Tendon involvement in the rheumatoid hand

K. M. BACKHOUSE,* APRIL G. L. KAY, E. N. COOMES, AND A. KATES

From the Charing Cross Hospital Medical School and the Department of Rheumatology, St. Mary Abbots Hospital, London

Tenosynovitis is commonly found in the hand in rheumatoid disease and may lead to rupture of certain tendons. Brewerton (1957) and Savill (1969) have both commented on the high proportion of patients attending medical outpatients for the disease who show tendon involvement, putting the proportions at 65 and 50 per cent. respectively. Edström (1945) gave a lower figure of 42 per cent. Kellgren and Ball (1950) reported an incidence of 37 flexor and extensor tendon lesions in 100 consecutive cases of rheumatoid arthritis. They described the positions of lesions seen clinically and also in fifteen cases subjected to open operation. The extent of surgery was not stated but it appears to have been fairly local and directed to individual digital tendon lesions. These figures appear high, but should probably be higher still, in view of the great difficulty of clinical examination of flexor tendons separated from the skin by thick coverings of palmar fascia and flexor retinaculum. Tenosynovial thickenings are commonly found at operation for median nerve decompression in patients in whom rheumatoid arthritis had not been suspected clinically.

Tendon rupture in rheumatoid arthritis has been attributed directly to tenosynovitis, but other causative agents have been postulated. Bäckdahl (1963) pointed to the caput ulnae as an important focal feature leading to damage and eventual rupture of those extensor digital tendons related to the disorganized and hypermobile bone end. Vaughan-Jackson (1959) pointed out that damaged bone, in certain cases, may lead to roughness and spicule formation, which abrades the tendons.

Kay (1971), in her study of the natural history of rheumatoid disease in the hand, produced clinical evidence of different degrees of involvement of the long flexor tendons to the various fingers. This work, however, called for more detailed examination, which was possible where open operation was indicated. On the flexor aspect of the hand, where clinical assessment is extremely difficult, the clear view offered by surgery was both valuable and salutory.

Preliminary observations at surgery had pointed to characteristic patterns of tendons and sites most affected, and these it was decided to plot.

Methods

The present study is based on seventy hand operations, 44 on the palmar aspect and 26 on the dorsum. Both assessments and operations were carried out by two of us working together (K. M. B. and A. K.). The main reasons for surgery were severe unresolving tenosynovitis, median nerve compression in known rheumatoid arthritis or diseased inferior radioulnar joint. Cases of tendon rupture were not included in the series, unless of recent occurrence.

All hands were examined carefully preoperatively and a plot of tendon involvement was made which could later be compared with that found at operation. Some of the patients had been followed in the natural history study of the rheumatoid hand (Kay, 1971) and for various reasons required operation. These patients therefore had detailed hand reports over several years.

A full examination of the synovium covered flexor or extensor tendons was made in each case, and patients in whom wide surgical exposure was considered advisable (i.e. major dorsal or palmar tenosynovial clearance) were included in the series. It should be pointed out, however, that in some cases the operation was begun as a relatively simple median nerve decompression and was extended to flexor tendon clearance when it was realised that the tendons were far more involved than had been appreciated preoperatively.

The extensor tendons were exposed by a longitudinal incision with a bend across the skin creases at the wrist (Fig. 1B). This gave both adequate thumb and digital tendon clearance and a ready approach to the distal radioulnar joint without risk of scar contracture.

The flexor exposure consisted of one incision along the distal palmar crease and a second along the thenar crease, which either curved across the wrist or had a narrow skin bridge there, and was then carried for
Granulomatous lesions
Thickened synovium on the tendon which could easily be stripped off leaving a shiny surface.
(2) Synovium sticking to the tendon leaving dullness and surface damage on removal.
(3) Granulomatous lesions in tendon with disruption to fibrous strands.
(4) Regions of necrotic material within the tendon leaving only peripheral tendon strands.
(5) Stretched thinned tendon.
(6) Tendon rupture.

Grades 4 and 5 would appear to be two different types of prerupture states in tendons. Hence, the numbering does not necessarily represent a chronological sequence in the course of the disease. Grades 1 and 2 are essentially synovitis with no more than surface lesions to the tendon, whereas Grades 3 to 6 show different forms of intrinsic destruction.

Observations
Carpal Tendons
These almost always showed less synovitis and damage than the long tendons to the fingers. Of the 44 flexor cases, none showed synovitis of flexor carpi ulnaris: flexor carpi radialis was comparably free in all except two cases. One of these showed only light involvement in the carpal tunnel; the other had mild synovitis proximally and a necrotic lesion near the insertion.

