TENDON LESIONS IN RHEUMATOID ARTHRITIS
A CLINICO-PATHOLOGICAL STUDY

BY

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In his treatise on the nature and cure of rheumatism, Scudamore (1827) described the tendons as " knotty at their insertions and often contracted "; and Stockman (1904) used the term " tendinous rheumatism ". Klinge (1933) described microscopic lesions in the tendons and joint capsules of patients dying of active rheumatic heart disease, but the typical rheumatoid tendon lesions have been adequately described only by Scandinavian workers.

Helweg (1924) stated that snapping fingers were very common in patients suffering from rheumatoid arthritis. He noticed that in such cases large nodules could be felt in the palm of the hand, and that these nodules moved with the long flexor tendons of the fingers to which they were presumably attached. Sandberg (1941) reported a case of rheumatoid arthritis with snapping of all ten digits of both hands. At operation large glassy nodules were found in the long flexor tendons at the level of the metacarpo-phalangeal joints. Both Kahlmeter (1933) and Edström (1945) have stressed the importance of " tendinitis " as a cause of disability in rheumatic subjects, and in a survey of the Lund material Edström found clinical evidence of " tendinitis " and " peritendinitis " in 48 per cent. of 391 cases of chronic rheumatic polyarthritis. The long flexors of the fingers were the tendons most often involved, being affected in 38 per cent. of Edström's cases.

There is a large surgical literature on the subject of the snapping finger. In recent reviews (Compère, 1933; Henri, 1947) and in other important reports (Marchesi, 1905) the snapping finger or thumb was usually an isolated lesion associated with trauma, tumours, or occupational hazards such as piano playing; generalized rheumatic disease being exceptional in these cases. Most authors consider stenosis of the tendon sheaths to be an important cause of snapping, though nodules in the tendons also play their part. True stenosing tendovaginitis, as described by De Quervain (1895) and more recent writers, Finkelstein (1930) and Henri (1947), seems to be a rather different condition, being largely confined to the region of the radial styloid and being characterized by massive thickening of the fibrous sheath.

We have recently made a study of the tendon lesions of rheumatoid arthritis, and have compared them with a few cases of isolated snapping digits and of De Quervain's disease.

Clinical Material

During the past eighteen months we have explored the long flexor tendons of the fingers in fifteen cases of rheumatoid arthritis and in two cases that presented isolated snapping digits. In some of these cases advanced tendon lesions were

TABLE

INCIDENCE IN RHEUMATOID ARTHRITIS

Cases	Authors'	Edström's
With tendon lesions	42	188
Without tendon lesions	58	203
Total cases	100	391*
Sites of tendon lesions:		
Arms: Flexors only	31	125
Extensors only	0	26
Both flexors and extensors ..	6	12
Legs: Flexors only	2	24
Extensors only	2	12
Both flexors and extensors ..	3	4

* Edström's figures include both "tendinitis" and "peritendinitis".

known to be present and the operation was done as a therapeutic procedure, while in others the operation was done as a biopsy in early cases judged to have only minimal lesions; the following account of the pathological anatomy and clinical manifestation of the rheumatoid tendon lesion is largely based on this operative material.

We have also made a systematic clinical study of the incidence of these tendon lesions in rheumatoid arthritis, and the Table shows our findings in the last 100 cases seen in this department, together with Edström's figures for comparison.

Tendon lesions were judged to be present in forty-two of these patients, and in 37 of them the tendons of the long flexors of the fingers were affected. Other tendons affected were the extensors of the fingers, the tendons around the ankle joint, and the Achilles tendon. Our findings therefore agree with those of Edström and require no further comment.

In most cases of rheumatoid arthritis the tendon lesions are only one aspect of a generalized process involving the joints, muscles, and occasionally the viscera. But in rare cases the tendon lesions may be the chief cause of disability. The long flexors of the fingers are the tendons usually affected in these cases, and we will therefore describe in some detail the anatomico-pathological changes in these tendons and the disability which they produce, as a clear understanding of the clinical picture is essential for the proper care of these patients. These tendon lesions also provide an excellent opportunity for studying the histological changes which are found in the fibrous tissue in active rheumatoid arthritis.

Anatomico-Pathological Considerations

The long flexor tendons enter the palm by passing under the volar carpal ligament, at which level they are surrounded by the palmar synovial sheath. In the distal half of the palm the synovial sheath is lacking, each tendon being covered

merely by a thin fibrous sheath which contains three well-developed transverse bands (Fig. 1, B, B, B). In dissections of normal hands we have found that the most proximal of these bands is formed by the transverse fibres in the distal border of the palmar fascia. The middle band arises from the deep transverse palmar ligament and encircles the tendon at the level of the metacarpal heads. The distal transverse band lies in the fibrous sheath that binds the tendon to the volar aspect of the proximal phalanx (Fig. 1, S). Between these encircling bands the fibrous sheath is thin and lax.

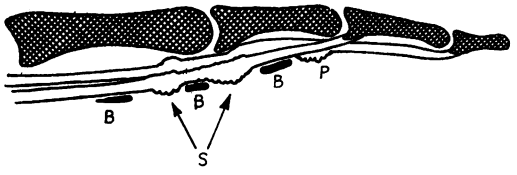

FIG. 1.—Diagram showing the sites of the nodule formation on the flexor sublimis tendon (S) and the profundus tendon (P). The three thick transverse bands in the fibrous sheath are marked (B).

In rheumatoid arthritis pathological changes may be found in both the tendons and their sheaths. The least change appears to be a dulling of the surface of the tendon. At a more advanced stage there is a thickening of the tendon, which looks yellow and streaked with grey gelatinous material. Frequently the tendon is nodular, and in extreme cases large cauliflower-like masses may be found (Figs 2 and 3). The nodules may lie in the centre of the tendon or on its surface, and some of the surface nodules look as if they had burst out from the centre of the tendon. In some severe cases the nodules become adherent to the tendon sheaths (Fig. 4). The nodules may be soft and gelatinous or firm and rubbery, and occasionally they may be quite hard.

These tendon changes are usually found in the palm at the level of the metacarpal heads, the tendons of flexor digitorum sublimis being more often affected than those of profundus. The index and medius are more commonly affected than the other fingers, and the thumb is but rarely involved. Occasionally one finds a large nodule in the profundus tendon just after it has come forward between the two heads of sublimis at the level of the proximal phalanx (Fig. 1, P), but nodules in other situations on the flexor tendons are uncommon.

The transverse fibrous bands in the tendon sheaths may soften and disintegrate, in which case the thickening and nodulation of the tendons causes little disability. But if the transverse bands remain intact, the thickened tendons become jammed under these bands, so interfering with active flexion of the fingers. Small nodules give rise to snapping fingers, and large nodules become completely impacted between two transverse bands so that all active flexion of the interphalangeal joints is lost.

The synovial sheaths in the palm and fingers usually contain a slight excess of fluid and there may be some proliferation and engorgement of the synovial tissue, but these changes are slight compared with the striking nodulation and swelling of the tendons themselves.

