TENDON SURGERY OF THE HAND

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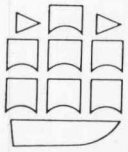

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Preface

Tendon repair is a *sine qua non* for the functional recovery of a wounded hand. Also, the disabilities of afflictions such as rheumatoid arthritis are directly linked to the destruction of tendons, whose repair can then produce spectacular recovery. Finally, permanent motor paralysis can be corrected by transfers of intact motor units into the paralysed musculotendinous groups that are inert.

Tendon surgery is difficult. Adequate time and care are necessary to obtain satisfactory results. Not only a general knowledge of hand surgery is necessary, but a special knowledge of the anatomy, physiology and pathology of tendons.

Well-known authors from several continents, for the most part members of GEM, were willing to be associated with this work. We have been able to include some very recent articles concerning the situation, thereby updating our work.

Since it is impossible to thank the twenty-one authors individually, I would like to express my sincere thanks to all for their precious collaboration.

My thanks also to Dr F. Cagli, scientific consultant in my service, as well as to Dr Olivier Reinberg, resident in plastic surgery, who translated several articles, particularly those written by me.

Finally, I thank my friend, Professor Raoul Tubiana, Director of Monograph Editions of GEM for entrusting me with this work, l'Expansion Scientifique Française and Churchill Livingstone for their care taken and Mr John Hueston of Melbourne for his great help with the English edition.

As a personal note, I would like to underline the great progress that has been made since the publication of my first book in 1952 'Chirurgie réparatrice et fonctionnelle des tendons de la main'. Some chapters are still valid, but many others are obsolete, and it is very rewarding to have been the author and editor of its updating twenty-seven years later.

I hope that our work will serve its purpose.

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Contents

1. *An Introduction to Tendon Surgery*

C. Verdan

Just as the primary function of a muscle is to contract and then to relax, so the essential requirement of an intact tendon is to glide. Our main concern with tendon repairs will always be to get them moving, for an adherent tendon repair is a total failure. This state of affairs may be considered sometimes as a temporary stage, to be followed by a subsequent tenolysis, but, as a rule, our aim will be to obtain not merely a solid tendon, but a gliding one. The gliding apparatus, which plays so essential a part in every planning of repairing methods varies at different levels, depending on the amplitude, the direction and the situation of each tendon in relationship to bones and joints in the various anatomical arrangements of the forearm and hand. The sheaths and pulleys occur wherever tendons pass across a concave joint line—on the dorsum of the wrist or on the volar aspect of the fingers and wrist. The gliding system is then reinforced with additional fibrous pulleys, the most powerful being the palmar carpal retinaculum. At different levels, the force exerted depends on shifts of direction and the gliding amplitude; which, for the long flexor tendons is 4 cm at the base of the fingers and more than 8 cm at the wrist (Fig. 1.1).

The *nutritional system* of tendons surrounded with paratenon is supplied along the whole net of connective tissue, while the vascular organization of the free tendons within the sheaths is far more complex. This blood supply has a great importance which has just recently been fully recognized. Professor Winckler will deal with the vasculature of tendons in his contribution to this monograph.

Mr. Lindsay and Dr Potenza deal with the subject of healing in tendon injuries and their experimental works appear later in the monograph. We want to pay tribute to Marc Iselin, as he was the first, together with Lafaury, to describe the phenomenon of degeneration of cut tendons: yellow degeneration, lysis, hyaline degeneration or tendinoma, and now it is clear that these changes seem to be linked with disturbances of tendinous vascularity, as Peacock, Caplan *et al.*, Urbaniak *et al.*, and others have also confirmed.

Special attention has been given to the repair process after tendon suture and the two theories of such repair. The theory of *axial healing*, by which the tenocytes of the two tendon ends proliferate longitudinally, and the theory of *peripheral healing*, whereby new fibroblasts are derived from vessels invading the tendinous defect from the periphery, that is either from the paratenon or the synovial sheath. This problem is fundamental in endosynovial healing, since granulation tissue carrying vessels will necessarily fill all available space between the sheath and tendon and therefore create adhesions, sometimes large ones, that tend to spread far beyond the tendinous callus itself.

