depends upon various factors. In the severe type following Colles' fracture, permanent limitation of movement is the rule, while mild cases following slight sprains may make a relatively complete recovery. Finally, collapse of the rarefied semi-lunar bone may take place from the centripetal pressure of the surrounding carpal bones, initiating one form of the so-called Kienboch's disease. This condition may in itself be responsible for persistent symptoms due to chronic arthritic changes. In the shoulder, pain, which, is at first severe, diminishes very gradually, and limitation of abduction and lateral rotation is nearly always persistent and very crippling.

It is doubtful to what extent the ultimate prognosis can be affected by any of the simpler means of treatment at our disposal. It is only fair to add, however, that a considerable body of evidence exists which suggests that operations on the autonomic nervous system provide a rapid cure for the condition.

Treatment.

Treatment may be discussed under three headings—(1) simple conservative measures; (2) acetylcholine; and (3) sympathectomy, which may be either of the periarterial variety or a ganglionectomy.

Simple conservative measures are designed to provide for amelioration of pain and increase of movement. The results are very variable. The use, for instance, of diathermy or radiant heat may relieve pain in one case and aggravate it in another. Massage is seldom helpful and is often rendered impossible because of pain, but voluntary use of the affected joints within the limits imposed by pain would appear to be indicated, rather than complete rest on the one hand, or forcible movements on the other. In those common cases where the foot is affected, the support of elastoplast bandages is greatly appreciated by the patient.

Manipulation under anæsthesia to break down adhesions has been used frequently in the case of the shoulder-joint, being followed in many cases by splinting in an abducted position. We have scarcely ever seen beneficial results from this method of treatment, at all events in the active stage. Theoretically, one would imagine that such a traumatising measure would be strongly contra-indicated as liable to increase and prolong the hyperæmia which is the ultimate cause of the osteoporosis. After the lapse of time, when recalcification has taken place, it is quite possible that manipulation might be very helpful. This is a point upon which we should not care to give a definite opinion. Deep X-ray therapy has been said to improve the condition, but of this method we have no experience.[16]

The injection of acetylcholine subcutaneously or intramuscularly has been said to result in rapid loss of pain, increase in movement, and early recalcification. The rationale of this method of treatment appears to be as follows:—It is now widely suggested that motor nerve impulses do not pass directly through the motor nerve-endings to the effector organ, say a muscle fibre, and act as a direct stimulant, but that, under the influence of the nerve impulse, a chemical substance is liberated in the end organ, and that this chemical effector produces the contraction of the muscle fibre. Especially is this theory accepted in the case of the parasympathetic system. The chemical effector produced at the parasympathetic end organs is identified as acetylcholine, a drug which experimentally reproduces all the actions attributed to the parasympathetic system. In the animal, its injection is said to lead to a profound, though very temporary fall in blood pressure, which is believed to be due, not, as one might suspect, to a

cardiac vagus inhibition, but to an active dilatation of the peripheral arterioles, the capillaries remaining contracted. If this be true, the injection of acetylcholine would be tantamount to a temporary sympathectomy. A body of literature does, indeed, exist which tends to show that the use of this drug can relieve the acute crises of vasospastic conditions such as Raynaud's disease and causalgia.

When acetylcholine is produced at parasympathetic nerve-endings, it is clearly necessary that a mechanism shall exist for its rapid destruction in order to prevent a permanent vaso-dilation, or an accumulation of the drug in the blood-stream with a consequent progressive and fatal fall of blood pressure. This is carried out by means of a ferment which exists in human blood, and which is capable of causing almost immediate dissociation of acetylcholine into acetic acid and the relatively inactive choline. It is, indeed, difficult to see how this normal efficient mechanism can allow of a generalised therapeutic action after injection.

We have administered courses of acetylcholine in relatively large doses to a number of patients suffering from post-traumatic osteodystrophy, and in many cases they have expressed themselves as benefited by the injections. Observation has not, however, shown that these cases showed any striking departure from the normal tendency to slow spontaneous improvement. Further, after such injections we have been unable to demonstrate any definite fall of blood pressure, or any dilatation of the branches of the central artery of the retina as viewed by the ophthalmoscope. Finally, it is not to be expected that the injection of a vasodilator would be beneficial to a lesion which, we shall see, is associated with a pathological hyperæmia. The suggestion that acetylcholine is of value probably results from the faulty belief that the dramatic cures produced by sympathectomy in these cases are the outcome of the hyperæmia engendered by that operation. Whatever the value of the drug may be in other conditions, it is our belief that it is useless in the treatment of post-traumatic osteodystrophy.