Clinical Picture

In those rare cases in which the tendon lesions are the sole cause of disability in the hand, the patients may complain of snapping fingers, but more frequently

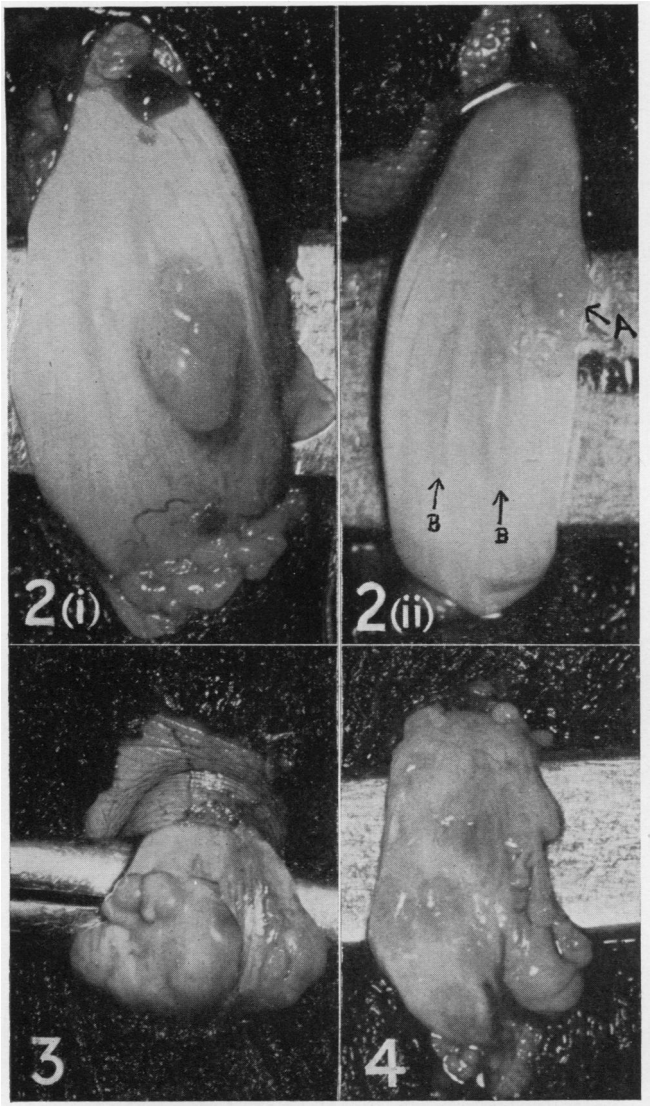

FIG. 2.—Operative exposure of flexor sublimis tendons in palm, showing (i) a typical tendon nodule and (ii) less severe changes, such as a small nodule at (A) and ridges of similar tissue at (B). The patient, a man 36 years old, had suffered from a low-grade rheumatoid process for 12 years. Joint changes were not severe, but he had multiple large necrobiotic nodules and enlargement of the spleen and lymph glands. The blood sedimentation rate was 30 mm. Westergren. After operation, hand function was greatly improved.

FIG. 4.—Operative exposure of flexor sublimis tendon in the palm, showing advanced replacement of tendon with nodular tissue. The adherent tendon sheath has been dissected off the tendon. All flexor tendons of both hands were affected. This patient, a woman 54 years old had suffered from rheumatoid arthritis for 6 years. There were severe arthritic changes in most of the joints and the disease was active at the time of operation. The blood sedimentation rate was 80 mm. Westergren. After the operation hand function was, if anything, worse.

FIG. 3.—Operative exposure of large firm nodule in flexor profundus tendon opposite proximal phalanx of index finger. The patient, a man 37 years old, had suffered for 3 years from typical rheumatoid arthritis, in clinical remission at the time of operation. The blood sedimentation rate was 18 mm. Westergren. This nodule was impacted, and operation was required to restore active flexion of the index finger.

the complaint is simply of weak and painful fingers. When asked to make a fist, the patients flex their metacarpo-phalangeal joints, but leave the interphalangeal joints fully extended. The interphalangeal joints can, however, be moved passively and painlessly through a full range and the joints themselves are not tender.

Palpation of the palm reveals firm, tender swellings in the region of the flexor tendons at the level of the metacarpal heads, and these swellings may be felt to move with the tendons as the fingers are passively flexed and extended.

Some patients are able to work their fingers loose during the day so that the full picture is only present in the early morning; but, though some active flexion may be regained, this movement usually has a snapping element and is at best extremely weak and painful. Because of the weakness and loss of flexion of the fingers, these patients are unable to hold objects such as tools and cooking utensils and have difficulty in opening doors; above all the women are unable to sew or peel potatoes, which is a serious handicap to many of these patients who are forced to lead a sedentary life because of their disease. In the majority of patients, however, the tendon lesions are accompanied by varying degrees of arthritis in the joints of the hands, the tendon lesions being relatively unimportant, and in these cases there may be some difficulty in deciding what proportion, if any, of the disability is attributable to the tendon lesions.

Treatment

The majority of rheumatoid tendon lesions require no special treatment. In the early acute cases the tendon thickening subsides as the disease process remits, and full movement may be regained. In severe cases the fibrous sheaths soften as the disease progresses and full movement may be restored in spite of massive nodule formation.

When pain is a prominent feature, relief may be obtained by a volar plaster slab which supports the hand in the position of rest with the wrist in extension and the fingers in partial flexion, though this should be removed once a day for gentle exercises. Helweg recommends massage and Edström uses *x*-ray therapy to hasten resolution in these cases, but we have not found it necessary to use either of these forms of treatment.

The important cases are those which fail to regain active movement of the fingers although the disease process is relatively quiescent as judged by good general condition, low sedimentation rate, and absence of active arthritis. These patients often present massive nodulation of the tendons as well as large nodules elsewhere in the body, and these cases, we think, may be usefully treated by operative incision of the tendon sheaths and such paring of nodules as seems expedient.

The operation is conveniently done under local anaesthesia. The tendons are exposed by a transverse incision in the creases of the palm and finger according to the site of the lesion.* The tendon sheaths are then slit open; with a little burrowing the incision in the tendon sheath can be carried well up into the finger so that all three constricting bands are divided. Both the sublimis and the profundus tendons are then hooked up and any obvious nodules removed until the tendons move quite freely. Only the skin is sutured, and the incision is protected by a plaster cast with a volar slab extending to the middle

* In some cases incisions were made along the length of the tendon to facilitate exposure for photography.

of the basal phalanx, so immobilizing the metacarpo-phalangeal joints and helping to break the patient's habit of flexing the finger at this joint only. Active flexion of the interphalangeal joints is started at once, and in a favourable case without concomitant arthritis a good grip can be regained in three to four weeks.

Postoperative Results

Seventeen patients have been operated on in this way. In nine the operation was planned as a therapeutic procedure and the results have been fairly satisfactory in that increased movement and a stronger grip have been maintained for periods of three months to one year. In eight patients the operation was essentially exploratory. One of these patients, who had very active disease and much concomitant arthritis, is worse, but the remainder are if anything slightly improved though the result would not have warranted an operation as a purely therapeutic procedure. We therefore suggest that the value of operative treatment may be worth exploring in a few carefully selected cases in which the disease process is not very active; but the long-term results are as yet unknown, and in at least one of our cases there has been a recurrence of snapping. On the other hand, if any operative reconstruction of the rheumatoid hand is being considered, the proportion of the disability due to tendon lesions must be carefully assessed, as any attack on the joints may prove fruitless if the chief cause of disability is in the tendons.