This peripheral healing process has been demonstrated. Axial healing has also been demonstrated but depends upon a blood supply from both cut ends. If these tendon ends are not perfectly opposed, fibres will fasten themselves onto local fixed points. Recent work of Weeks stresses the role played by hyaluronic acid in changing the order and organization of collagen fibres and of dissolving adhesions. In this respect, the synovial fluid secreted within the sheaths contains the hyaluronic acid and can thus prevent adhesions wherever the sheath is still intact, whereas adhesions are more easily formed if the synovial sheath has been opened.

Matthews and Richards, 1974, have shown experimentally on the dog's paw that flexor tendons have an active potential for repair and remodelling when partially cut and maintained in an intact sheath, without the formation of adhesions. But we know that this is not the case when the tendon has been totally cut and thereafter sewn together in a sheath which must be opened in order to repair the tendon.

The same authors have later (1976) also demonstrated on rabbits that the pathogenesis of adhesions appears to depend on other factors than the healing characteristics of the tissue itself, namely: *opening* or *excision* of the sheath, *inserting of suture material*, *immobilization*. The immobilization alone has no influence on adhesion formation if the sheath is intact and if the tendon is not completely severed. The trauma caused by the suturing of an incompletely cut tendon, which is subsequently reintroduced into a normal sheath does not create adhesions. The excision of an area of sheath-overlying the injured tendon does not in itself affect the reparative activity of the tendon cells, and the synovial layer of the sheath is rapidly reconstituted. The combined effect of splintage and suture in an intact sheath also allows healing mainly by tendon cell proliferation and mild to moderate adhesions were seen in one half of the specimens. These adhesions are absorbed once splintage is discarded. Combining suture of an incompletely severed tendon and sheath excision results in an adhesive response which contributes to the repair of the tendon defect. But the adhesions also tend to absorb or remodel in such a way as to allow gliding. In the same way, immobilization and removal of the sheath of the traumatized tendon leads to mild and transient adhesions.

However, when all *three variables* were introduced *simultaneously*, that is, suture, sheath excision and splintage, the result was a profuse and lasting adhesive response by the ingrowth of fibroblastic repair tissue derived from the peri-sheath layers. The intrinsic repair response of the tendon cells was suppressed.