The extensor tendons were more frequently affected. Of abductor pollicis longus and extensor pollicis brevis,
ten showed Grade 1 or 2 lesions proximally, nine under the retinaculum and twelve distally; one had a stretched, thinned pair of tendons under and distal to the retinaculum. The extensor carpi radialis tendons showed superficial (Grade 1 or 2) involvement in thirteen cases proximally, nine under and ten distal to the retinaculum, while two had granulomatous lesions within and distal to the fibro-osseous compartment; two others had necrotic tendons under the retinaculum and one distally. Extensor carpi ulnaris showed a comparable incidence of synovitis; there were nine superficial lesions proximally and seven distally, but in addition there was one granulomatous lesion proximally and three distally as well as three necrotic tendons under and distal to the retinaculum.

**DIGITAL TENDONS**

Extensor pollicis longus showed sixteen Grade 1 or 2 lesions proximally, twelve under the retinaculum and seven distally. Severer lesions related to the retinaculum consisted of one granuloma proximally, two under and distal; thinned stretched tendons, two under and five distally, and ruptured tendons two under and three distally. The dorsal tubercle of the radius (Lister’s tubercle) is considered as being under the retinaculum. The distal lesions could be brought to this point only by full thumb extension and were therefore beyond the range of possible friction from the tubercle.

Most of the other extensor tendons showed Grade 1 or 2 synovitis; the severer lesions (3 to 6) were all found under or distal to the retinaculum. The incidence of these was approximately the same for each finger except the ring, which was the highest:

- **Viz. index 1 1 middle 2, ring 4, little 2 3**
- the upper figures being beneath the retinaculum and the lower distal. The ruptures were all distal to the retinaculum but in that part of the tendon zone which would just pass beneath it in fullest extension; finger tendons so affected were five little, five ring, two mid, and one each index tendon.

On the flexor aspect the profundus tendons were more severely involved than superficialis (sublimis). Although a high proportion of sublimis tendons showed synovitis, this was nearly always of a lesser degree in the palm and wrist; only three cases showed Grade 3 to 6 damage distal to the carpal tunnel and none within. Profundus damage was, in every case, more severe than sublimis damage in this region. Most cases in this series showed Grade 3 lesions. There was a small number of Grade 4 to 6 lesions, which were found distal to the retinaculum. There was no difference in incidence in individual finger tendons, but the bulk of synovial material was usually greatest around the middle finger tendons followed closely by the index, then the ring, and least around the little.

Within the fingers, as distinct from the wrist and palm, the digital tendons presented a very different picture. The incidence of synovitis was much less than in the more proximal region. The number of patients showing minor synovitis (Grades 1 and 2) was essentially the same around the two tendons, but sublimis showed more severe lesions. These were found in the zone of the tendon that moved in and out of the fibrous flexor sheath, though one patient had more distal Grade 3 lesions in all fingers and another had a similar lesion in the middle finger alone.

Flexor pollicis longus was far less commonly involved than the finger tendons. In this series, it was often found to be apparently clear of synovitis while the other tendons were heavily cuffed or disrupted. Severer lesions were, all except one, found distal to the retinaculum, close to the metacarpophalangeal joint, which was also the site of the single rupture found in the series. Granulomatous lesions were on occasions seen only on the deep surface of the tendon.

Bone spicules were found in only two cases in relation to damaged tendon. In one, Lister’s tubercle had been eroded and the extensor pollicis longus was thinned and stretched. In the other a granulomatous lesion was found on the deep surface of a tendon running over the dorsum of the radius and there was a subjacent granuloma of the bone. Both lesions of tendon and bone had smooth surfaces until the lesions were cleaned out; then the bone hollow had rough edges. In a third case the grossly disorganized distal end of ulna had ulcerated through the dorsal joint capsule, but the extensor digitii minimi tendon running over the ulcerated bone had only a Grade 1 lesion; more severe lesions were found on the extensor tendons to the index and middle fingers which were not related to the ulcerated bone.

There was good correlation between clinical observations and surgical findings on the extensor aspect so far as synovial bulk was concerned. The degree of tendon damage was much less easy to assess. Considerable tenosynovial bulk often showed little or no tendon damage, whereas relatively little swelling might overlie more severe tendon damage, difficult to assess clinically. Localized pain on a tendon under load was the most consistent indication of tendon damage.

In the palm the thick fibrous flexor retinaculum precluded effective indication of tenosynovial swelling in this region. Proximal to the wrist, the synovial bulk usually needed to be considerable before it was readily apparent, the synovitis usually being greatest around the deeper, profundus tendons. A fair clinical/operative correlation of tenosynovial bulk was possible distal to the retinaculum but, as on the extensor aspect, there was no relationship between bulk and underlying tendon damage. Here again, pain under load was a useful indication of damage. Blocking of digital movement appeared to be due more to synovial bulk than to tendon disruption, but tendon thickening obviously played a part in producing the restricting mass.