The last therapeutic method which comes under consideration is that of *sympathectomy*. Introduced in the treatment of post-traumatic osteodystrophy by its greatest exponent, Leriche, a large body of literature has grown up to show that the operation, if carried out reasonably early, gives immediate and

lasting relief to sufferers from this condition. It may appear paradoxical that an operation which is widely known to be followed by hyperæmia should give such relief, and it is probably for this reason that the method of treatment has not been extensively applied by the cautious Briton in spite of the glowing accounts from our more enthusiastic Gallic neighbours. You have heard to-night of three typical cases of osteodystrophy which obtained dramatic immediate relief from sympathectomy, and it is our belief that the apparent paradox of hyperæmia curing hyperæmia may be at least partially resolved by a clinical consideration.

Ætiology.

Whatever the complexities of calcification and decalcification may be, and however little we may as yet understand the control of the fine chemical processes at work, it is now very generally admitted that hyperæmia is a necessary concomitant of decalcification, and *vice versa*. It is therefore within the bounds of human certainty that the form of osteoporosis under consideration results from or is accompanied by hyperæmia. Leriche has applied this knowledge in the form of a theory which states that injury, acting through a local autonomic axon reflex, is productive of the hyperæmia. So far so good. Hyperæmia is a necessary result of injury in order that the normal inflammatory reaction of healing may take place. Such a theory, however, does not explain why the normal healing reaction should in certain cases be magnified into a prolonged hyperæmia sufficient to cause marked decalcification of the neighbouring bones, nor does it suggest why hyperæmia and osteoporosis of the post-traumatic type should be so constantly associated with severe pain. Osteoporosis of corresponding degree from other causes is certainly not accompanied by the same amount of disability.

Other somewhat similar theories might be suggested. It might be thought that the hyperæmia in question results from the liberation of histamine from the injured tissues or from the failure of the specific blood ferment to destroy acetylcholine in the neighbourhood of the injury. All such theories have the same loop-holes, and it is noticeable that they are all primarily concerned with an explanation of the hyperæmia which is present.

We feel that a new outlook on this question is to be obtained from a consideration of the results of sympathectomy. Over a large number of recorded cases the post-operative history has been the same. A patient has, let us say, a stiff painful wrist following an injury, and a radiogram shows marked osteoporosis. A sympathectomy is carried out, and it is worth noting that the immediate results appear to differ little according to whether a ganglionectomy or a periarterial sympathectomy is chosen, in spite of the acknowledged fact that the hyperæmia following the latter is of a very temporary nature. The result of this operation is striking and consists of immediate loss of pain, recovery of movement in the course of weeks, and recalcification in the course of months. Such a story, when repeated over and over again in the literature, suggests three important conclusions:

1. The fact that recovery from pain and recovery of movement can take place while the osteoporosis remains unaltered means that the disability does not in any way arise from the hyperæmia and the rarefaction of the bones. The osteoporosis is an accompanying manifestation, and helpful in diagnosis, but nothing more. It need not continue to fill the clinical picture to the virtual exclusion of the other features of the condition.

2. The fact that recovery is claimed to occur as completely after the admittedly very temporary hyperæmia of a periarterial sympathectomy as after a ganglionectomy, can have only one explanation. Sympathectomy does not cause recovery from post-traumatic osteodystrophy by virtue of division of vasoconstrictor fibres and resulting hyperæmia.

3. The relief of pain after operation is almost immediate. The patient, whose wrist was very painful before entering the theatre, awakes from an anæsthetic practically free from his pain. There can, we think, be only one explanation of such a phenomenon. The operative procedure has succeeded in dividing the afferent tracts by which the patient's painful sensations were carried.

These three simple clinical conclusions enable us to build up a theory of ætiology which, to some extent at least, meets the facts. In cases of post-traumatic osteodystrophy, an injury has set up a persistent reflex consisting on the one hand of a painful sensation carried by afferent fibres running in the sympathetic system, and on the other of a reflex efferent

impulse causing vasodilation and consequent osteoporosis. Probably a vicious circle is set up, the local hyperæmia keeping up the pain and the pain keeping up the hyperæmia, with the accompanying stiffness as the result of the pain.

A sympathectomy cuts the afferent path of the reflex. Pain disappears at once and normal use of the affected joint soon results in return of movement. Hyperæmia settles down, and gradual recalcification takes place.

The presence of afferent autonomic pain-bearing fibres is essential to such a conception of post-traumatic osteodystrophy. Unfortunately very little is known of such fibres in the limbs. They exist in the trunk, for afferent fibres from the abdomen are capable of carrying painful sensations on stretching of the walls of the hollow viscera. Clinical experience makes it very likely that the periarterial nerve plexus contains afferent sensory fibres. Those who have removed the appendix under local anæsthesia know how necessary it is to anæsthetise the mesentery carefully owing to the severe pain produced by crushing the appendicular artery. The presence of this arterial sensation is also evident in the limbs. In dealing with an incised accidental wound without anæsthesia, pain is not a very marked feature save on stitching the skin, and even more so on catching and ligating bleeding points. Infiltration anæsthesia which is not quite perfect will frequently suffice for the ordinary tissues, while blood-vessels retain their painful reaction to cutting and especially to crushing.