In summary, these experiments confirm the classical clinical

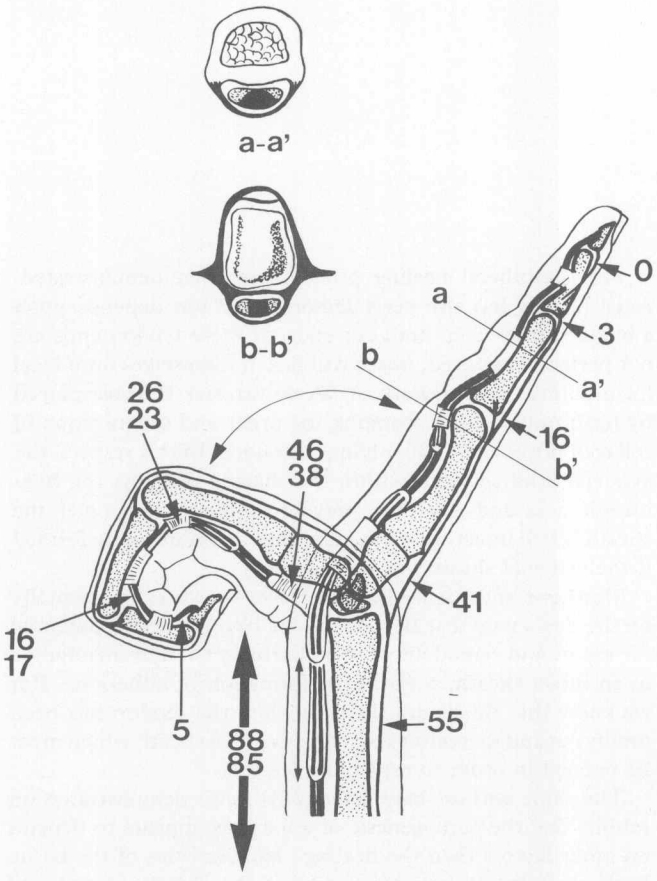

Figure 1.1
Diagrammatic sagittal-section of the hand, passing through the middle finger and the wrist, in the extension position and complete flexion of all articulations. The scale of movement of the flexor and extensor tendons is expressed in millimetres, at different levels. For the flexors, the upper figure refers to the superficial tendon, and the lower to the deep.
a–a′: section across the pulley of the proximal phalanx. The superficial flexor is divided into its two lateral strips and passes in depth, in contact to the phalanx. The deep flexor has thus become superficial and continues alone until it leaves the middle third of the middle phalanx.
b–b′: section across the metacarpophalangeal volar plate and the first annular pulley. The deep intermetacarpal ligament does not reach the bone but merely involves the volar plate. It should be called the 'interglenoidal ligament'. This point where the fibrous elements cross, on which the hand grip is concentrated, is particularly resistant.

observation of the dominant influence of iatrogenic factors on the healing of injured digital flexor tendons.

Studies of *tendon graft vascularity* show vessels coming from the outside, all along the graft, in a perpendicular direction to the tendon. However Teneff and Fonda have observed an anastomosis appearing towards the 15th day between the perpendicular and the longitudinal networks, the latter coming from both tendon ends. Later, the longitudinal circulation prevails and the perpendicular vessels are gradually absorbed. That means that if the graft is to recover full mobility, a system of mesotenons will have been shaped, as being the only physiological device capable of giving an adequate permanent blood supply without interfering with the motion of the tendon itself.

Urbaniak, Bright, Gill and Goldner (1974), have shown in an experimental study on dogs and chimpanzees, that vessels bearing adhesions in flexor tendon grafts form all along the new tendon, as well as after a two stage procedure with Hunter rod, as after a conventional one-stage grafting, the latter giving in general more, and denser, adhesions which restrict movements. One year after a two-stage grafting procedure, they could observe a well developed loose reticular mesotenon especially on the dorsal surface of the graft. This neo-mesotenon transmitted vessels to and from the tendon and permitted gliding, in almost the same way as with normal anatomical conditions.

A great deal of micro-anatomical studies of the vascularization of tendon and sheath have been published during these last two years. They have brought an accurate knowledge of the vessel network inside and outside the flexor mechanism and a better comprehension of the healing processes, but none of them have really permitted any advancement of the formerly known methods of repair. The most baffling experiment on rabbits has recently been carried out by Lundborg (1977) who has been able to show that a fresh autogenous flexor tendon graft, consisting of two segments sutured together end to end and transplanted freely in an intact knee joint capsule, do heal. This is truly a tissue culture *in vivo*. The two segments show a fibroblastic repair and after three weeks the resistance to rupture seems as good as after a conventional suture, without any adhesion to the synovial layer of the knee capsule.

Lundborg's interpretation is that these rather impressive and technically excellent histological photos represent an intrinsic proliferation of the tenocytes, which is in contradiction to the theory which militates for extrinsic support. He also believes that the surrounding synovial fluid was able to nourish the graft. Taking into consideration the technical quality of these photos, it is the author's opinion that the fibroblastic proliferation represents a seeding of cells originating in the synovial of the knee, analogous to the free recellularization of lyophilized heterogenous grafts described by Potenza.

The remaining tenocytes inside the tendon, along the collagen bundles, do not die. But the proliferation on the surface, especially over the cut ends and the cells invading the suture line, seem to have their origin, either from the epitendinous layer or from free moving cells in the synovial fluid.