One of the interesting features of these tendon lesions is the opportunity they provide for studying the microscopic changes which occur in fibrous tissue during the early and active stages of rheumatoid arthritis.

Histological Findings

Previous histological studies of rheumatoid arthritis have been largely confined to synovial membrane, muscle, and subcutaneous tissue, although Klinge (1933) mentioned inflammatory lesions occurring in the tendinous structures around joints. But the only histological description we have found of the typical rheumatoid tendon lesion is Lundquist's account of Sandberg's case (1941). This report is brief, stating that the tendon sheath was infiltrated with plasma cells and lymphocytes and showed fibroblastic proliferation and occasional fibrinoid degeneration; the tendons were described as being infiltrated in places with " chronic inflammatory altered " connective tissue. We will therefore describe the tendon and tendon sheath changes in some detail. For convenience the tendon and tendon sheath will be considered separately.

Material and Methods.—From the fifteen cases of rheumatoid arthritis subjected to operation, thirty-eight biopsies of the flexor tendons and fifteen biopsies of tendon sheath were taken for histological examination. For comparison we have examined tendons and tendon sheaths in five cases of De Quervain's disease. Paraffin sections were cut after fixation in either 10 per cent. formol saline, 4 per cent. lead acetate, or Carnoy's fluid. The stains used included toluidine blue, Haidenhain's azan, Mayer's haemalum and eosin, Weigert's elastic stain, and Van Giesons stain combination, Gomori's reticulin method, Lendrum's phloxine tartrazine, and Pollack's rapid trichrome method. Occasionally sections were stained for bacteria by the Gram and Ziehl-Neelsen stains. In a number of cases the biopsies were studied in serial section.

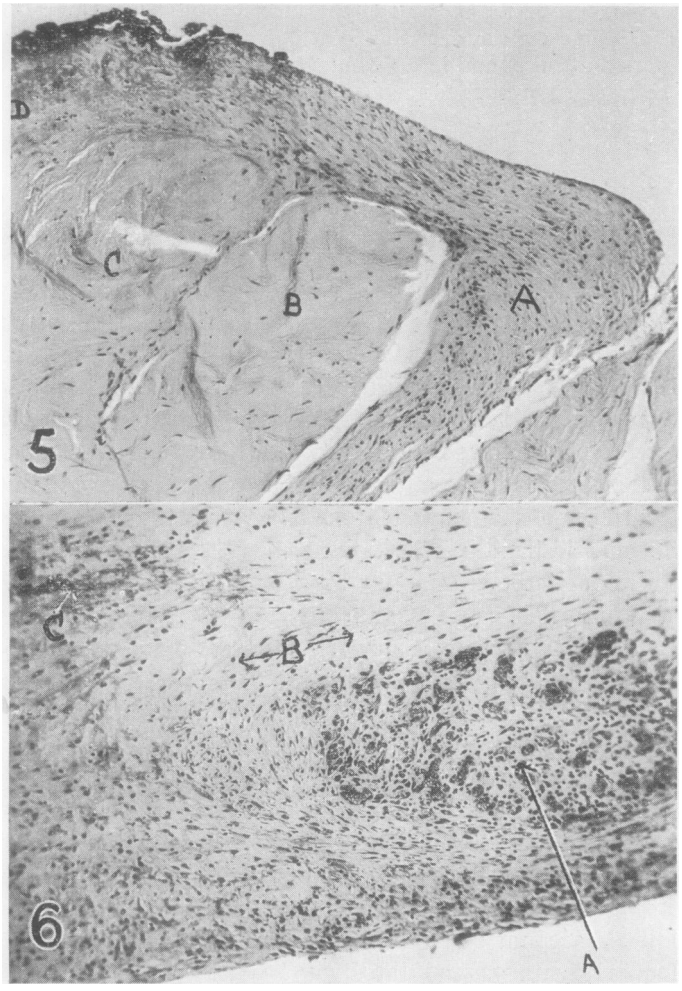

FIG. 5.—Fibrous thickening of the mesotenon (A). Normal mesotenon between healthy tendon bundles (B) and (C). At (D) the thickened mesotenon shows fibrinoid degeneration. Haemalum and eosin. ×75.

FIG. 6.—Grossly thickened peritenon showing characteristic congeries of young granulation tissue (A), areas of mature collagen (B), and a focus of fibrinoid degeneration (C). Haemalum and eosin. ×80.

Tendon.*—There were two cases in which at operation the only change noted was a dull streaky appearance of the surface. In one the changes were limited to the peritenon. This was thickened by old fibrosis, though the inner zone showed some oedematous loosening of the fibres and dilated capillaries, but no inflammatory infiltration. In the other case the findings in the peritenon were similar, except that the oedema was more marked. The mesotenon, however, was also thickened in parts, mainly by old fibrosis (Fig. 5, A), though in one region the tissue contained foci of fibrinoid necrosis (Fig. 5, D).

In two instances the tendon was found at operation to be grossly enlarged but not nodular. The pathological basis of this alteration was a widespread

* The loose connective tissue separating the tendon bundles will be designated "mesotenon". "Peritenon" refers to the loose connective tissue lying on the exterior of the tendon and binding the bundles together.

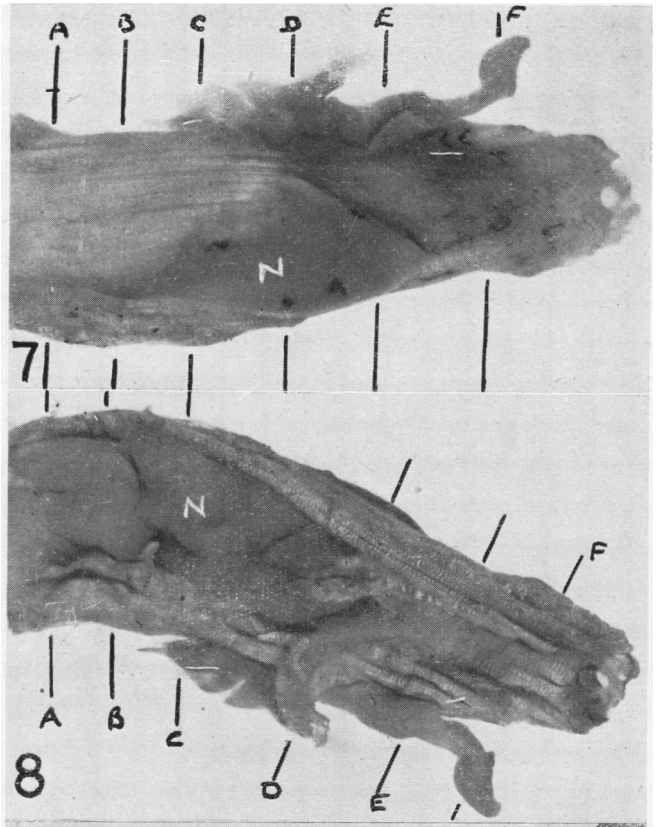

FIG. 7.—Superficial aspect of the biopsy taken from the tendon in Fig. 2. N=the surface nodule. The lines A to F in Figs 7 and 8 represent planes of section shown in Figs 9 to 14. ×4.