**Discussion**

The observations made in the palm agreed in sitting with those of tenosynovial nodule formation in relation to the digits, as described by Kellgren and Ball (1950). Our own observations were, however, mainly made on cases with more extensive disease. Kellgren and Ball did not mention the relative severity of involvement of the profundus and sublimis (superficialis) tendons. Probably this was due to their surgical procedures being fairly local and directed to individual clinically apparent tendon nodules, rather than to a full tenosynovial clearance.
### Table I
Sums of all grades (1 to 6) of disease of each tendon at each site in all patients

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<tr>
<th>Extensors (26)</th>
<th>Abductor pollicis longus and extensor pollicis brevis</th>
<th>Extensor carpi radialis and brevis</th>
<th>Extensor pollicis longus</th>
<th>Extensor indicis</th>
<th>Communis Index</th>
<th>Middle</th>
<th>Ring</th>
<th>Little</th>
<th>Extensor digiti minimi</th>
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<th>Sublimis</th>
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<td>Finger in sheath</td>
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<td>7</td>
<td>8</td>
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### Table II
Number of grade 1 and 2 and 3 to 6 lesions at each site in all patients

<table>
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<th>Extensors (26)</th>
<th>Abductor pollicis longus and extensor pollicis brevis</th>
<th>Extensor carpi radialis and brevis</th>
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<th>Communis Index</th>
<th>Middle</th>
<th>Ring</th>
<th>Little</th>
<th>Extensor digiti minimi</th>
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<tr>
<td>Finger in sheath</td>
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<td>1</td>
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The present series might be considered loaded towards cases of greatest synovial bulk on the flexor aspect, because they were selected for operation mainly on account of median nerve compression. But the comparison with Kellgren and Ball's more local pictures, together with the consistency of our findings, make such a loading unlikely to be important.

The fact that all the cases of median nerve compression were found at operation to be associated with marked synovitis, particularly of flexor digitorum profundus, even when this was apparent clinically only as minimal thickening along the tendons distal to the retinaculum, indicates that the clinical diagnosis of palmar tenosynovitis is not easy. Yet this distal region is the only one readily accessible clinically, albeit under a thick palmar aponeurosis and, in addition to the secondary median nerve compression, is the one most likely to suffer severe tendon damage.

Although the cause of rheumatoid disease is still debated, some factors influencing its course are well known. During the active phases rest is useful in reducing synovial pathology, whereas exercise increases it. So far as the metacarpophalangeal joints are concerned, Castillo, El Sallah, and Scott (1965) have shown that, in the natural history of the disease, although exercise may maintain the hands in a good functional state, it is likely to lead to an increased destruction of the bone related to the joint. No comparable studies have been made on tendons, but as these structures may be related to diseased synovium, it might be expected that exercise could increase tendon damage.

The present study is based on too small a number of patients to quantitate the inter-relationship between amount of exercise and tendon damage. Differences in activity of individual tendons in the hand and incidence of synovitis and tendon destructive lesions are, however, of interest.

Direct comparison between tendons having widely different function and range of movement is not valid as, for example, between the long digital tendons and the shorter ones which are active over the wrist.

Among the wrist tendons with comparable range of movement, comparison is more acceptable. The dominant wrist movement is flexion and ulnar deviation (Backhouse, 1968), radial deviation and extension being the counter (antagonist) movement. Flexor carpi ulnaris is the main prime mover of the wrist and the extensor digiti minimi muscles the antagonists; the extensor carpi ulnaris, flexor carpi radialis, and abductor pollicis longus act as synergists and fixators. But flexor carpi ulnaris is not involved in the tenosynovial system at the wrist in relation to the retinaculi and, as we have shown, is spared in the disease. Numerically in our series the extensor carpi radialis tendons were most often affected by synovitis but, on the other hand, the proportion of severer lesions in those tendons affected was greatest in exterior carpi ulnaris. The major lesions on the individual tendons were situated distal to and, to a lesser degree, under the retinaculum. Although the amount of movement could be accepted as being related to the incidence of tenosynovitis, it could not account for the sitting of the more destructive lesions of the wrist tendons.

The two long digital flexors show a difference in functional activity; flexor profundus acts in practically all flexor movements, in consort with the interossei, but the flexor superficialis (sublimis) plays only an accessory role, particularly in power movements (Backhouse, 1968). In the wrist and the palm the amount of use of each tendon matched the pattern of tenosynovitis found. But the whole length of each tendon is obviously used and if tendon work were the prime consideration then all parts of the tendon having a synovial sheath should be equally affected. As in the case of the shorter tendons, damage is greatest at the distal end of the palmar synovial sheath and distal to the flexor retinaculum. This section of tendon in turn is far more affected than that of the digital synovium where, if severe lesions occur, they do so proximal to the fibrous flexor sheath.