It will be quite interesting to know the result of the new series of experiments announced by Lundborg, with isotopic marked fibroblasts, with which it seems possible to differentiate these different origins.

Whatever the situation may be, adhesions are until now certainly not a technically avoidable accident, but rather a consequence of the physiological healing process. As long as we have no technical solution to the problem of accurately maintaining the two cut ends in an intact synovial sheath without interfering with the blood supply (as we had searched for in 1951), adhesions will remain a biological inevitability. The problem

consists of knowing the best means to facilitate their dissolution and disappearance, after they have occurred. A pharmacological fibrinolysis, local or by a general administration, is still largely a dream and so far the only effective treatment of adhesions is physical activity by the patient himself, supervised by a physical therapist, often with the help of some mechanical traction splintage. We will deal later in the monograph with the problem of tenolysis, or surgical adhesiotomy in this respect.

The studies of Mason and Shearon, and subsequently Mason and Allen in 1933, demonstrated that the tendinous callus undergoes many changes; a softening about the 10th day, then a hypertrophy if motion is undertaken too soon, with the implication that tendon sutures must remain at rest for three weeks. This is the standard time for the healing of a tendon. After that period, patients may be asked carefully to move their digit and increase little by little the amplitude of these movements. Quite often, this period of rest has to be prolonged rather than shortened, chiefly with the extensor tendons.

In principle, therefore, the most important thing to aim at is not the strength of suture material, but an exact opposition of the cut surfaces. To this end sutures will have to be as fine as possible, and best tolerated by the surrounding tissues.

Certain basic distinctions are necessary—such as between a *new wound* and a *scarred wound*, and between *tidy wounds* and *contused* wounds.

With old scarred wounds, one has time to consider the pros and cons before deciding upon a secondary operation.

With new primary wounds, the following three basic questions ought to be posed:

'Am I in a position to handle such a case?'
'Have I the necessary equipment?'
'Have I the time to deal with it?'

If every physician called to undertake tendon surgery would sincerely answer the foregoing questions, results would certainly become more favourable. We feel it is mandatory to raise such questions for the sake of patients with mutilated hands. A tendon repair is a difficult undertaking and it should only be entrusted to a competent team, with plenty of time and in the proper setting.

Unfortunately there is a confusion between a 'primary operation', meant as 'emergency surgery', made by anyone, anywhere, anyhow, and a 'secondary operation' as being a specialized surgery, made in a well organized centre by highly trained surgeons. If we want progress in tendon surgery, we must at all costs establish the importance of emergency surgery and organize ourselves in such a manner that it be done by competent surgeons, capable of teaching our residents the best techniques. Improvisation, still too frequent in emergency units, must henceforward be avoided.

May the following chapters safeguard the living strength of our manual workers, the masterpiece of all instruments, the hand.

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ANATOMY AND PHYSIOLOGY

2. *Normal Anatomy of the Flexor and Extensor Tendons of the Hand*

G. Winckler

The palmar surface and the dorsal surface of the hand, each presents its own special characteristics.

PALMAR SURFACE

The palmar skin, well supplied with sweat glands and crease lines, is not very mobile. The subcutaneous fat, which is quite thick except in the thenar eminence, is traversed and compartmented by strands of connective tissue which connect it directly to the superficial palmar aponeurosis. There is thus very little fascia superficialis or subcutaneous areolar tissue enabling it to glide over the deeper layers.

This palmar surface can be divided into six regions with their own types of morphological and functional (and hence pathological) behaviour.

1. *The thenar eminence* is crossed by the tendon of the flexor pollicis longus muscle. The tendon follows an undulating course, first doubling back round the trapezium, then travelling the length of the first phalanx, against which it is firmly held by a fibrous flexor sheath (Fig. 2.1).