FIG. 8.—Deep aspect of the biopsy shown in Fig. 7. In the substance of the tendon there is a large nodule (N) well demarcated from the tendon bundles in parts, but merging with them in other places. ×4.

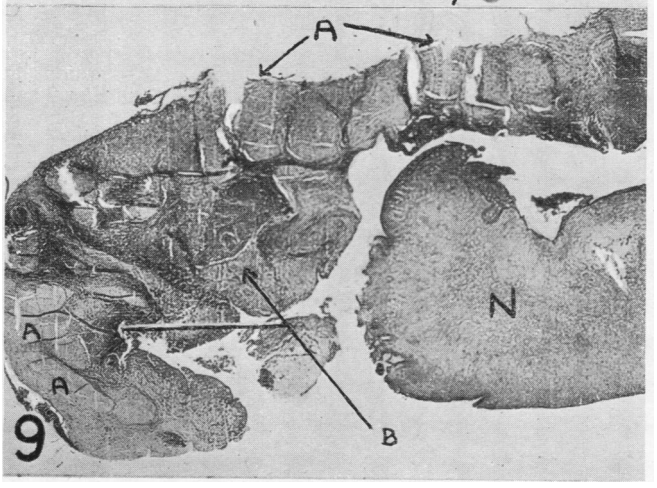

FIG. 9.—Section at Plane A in Figs 7 and 8. Many of the superficial tendon bundles are normal (A), but the deeper bundles are necrotic (B). Nodule (N) consists of a granular necrotic fibrinoid mass. Note absence of inflammatory reaction. Haemalum and eosin. ×20.

replacement of the peritenon and mesotenon by a thick layer of granulation tissue, a feature of which was the considerable histological variation found in neighbouring microscopic fields, especially in the more active parts. Areas of proliferating

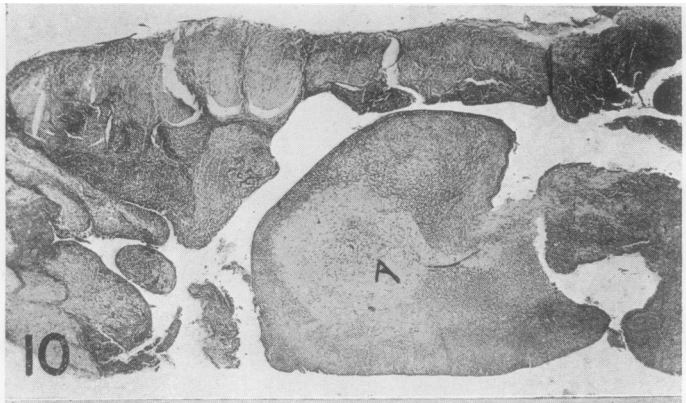

FIG. 10.—Section at Plane B in Figs 7 and 8. Changes are shown similar to those in Fig. 9, but in the centre of the necrotic nodule an area of proliferating capillaries and fibroblasts is present (A). Haemalum and eosin. ×20.

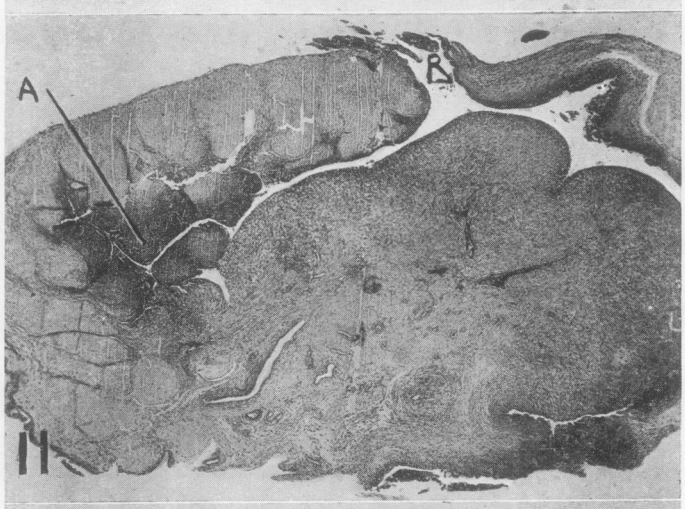

FIG. 11.—Section at Plane C in Figs 7 and 8. Necrotic nodule is largely replaced by granulation tissue which is separating the overlying tendon bundles and is about to break through the ruptured peritenon at (B). Necrosis of the deep tendon bundles is present at (A). Haemalum and eosin. ×20.

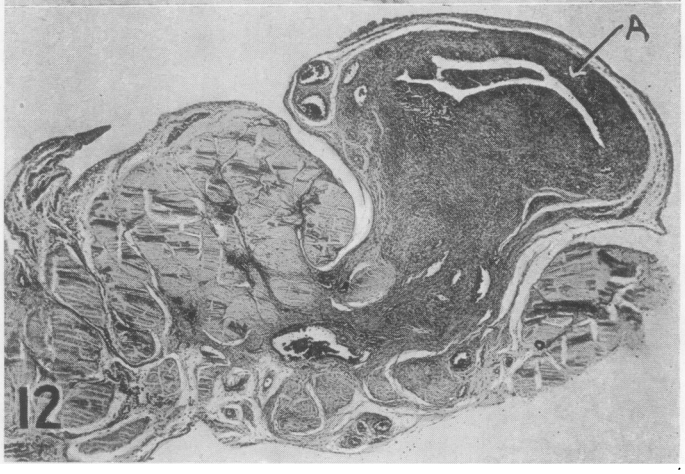

FIG. 12.—Section at Plane D in Figs 7 and 8 shows the surface nodule connected to deep tissue by a stalk. The upper part contains a typical necrobiotic focus with peripheral palisade (A). Haemalum and eosin. ×20.

capillaries and young fibroblasts, surrounded by a collar of fine connective tissue fibres, were scattered here and there (Fig. 6, A). Between these foci, fields of more

compact collagen and areas of fibrinoid necrosis were irregularly disposed (Fig. 6, B and C). Some scanty infiltration with polymorphs and lymphocytes was present. Apart from occasional small areas showing early degeneration, the tendon bundles were normal.

These changes showed no close resemblance to the Aschoff nodule, the subcutaneous necrobiotic nodule of rheumatoid arthritis, or the subcutaneous nodule of rheumatic fever (Bennett and others, 1940).

Thus in apparently less severe cases the lesions in the tendons were mainly found in the mesotenon and peritenon, in which thickening by granulation tissue or fibrosis was the dominant feature.

The majority of the tendon biopsies showing nodule formation were essentially similar, and their main pathological features may be illustrated by describing the findings in one fairly typical case.