It is a well recognised fact that a sympathectomy in one limb may materially benefit a vaso-spastic condition in the opposite limb as well as on the side operated upon. Surely this suggests a crossed reflex action from division of afferent fibres.

Finally, Friedrich [6] has shown that arterial pain-sense persists after division of the ordinary sensory nerves to a limb, while Hellwig [8] claims that it is abolished by a periarterial sympathectomy.

It is our thesis that a future lies before the fuller investigation of the anatomy and function of the afferent side of the autonomic reflex arc and of the effects following upon the division of these fibres. Such afferent tracts may well prove to be as important in clinical work as the efferent vaso-constrictor fibres which are so much in the surgical eye at the moment.

We have to thank Professor John Fraser for his interest in this study, and for permission to make use of two cases upon whom he operated.

NOTE.—Since this paper was read, two of the cases treated by periarterial sympathectomy have suffered a return of pain. No reference to such an eventuality is contained in the voluminous literature—mostly French—on this subject, but it is in keeping with modern British views on the evanescent effects of the operation. Our series, however, is yet too small to justify a dogmatic statement, and we hope at a future date to consider more fully the results of autonomic nervous system operations in their relation to osteodystrophy.

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DISCUSSION.

Professor John Fraser said—The authors are to be warmly congratulated on their paper; they have investigated what at first sight might seem to be a relatively minor point in pathology, on evidence which I am sure must have come under the observation of all of us many times, but which we have seen without appreciation of its significance. Mr Middleton and Mr Bruce have shown, however, that this point which we have so often failed to observe in its proper proportions is really a matter of supreme significance in the case of joint difficulties which are apt to arise as the result of what at first might seem to be minor injuries. There are one or two points, however,

about which I am not clear, and I venture to ask the speakers if they can throw some light upon them.

There is the question of the localisation of this disturbance. That there must be some general factor at work is, I think, certain, otherwise the occurrence of the disturbance would have a much more general distribution than it has. Age seems to be important, for, so far as I know, the condition is not met with in children, but it is unlikely that the age factor in itself is the only reason. There must surely be some additional element, probably of a toxic character, localised in the area of the joint, and acting as the devitalising factor.

My second difficulty is in regard to the explanation of the benefits which follow sympathectomy. I feel sure from what Mr Middleton and Mr Bruce have said that they belong to the order of Greigites—that they group themselves with those who believe that decalcification and calcification are intimately related to variations in vascularity, that it is a biochemical process, and not due to the active constructive work of bone cells. If they believe that the decalcified areas which they have so well shown and described are the result of a hyperæmia, I find it difficult to understand why the operation of sympathectomy should prove of such striking benefit, for surely one of the most outstanding results of sympathectomy is a vasodilation with resulting hyperæmia. Mr Middleton has suggested that the explanation of the benefit of sympathectomy may arise from the division of the afferent fibres as they pass along the line of the main blood-vessel, and I join issue with him on that point, because I cannot conceive it likely that any great bulk of afferent fibres is concentrated in the small area of the vessel wall. I believe that it is much more likely that the afferent stream is a very widespread one, arranged on a segmental basis. I feel that we have yet a good deal to learn about the very elements of this whole question.

My last criticism (if I may call it so) is in respect of the name which Mr Middleton and Mr Bruce have adopted—"Post-traumatic Osteodystrophy at Joints." In some way, it does not seem to ring true, and even so far as the individual words are concerned, there is an element of contradiction.

Mr J. W. Struthers spoke.

Mr Walter Mercer said—I should like to bring to the notice of the authors of this communication a paper which I heard some years ago, of which I have now only a very vague recollection. It was on two cases of post-traumatic osteodystrophy, in both of which the injury was of a very minor type—so far as I remember, the injury in one case was a dog-bite, in the other some other minor injury. This author showed slides exactly like what we have seen to-night, and he put the

condition down to what we called a neurotrophic lesion of the dorsal interosseous nerve. It was interesting that in both cases he tried conservative treatment first, and without benefit; but later, in one case he injected the dorsal interosseous nerve, just as it crosses the posterior aspect of the lower end of the radius, with some solution of iodine, and in the other case he actually severed the nerve. He got, he said, what I take to be an equally dramatic result from his operation. It seems to me that this may open up for the authors a new conception of the ætiology.

Mr Middleton replied.