The tendon therefore passes through two zones, one of which is loose within the muscles of the thenar eminence, the other is constricted within the fibrous sheath.

2. *The hypothenar eminence*, or heel of the hand, is a support where the subcutaneous fatty layer, which encloses the palmaris brevis muscle, is quite well developed. The hypothenar muscles are skirted on the radial side by the flexor tendons of the little finger, surrounded by their common synovial sheath (Fig. 2.1).

The deep terminal branch of the ulnar nerve and the deep branch of the ulnar artery pass between the abductor digiti minimi and the flexor digiti minimi muscles.

3. *The carpal tunnel*, which extends for four centimetres beyond the distal crease of the wrist, is crossed by the solid flexor retinaculum. The tunnel encloses the flexor digitorum superficialis and profundus and the flexor pollicis longus tendons arranged on two levels as well as the median nerve. The tendon of the flexor carpi radialis muscle lies deeper, reaching the base of the second and third metacarpal after passing through an osteofibrous tunnel and synovial sheath of its own.

This osteofibrous carpal tunnel is a *constricted zone* designed for containing and allowing to glide the various tendons with synovial sheaths during wrist flexion.

4. *The palmar region*, between the thenar and hypothenar eminences, is triangular in shape. It is a *loose zone*, covered by the middle portion of the palmar aponeurosis, which is made up of longitudinal pretendinous fibres which are firmly fixed by perforating sagittal septa on each side of the metacarpophalangeal joint. Transverse fibres, deep to the longitudinal fibres, are more proximal than the superficial transverse (natatory) ligament.

Between the superficial transverse ligament, the pretendinous bands and the natatory ligament, situated slightly behind the metacarpo-phalangeal crease, are *three pads of fatty tissue*. These pads, with their abundant lamellated corpuscles, are traversed by the collateral nerves of the three last interdigital spaces and the beginning of the digital arteries, coming from the superficial interosseous arteries.

The distal palmar crease permits localization in depth of the metacarpophalangeal joints, which are easier to locate, however, on the dorsal surface.

The superficial palmar arch traverses the palmar region of the hand, obliquely from the lateral margin of the pisiform bone towards the second interdigital space, directly under the central portion of the palmar aponeurosis. At a deeper level are the superficial branches of the ulnar nerve and those of the median nerve; finally come the tendons of the flexor digitorum

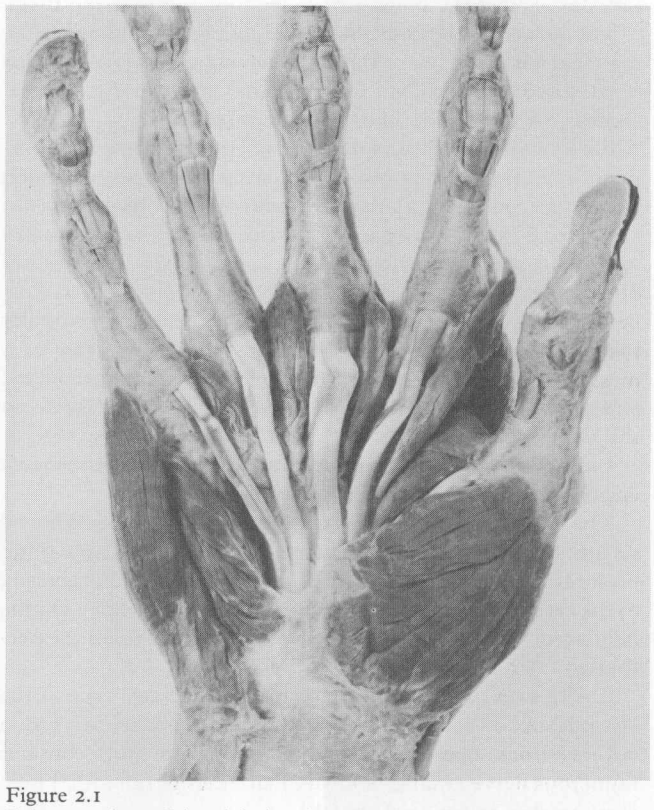

Figure 2.1
Palmar surface of the right hand.

superficialis and flexor digitorum profundus muscles arranged fanwise at their two levels.