At operation a medium-sized nodule (0·75 cm. by 0·5 cm.) was found on the surface of the flexor sublimis tendon of the middle finger (Figs 2 and 7). Naked-eye examination of the biopsy revealed another large nodule on its deep aspect (Fig. 8). The whole specimen was cut serially in transverse section, and the histology will be depicted by describing the changes noted in passing through the series from the plane A to the plane F, as indicated in Figs 7 and 8.

At the level A (Fig. 9), the nodule on the deep aspect consisted of a granular mass of fibrinoid necrotic material loosely attached to the overlying tendon bundles. Many of the latter, although still recognizable as such by virtue of the preservation of the general tendon bundle pattern, showed varying degrees of fibrinoid degeneration. The intervening mesotenon also showed fibrinoid necrosis and in some places was thus involved when the enclosed tendon bundle appeared normal. Only the most superficial part of the biopsy was entirely normal at this level. There was, hereabouts, a striking absence of inflammatory cellular infiltration or proliferative activity in relation to the necrotic and degenerative areas.

In the region B the picture was altered by the appearance in the interior of the necrotic nodule of a small area of proliferating capillaries and fibroblasts (Fig. 10). In succeeding sections this area of granulation tissue rapidly enlarged until it almost completely replaced the necrotic nodule and surrounding necrotic tendon bundles (Fig. 11). Although generally homogeneous, there was in places some lack of uniformity in this granulation tissue, areas of young fibroblasts and capillaries being intermingled with more mature collagen fibres and small foci of fibrinoid necrosis. As the region C was approached, the deep nodule of granulation tissue increased further in size and, pushing between the superficial tendon bundles, moved towards the surface (Fig. 11). At this point the peritenon was necrotic and ultimately ruptured (Fig. 11, B); and through this breach the enlarging nodule of granulation tissue emerged to appear on the superficial aspect of the tendon as a surface nodule (Fig. 12). The greater part of the circumference of this nodule was entirely free from the adjacent surface of the tendon, but its base remained attached to the interior of the tendon by a short stalk (Fig. 12). From the plane C (Fig. 11) to the end of the series the deep nodule was gradually replaced by normal tendon bundles. About the level D (Fig. 12) the structure of the superficial nodule presented an even more varied histological pattern than that previously noted in the granulation tissue in the deep part of the biopsy. Areas of fairly compact collagen occurred in juxtaposition with foci of capillaries and proliferating fibroblasts and areas of fibrinoid necrosis. In other places widely dilated capillaries were found embedded in fine fibrous tissue, and in still other areas numerous swollen fibroblast nuclei lying in an avascular

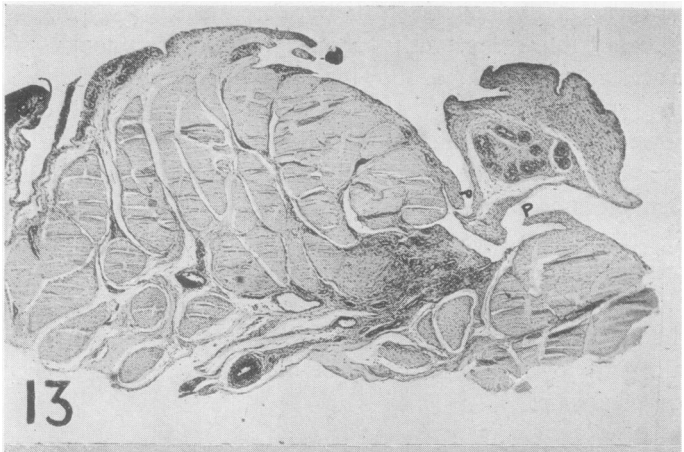

FIG. 13.—Section at Plane E in Figs 7 and 8. The surface nodule appears disconnected from the biopsy. At its base the free ends of the ruptured peritenon (P) can be seen. At this level the nodule consists largely of relatively mature fibrous tissue. Haemalum and eosin. ×20.

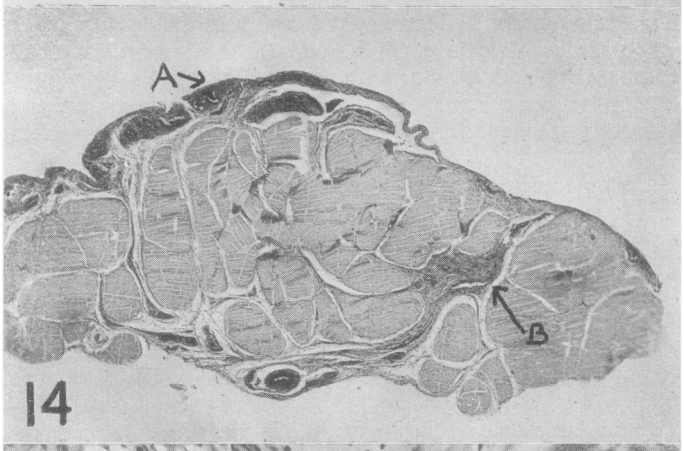

FIG. 14.—Section at Plane F in Figs 7 and 8. Note focal thickening of the peritenon (A) and increased vascularity of the tendon. At (B) there is some fibrous thickening of the mesotenon. Haemalum and eosin. ×20.

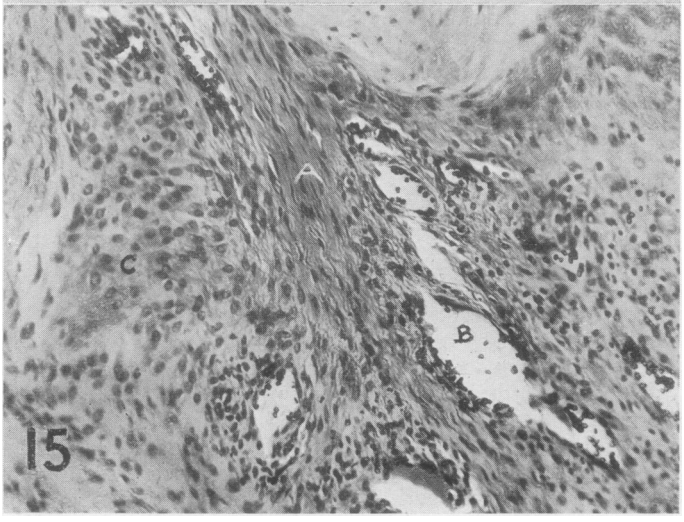

FIG. 15.—Part of the surface nodule in Fig. 12, showing degrees of maturation of the fibrous tissue: a stand of mature collagen (A); dilated capillaries (B); a group of swollen fibroblasts (C). Haemalum and eosin. ×140.