The amount of work being carried out by a tendon and muscle obviously cannot be the prime factor in the aetiology of lesions. But synovial stressing, although linked with muscle activity, is not directly dependent upon it; synovial loading is a product of muscle power output and change of tendon direction. Thus at any one time, if the power output of the muscle is constant, the load on the concave surface synovium will be directly proportional to change of direction of the tendon. On other surfaces the load will be related to the pressure of surrounding structures and most marked in confined spaces. This may be considerable if there is much synovial swelling.

If damage were directly related to synovial loading, then sites of tendon disruption would be exactly related to sites where tendons change direction. The observations could be brought into line with such a hypothesis in certain sites but not always so. Extensor pollicis longus shows the greatest change of direction at Lister’s tubercle, but although synovial and tendon disorganization is commonly found there it is greater distally where direct synovial pressure should be less. In the case of flexor pollicis longus, the greatest damage occurs at the point where the tendon winds round the carpal prominence into the thumb and is found to be greatest on the deep (i.e. concave) surface. But the incidence is much less than that of the comparable flexor digitorum profundus tendons.
The variation in damage to the individual extensor digitorum tendons may also be linked with loading due to change of direction. The tendons to the index finger run in a straight line from forearm to finger with only dorsiflexion as a direction change when the hand is in a position of function. Because of the ulnar deviation of the wrist in function, the more medial tendons have an ulnar as well as dorsal change, greatest in the little finger. The degree of direction change is thus comparable to the incidence of tendon lesions.

Damage secondary to synovial loading obviously occurs, and friction in the presence of a diseased synovium can be seen to be a significant factor, within the palm. Although flexor profundus is more generally active than sublimis and therefore more continuously stressed, the lesions on the tendons also reflect the changes of direction; dorsally, under the flexor retinaculum, where profundus take the direct load of both tendons, and ventrally, at the entrance to the fingers, where sublimis takes the load of both.

At the termination of a synovial sheath the synovium normally rolls around from parietal to visceral layer as the tendon moves. Normal synovium has little bulk and efficient lubrication results in easy movement. In rheumatoid disease synovial bulk may be enormously increased and lubrication less efficient. Easy movement of the synovium is thus no longer possible; it will neither slip easily under the retinaculi nor will it move readily round the corner from parietal to visceral layer. Furthermore there is often a great deal of fluid and debris within the synovium. When a tendon is pulled under a retinaculum under load, the passage of the diseased, thickened synovial sheath is difficult because of mechanical blocking. Thus, synovium and contents tend to be forced distalwards to the blind synovial end, which is thus subjected to a pump-like action with increased pressure on itself and the underlying tendon (Fig. 2).

As the end of the synovial sheath is the site of greatest damage, no matter what the tendon, it would appear that such an effect distal to the nearby direct tendon-loading zone could be the cause of so much tendon damage. It is interesting to note that, at the metacarpophalangeal joint of fingers, the site of maximal bone damage is related to a mass of synovium underlying the collateral ligaments. Here a similar pumping action on hypertrophic synovium can be postulated, as collateral ligaments move over synovium filling the saucer-shaped side of the metacarpal heads.

The frequent damage observed in extensor carpi ulnaris compared with the frequency of synovitis could be due to direct trauma from the distal end of the ulna, particularly where this is disorganized. The tendon moves considerably in relation to the bone, lying dorsal to it in supination but slipping round to the medial border in pronation. It and extensor digiti minimi are the only tendons directly related to the ulnar head. But, conversely, severely disorganized bone does not necessarily lead to tendon damage.

There is no evidence that spicule formation is an important factor in tendon disruption. In both cases in which such bone formations were found, there was thick synovium and tissue separating the structures, and the disruptive forces could have been the same for bone and tendon.

Summary

(1) Sites of tendon damage secondary to rheumatoid tenosynovitis are described in the hand as found at surgery.

(2) Certain tendons and sites are found to be more vulnerable to the disease than others, e.g. flexor digitorum profundus at the distal end of the palmar synovial sheath and retinaculum. The main sites in all tendons are distal to the wrist retinaculi or proximal to the digital fibrous flexor sheaths.

(3) The significance of possible causes of the changes are discussed.

(4) The incidence of tendon damage, particularly in the palm, is greater than often appears at routine clinical examination, because of the presence of a thick concealing palmar aponeurosis.

We should like to thank those colleagues who asked us to see the patients on whom this study was made, particularly Drs J. T. Scott, A. St.J. Dixon, H. Goodman, and A. C. Elkin.

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**FIG. 2** Diagrammatic representation of the proposed mechanism of tendon damage at the sites found. $P$—point of maximal pressure. $F$—collected synovial fluid and debris. $G$—granuloma or other tendon damage.
Fig. 1 was drawn by Mr. Frank B. Price.
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