In this loose zone the tendons are surrounded by *paratenon*. This is areolar connective tissue lying in several layers of planes and through which pass a large number of vessels. These arise from the neighbouring arches and after forming longitudinal or transverse loops, send a blood supply to the tendons. In this region only the tendons of the flexor superficialis and flexor profundus of the little finger are accompanied by a digitocarpal synovial sheath (Fig. 2.1).

Finally it is from the tendons of the flexor digitorum profundus muscle that the lumbrical muscles arise.

At a deeper level this palmar region, through which the tendons, vessels and nerves pass, is separated from the dorsal and palmar interosseous muscles, the deep palmar arch and the deep terminal branch of the ulnar nerve, by the *deep palmar aponeurosis*. This aponeurosis, which is thin, is strengthened at the level of the metacarpophalangeal joints by the *deep transverse ligament*, which separates the lumbricals and the neurovascular bundles from the interosseous muscles.

5. *The digital canals*, in which the flexor tendons of the fingers have their course, are *constricted zones* starting at the level of the metacarpophalangeal joints of the four fingers. The canals are characterized by the presence of *fibrous sheaths*, particularly well developed in the first and second phalanges but much weaker at the level of the joints with some significant strengthening elements, the cruciate and oblique ligaments. These digital fibrous sheaths, acting as pulleys, maintain the tendons against the bone surface without compressing them.

The *synovial sheaths* facilitate gliding of the tendons in the digital canals. There are three digitopalmar synovial sheaths (for the second, third and fourth digits) and two digitocarpal sheaths for the thumb and the little finger.

Each synovial sheath, essentially of connective tissue, comprises a visceral layer or *epitenon* adhering closely to the tendon and a parietal layer, lining the wall of the fibrous sheath, separated by a synovial space. The visceral layer has extensions into the inside of the tendon. These are the *endotenon*, a series of septa separating the fibre bundles from each other. Just before the end of the flexor tendons, a *mesotenon* of triangular shape joins the two layers of the synovial sheath, acting as a mesentery for the passage of vessels. Other more attenuated structures, the vinculum breve and vinculum longum, perform the same function (Fig. 2.2).

In brief, the epitenon, endotenon, mesotenon or the vincula enclose a significant number of vessels.

The tendons, covered by the visceral layer of their synovial sheath, end at the base of the distal phalanx in the case of the perforating tendon (deep flexor) and on the middle phalanx in the case of the perforated tendon (superficial flexor). On the palmar surface of the middle phalanx the two halves of the perforated tendon decussate to form a chiasma.

6. *The pulp region* corresponds to the palmar surface of the last phalanx of each digit. Here the extremely sensitive skin covers a thick, dense cellular fatty tissue. This pulp contains numerous nerve endings and vascular endings (glomi). There is no tendon or synovial sheath.