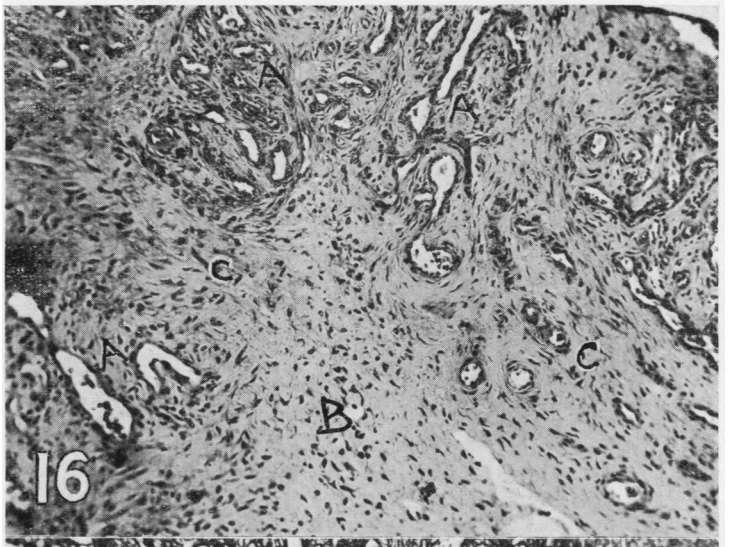

FIG. 16.—Part of a tendon nodule showing congeries of capillaries and fibroblasts (A), sparsely fibrous avascular area (B), and areas of mature collagen (C). Haemalum and eosin. ×95.

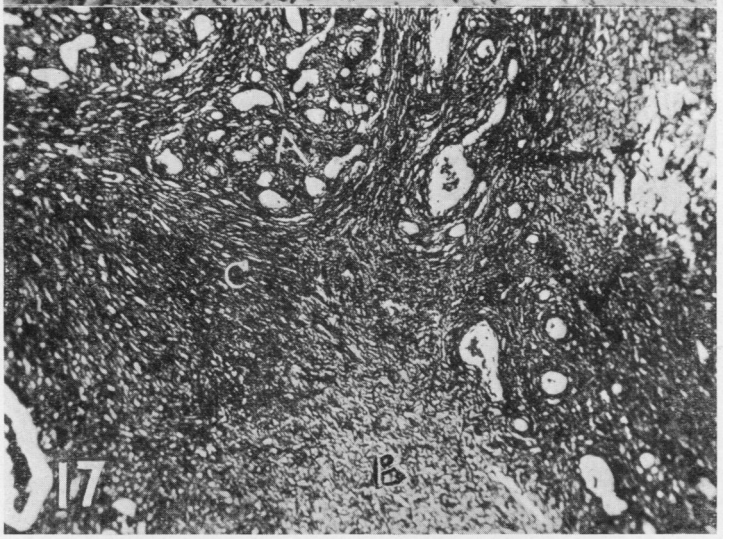

FIG. 17.—Consecutive section but one to that shown in Fig. 16, showing variations in fibre density. Gomori's reticulin method. ×95.

matrix predominated (Figs 15 and 16). Moreover, these various areas showed no regular orientation to each other, and their proportions varied in different parts of the nodule. Around some of the necrotic patches a palisade of connective tissue cells developed, the focus then being indistinguishable from the commonly occurring subcutaneous necrobiotic nodule (Fig. 12, A). Although polymorphs, lymphocytes, and plasma cells were present, especially in the vicinity of the necrotic foci and in areas of young granulation tissue, inflammatory infiltration was never a prominent feature in the nodule.

From the plane D to the end of the series there was a marked increase in the vascularity of the deeper parts of the tendon. At the level E (Fig. 13) the surface nodule appeared to be disconnected from the main part of the biopsy, though the point of its origin from the deeper parts of the tendon was still apparent from the rounded free ends of the ruptured peritenon. In this region (E) the nodule consisted mainly of relatively mature quiescent fibrous tissue.

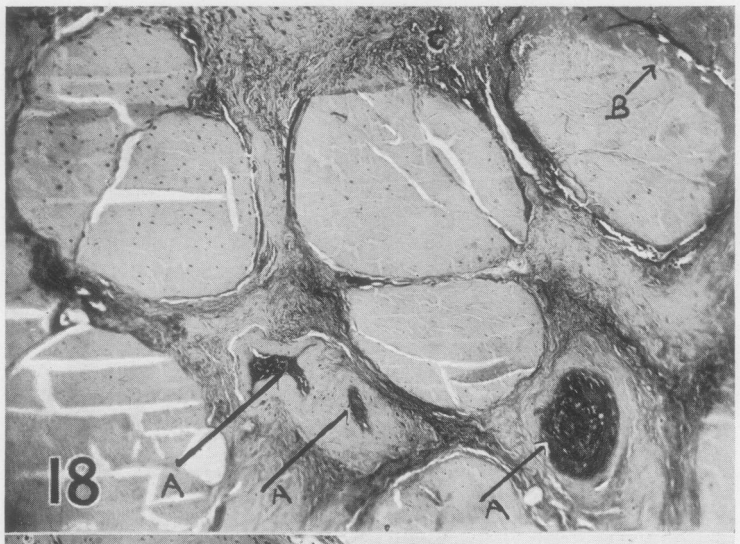

FIG. 18.—Focal necrosis in tendon bundles (A). At (B) the necrosis involves the periphery of the bundle. The mesotenon is irregularly thickened and shows fibrinoid necrosis at (C). Haemalum and eosin. ×75.

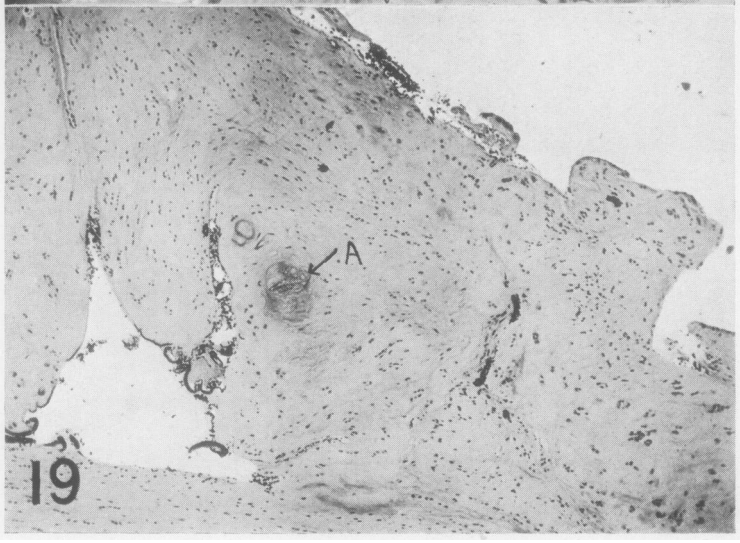

FIG. 19.—The base and part of a fibrocartilaginous type of tendon nodule with areas of mucoid degeneration (A). Haemalum and eosin. ×48.

In the remaining sections in the series the surface nodule gradually disappeared and normal tendon bundles occupied the deep parts of the biopsy. Finally the sections differed from normal tendon only in focal thickenings in the mesotenon and peritenon and some increase in vascularity (Fig. 14).

Some further remarks may serve to complete the above description of the changes occurring in this severe type of tendon lesion. From the nature of the material, casual sections can hardly be expected to reveal the findings described above. Further, when the biopsy consists only of the surface lesion (as happened in some of our cases), the complete picture of its pathogenesis will not be evident histologically. As in the case described above, surface nodules appear commonly to arise by the extension of granulation tissue originating in the deep parts of the tendon.