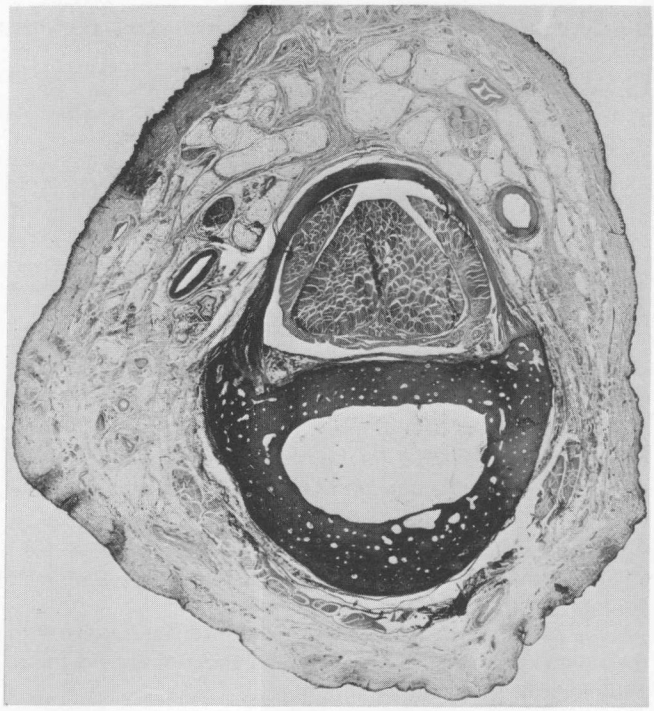

Figure 2.2
Cross-section of an adult finger. Note the phalanx on which the transverse ligament of the fibrous sheath is fixed; the synovial space; the tendon of the flexor digitorum profundus accompanied laterally by the two bands of the flexor superficialis. On the dorsal surface of the phalanx, the outspread fibrous complex of the extensor, the interossei and the lumbrical.

DORSAL SURFACE

The skin on the dorsal surface of the hand is thin, delicate and almost transparent, since the branches of the dorsal venous network of the hand are visible. Above all it is mobile. Hairs are present on the dorsal surface of the hand and of the first phalanx of the four last digits.

The fascia superficialis is very thin and in the underlying loose connective tissue lie the superficial veins and the cutaneous branches of the radial and ulnar nerves.

An aponeurosis or dorsal fascia of the hand separates these cutaneous layers from the extensor tendons.

Distal to the wrist, these tendons cross two well-defined zones.

A constricted zone, marked off by the *extensor retinaculum*, which helps to form the six osteofibrous tunnels in which the tendons are surrounded by synovial sheaths with the same general characteristics as those of the palmar surface.

A loose zone, taking up the dorsal surface of the hand, where three groups of tendons can generally be seen (Fig. 2.3).

The tendons for the thumb lie in the first and third tunnels (abductor pollicis longus, extensor pollicis brevis and extensor pollicis longus). They form the anatomical snuffbox, crossed by the median vein of the forearm accompanied by the superficial branch of the radial nerve, while deep below, against the bones, lies the radial artery.

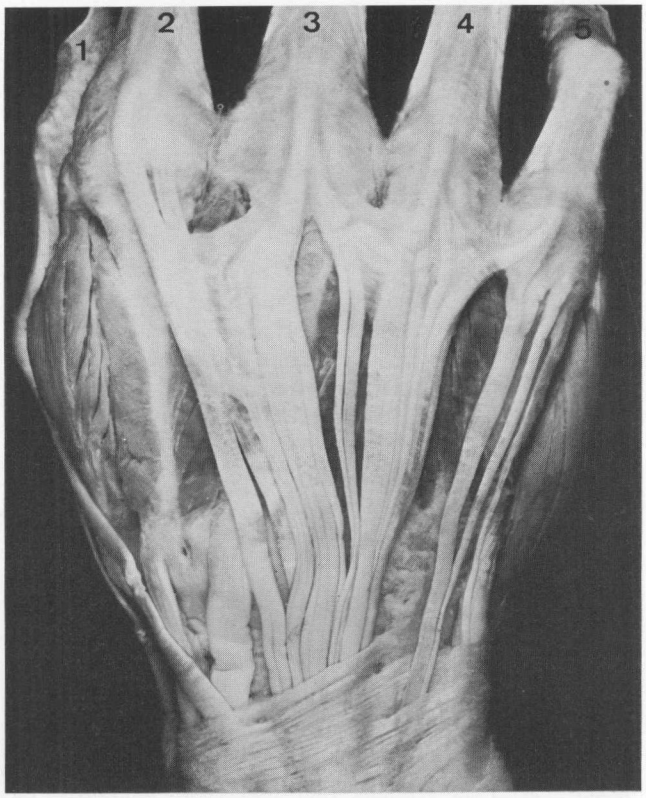

Figure 2.3
Right hand. Dorsal surface. A view of the various extensor tendons.