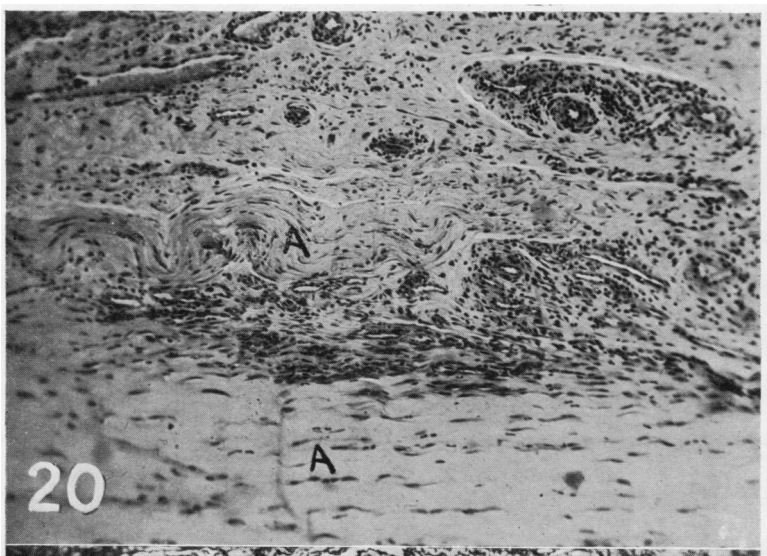

FIG.20.—Shows part of a normal tendon bundle (A) disappearing into an area of granulation tissue. The difficulty here is to know whether normal tendon has changed so as to present the appearance of granulation tissue, or whether the latter is merely replacing an area of necrotic tendon. Haemalum and eosin. ×75.

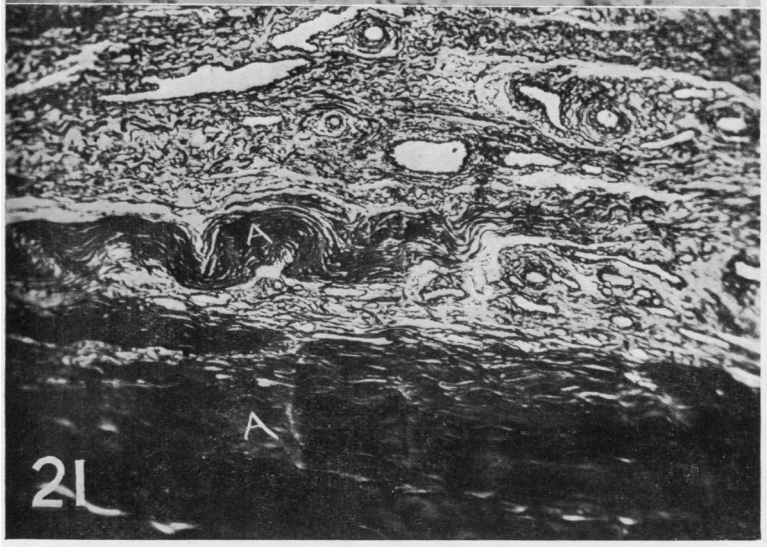

FIG. 21.—Consecutive section to that shown in Fig. 20, to illustrate the fibre pattern. Gomori's reticulin method. ×75.

Occasionally, however, small surface nodules may result from purely local connective tissue proliferations in the peritenon. Again, while massive tendon bundle necrosis was a feature in the case described, it was not uncommon to find merely a few fibres or part of a single bundle involved in the necrotizing process (Fig. 18).

In a few cases both superficial and deep nodules were made up mainly of old fibrous tissue containing some dilated capillaries and arterioles. But this quiescent form showed no close correlation with the clinical assessment of activity of the disease. Moreover, active and quiescent nodules were sometimes seen in one and the same patient.

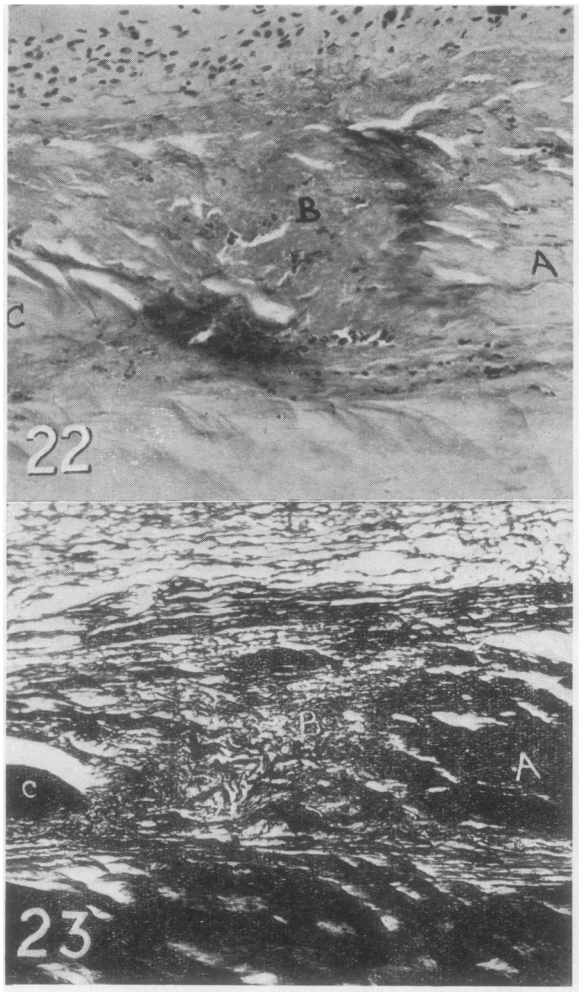

FIG. 22.—Early tendon fibre degeneration. Normal tendon (A) and (C) becomes indistinct at (B) where scattered polymorphs are present. Haemalum and eosin. ×110.

FIG. 23.—Consecutive section to that shown in Fig. 22. Note the altered fibre pattern in the area of degeneration. Gomori's reticulin method. ×110.

There was, however, one exceptionally hard nodule formed of almost completely avascular fibrocartilage containing small areas of mucoid degeneration (Fig. 19). The tendon bundles at the base of the nodule also showed a type of fibrocartilaginous change, though less marked and accompanied by considerable increase in vascularity. Clinically the patient had mild inactive rheumatoid arthritis and, although all intermediate stages between this and the common granulation tissue type of nodule were not seen, the suggestion may reasonably be made that the fibrocartilaginous nodule represents a possible end stage of the more usual surface lesion.

Tendon Sheath.—At the proximal and distal ends of the biopsy portions of the synovial sheaths of the palm and digit were usually seen. They commonly showed a chronic non-specific synovitis with lymphocytes and plasma cells infiltrating oedematous areas containing numerous dilated capillaries. In most cases the intervening fibrous sheath was either normal or showed minor changes, mainly consisting of small areas of oedema or fibrinoid necrosis associated with some capillary dilatation and slight inflammatory infiltration. Occasionally, however, areas of fibrinoid necrosis and granulation tissue were more extensive. When the surface layer of flattened cells was involved, deposits of fibrinoid material containing polymorphs were found.