The two wrist extensor tendons which terminate on the base of the second and third metacarpals have passed along the second osteofibrous tunnel.

Finally, the tendons of the various digital extensors and the tendon of the extensor carpi ulnaris muscle take their course along the fourth, fifth and sixth furrows.

The dorsal surface of the digits constitutes a special region. It serves for the final passage of the extensor tendons.

The tendons intended for the thumb end on the base of the first metacarpal and the base of the first and second phalanges. As for the extensor digitorum tendons, they divide into three, then insert on the phalanges, after receiving the tendons of the interossei and the lumbricals.

The extensor carpi ulnaris tendon inserts into the base of the fifth metacarpal.

Starting from the extensor retinaculum, as soon as the synovial sheaths terminate, all these tendons are surrounded by paratenon. This loose connective tissue, which extends connective-tissue septa between the fibre bundles of the tendons (endotenon) contains the vessels which ensure nutrition of the tendons. These vessels come from the dorsal metacarpal arteries or from the digital arteries, depending on the level.

THE BLOOD SUPPLY OF THE TENDONS

After Koelliker (1850) it was accepted for some time that *adult tendons* were structures without vessels whereas *young tendons* were vascularized. Nutrition was believed to take place by imbibition through the synovia of the synovial sheaths or the interstitial fluid of the neighbouring connective tissue.

Sappey (1866) and others (Ludwig & Schweigger-Seidel (1872), Arai (1907), Rau (1914), Mayer (1916), Dychno (1936), Edwards (1946), Braithwaite (1951), Brockis (1953), Peacock (1959), Lang (1963), Guse, Erler & Loetzke (1963), Smith (1966) and Dörfl (1969)) demonstrated, in most cases by means of vascular injections, that the vessels destined for the tendons had their points of origin and entry in different places: the musculotendinous junction, the osteotendinous joining, the extrasynovial regions of areolar tissue with paratenon and the synovial regions in the constricted areas, with synovial sheaths, mesotenons or vincula.

According to Edwards & Brockis there are intrinsic intratendinous, interfascicular vessels exhibiting an internal pattern of longitudinal vessels joined by transverse anastomoses making their way through the interfascicular connective tissue (endotenon).

When the vessels have been injected through the ulnar artery with gelatine containing Indian ink, they can be observed with the naked eye, with a magnifying glass and, if necessary, under the microscope.

The tendons of the hand pass through alternate *loose areas* where they are surrounded by areolar connective tissue (paratenon) and *constricted areas* (synovial sheaths).

1. In the *dorsal and palmar loose areas* the arterial vessels coming from the neighbouring deep arteries (dorsal metacarpal arteries, collateral digital arteries or superficial palmar arch) reach the paratenon on each side of the tendons. They generally divide into a T several times. The longitudinal branches remain on the surface in the paratenon of the interfascicular furrows. The arteries on the two sides of the tendon anastomose, thus forming a superficial vascular network of very loose mesh (Fig. 2.4).

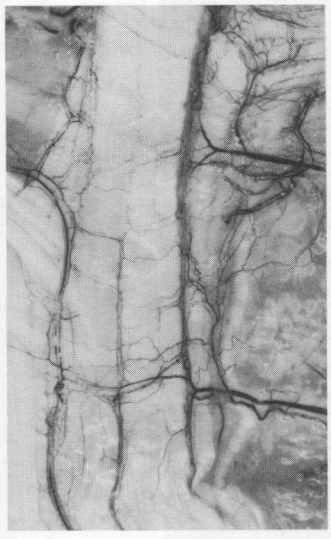

Figure 2.4
Arteries of the dorsal face of the extensor tendon of the ring finger (injected with dye).