The chronic synovitis may be merely secondary to the abnormal friction occasioned by the presence of nodules and enlargement of the tendon, for identical synovial sheath changes may be found at the limits of the thickened part of the sheath in De Quervain's disease, which is also characterized by abnormal movement

of the tendon in its sheath and which occurs mainly in non-rheumatic patients. Nevertheless, the average case of true stenosing tenosynovitis with its pronounced hyaline thickening of the sheath and minor tendon changes is easily distinguished both macroscopically and microscopically from the usual changes found in rheumatoid arthritis.

Early Tendon Changes.—In a number of biopsies we were able to study what appeared to be the early stages of tendon fibre degeneration. At first the compact fibrillar appearance of normal tendon became indistinct, or sometimes the fibres were regrouped into thick strands (Fig. 22). At this stage the fibres showed a tendency to stain blue after Mayer's haemalum and were often loosely infiltrated with polymorphs. After reticulin staining they were often fragmented and displayed a tendency to rupture and curl up on themselves (Fig. 23). As the typical fibrinoid appearance developed, the fibres often broke up into droplets. The tendon cells appeared first to enlarge and then to disappear. Occasionally degenerating fibres could be seen rejoining normal tendon. More often they disappeared into a reticulin network containing young fibroblasts and capillaries. Sometimes normal tendon fibres appeared to merge into a mass of granulation tissue, a finding which presented a fundamental difficulty in interpretation; for it was always uncertain whether the granulation tissue had merely replaced a necrotic area of tendon or whether the latter had degenerated in such a way as to produce a tissue with histological features resembling maturing granulation tissue (Figs 20 and 21).

Summary and Discussion of the Histological Findings.—The digital flexor tendons consist almost entirely of collagen fibres and ground substance. For this reason they are a particularly useful site in which to study the collagen changes in rheumatoid arthritis. In the main, our findings are in general agreement with what has already been deduced about these changes from studies in other sites such as the subcutaneous tissue. We will, therefore, draw attention here only to those of our findings which seem to be of particular interest.

In the tendon the essential lesion is often microscopic in dimension and almost always irregular in distribution ; and even in a single section individual lesions of varying severity and age may be encountered. The degree of alteration varies from an oedematous separation of the fibres to a reduction of the compact tendon fibre system into a swollen homogeneous granular mass containing at first a reticulin network, but later becoming completely afibrillar. In adjacent mesotenon or peritenon there is a pronounced formation of granulation tissue ; and the tendon nodule, the commonest cause of tendon disability, is an expression of the exuberant production of this tissue in the active stages of the disease.

Not infrequently, nodules occurring on the surface of the tendon can be regarded as a mass of granulation tissue isolated from parent connective tissue. And, since lesions identical with the subcutaneous necrobiotic focus of rheumatoid arthritis may occur in such nodules, it would seem that at least one form of genesis of this structure is a focal fibrinoid degeneration of performed nonspecific granulation tissue.

There is, in fact, much evidence in our material suggesting not only that parent

collagen (tendon bundle, peritenon and mesotenon) may be involved by fibrinoid necrosis, but that the associated granulation tissue may, at any stage in its development, also undergo similar degenerative changes. Thus, the usual course of maturation of this tissue is interrupted here and there by the occasional and irregular appearance in it of focal areas of degeneration; and these foci appear to act as stimuli for the further production of young granulation tissue. Such a sequence of events provides an explanation for the large amounts of granulation tissue produced; it may also explain the characteristic irregularity of the general histological pattern and the diversity in age and density of the collagen fibres found in this tissue (Fig. 17).

That this character of the granulation tissue in the active stages of rheumatoid arthritis is not limited to the tendon lesion is, perhaps, evidenced by the massive accumulations of pannus that may occur in the joints in this disease. But apart from this special feature the cellular tissue response is essentially non-specific—at least in the sense that an approach to diagnosis is possible only when the typical necrobiotic focus with characteristic palisade occurs, which is by no means a constant finding.

There was, nevertheless, ample opportunity to study the necrobiotic focus, especially the process by which it enlarges. This would appear to depend upon a necrosis of part of the peripheral palisade, which is promptly followed by the formation of a new palisade layer, and so on. The area of necrosis in the palisade layer occurs irregularly, first at one point, then at another, thereby accounting for the peculiar and characteristic serpiginous outline of the lesion. A similar account was given by Bennett and others (1940) of the subcutaneous necrobiotic focus. This finding also provides another example of the liability of the repair tissue to undergo focal fibrinoid necrosis with the resulting production of more granulation tissue.

The general problem of repair in tendon, particularly following trauma, was reviewed by Albertini (1929). It may, however, be stated that in the opinion of many workers the process is a form of replacement fibrosis, the basis of which is a connective-tissue proliferation in the mesotenon and peritenon. In our material granulation tissue not uncommonly appeared to be replacing necrotic tendon bundles. But the final stages of healing were not observed, and the ultimate process of repair of these tendon lesions remains an open question.

Summary

1. The clinical features of rheumatoid tendon lesions in the hand are described and the lesions are shown to occur in approximately half the cases of rheumatoid arthritis.

2. In fifteen cases the tendons were explored at operation, and this material was used for a study of the morbid anatomical changes in the tendon and tendon sheath.

3. Thirty-eight tendon biopsies and fifteen biopsies of the tendon sheath from these fifteen cases formed the basis of the histological study.

4. The histological findings in serial sections of one case are described in detail.

5. The mode of origin of the tendon nodule, the histological nature of the tissue reaction, and the early histological changes in the tendon fibres are described.

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Lesions Tendineuses dans l'Arthrite Rhumatismale; étude Clinique et Anatomo-Pathologique

RÉSUMÉ

1. On décrit les traits cliniques des lésions rhumatismales tendineuses de la main et on montre qu'elles surviennent à peu près dans la moitié des cas d'arthrite rhumatismale.
2. Dans quinze cas les tendons furent examinés à l'opération et le matériel recueilli fut utilisé pour étudier les changes anatomiques morbides du tendon et de sa gaine.
3. Trent huit prélèvements du tendon et quinze prélèvements de la gaine tendineuse de ces 15 cas forment la base de cette étude histologique.
4. On décrit d'une façon détaillée les résultats histologiques de l'examen des séries de coupes dans un cas.
5. On relate le mode de formation du nodule tendineux, la nature histologique de la réaction tissulaire et les changes histologiques des fibres tendineuses au début.

Lesiones Tendinosas en la Artritis Reumatoide: Estudio Clinico-Patologico

RESUMEN

1. Se describe los caracteres clínicos de las lesiones tendinosas de la mano y se demuestra que estas lesiones ocurren aproximadamente en la mitad de los casos de artritis reumatoide.
2. En quince casos los tendones fueron explorados en el curso de una operación y el material así recogido fué empleado para estudiar las alteraciones anatomo-patológicas del tendón y de su vaina.
3. Treinta y ocho biopsias del tendón y quince biopsias de la vaina tendinosa de estos quince casos forman la base del estudio histológico.
4. Se describe detalladamente los resultados histológicos de una serie de cortes en un caso.
5. Se describe el modo de originar del nódulo tendinoso, la naturaleza histológica de la reacción del tejido y las alteraciones histológicas tempranas de las fibras tendinosas.