2. DUPUYTREN'S DISEASE AND THE ANATOMY OF THE PALMODIGITAL APONEUROSES

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Baron Boyer stepped into immortality by claiming, around 1830, that surgery had reached the limit of its possibilities. Another of his unfortunate phrases was 'crispatura tendinum', which he used in describing the hand lesion which results in fixed flexion of the fingers. Dupuytren dissecting one such hand found bands which ran *alongside* the affected fingers. In 1832, he found that a retraction of the aponeurosis was in fact the main lesion in the disease which later was to bear his name. According to J. Windsor, Henry Cline demonstrated (as early as 1808) the favourable effect of fasciotomy and Astley Cooper in 1822 followed Cline's example. Does this mean that Dupuytren's fame is undeserved? Whatever the case may be, he believed that the disease was the result of trauma and that it attacked the aponeurotic structures.

But in 1834, Goyrand challenged these two facts. He denied that trauma was the main ætiological factor, and claimed that the retractile fibrous structures which cause fixed flexion of the fingers are independent of both the palmar aponeurosis and the tendons.

On the first point, Goyrand might well have been right. The real ætiology of Dupuytren's disease remains a mystery.

Must we now challenge Dupuytren's theory on the second point as well? This is just what Hueston has done in an important work published in 1963. According to this Australian surgeon, the nodules and the fibrous bands excised are quite separate from the palmar aponeurosis. In fact he believes them to be new fibrous formations, developing initially between the skin and the aponeurosis. They arise from none of the recognized anatomical structures, and constitute what he calls 'Dupuytren's tissue'.

As a result, surgical excision should not be determined by anatomical factors—a fact which rather indicates limited or selective resections.

Is this opinion wholly or partially true? This we would like to discuss as it is of primary importance to the surgeon.

If the lesions are independent new-formations, unrelated to the recognized anatomical elements, their excision will be haphazard and will be concentrated on avoiding neurovascular structures. If on the contrary the lesions develop in a well defined anatomical structure, e.g. the digitopalmar aponeurosis, their excision will be carried out in a systematic, orderly way. There will

be no unexpected anomalies, and only the uninitiated will have unpleasant surprises.

We must see first how much can be learned from microscopical examination. In this respect the works of Meyerding, and those of Nezeloff and Tubiana are precise and tend to agree. These authors have shown that two very different types of lesions are encountered in Dupuytren's disease.

In some areas, one finds fibroblastic activity, mitoses, little collagen, a disorderly arrangement with wavy or concentric patterns. In others, there is a paucity of cells, dense collagen arranged in parallel fibres and forming actual retractile bands.

One may accept the theory, proposed by a number of authors, that the first type of lesion represents the early, progressive stage of the disease, while the other represents a late, stabilized stage. As this theory has never yet been confirmed, we feel free to suggest an alternative one: the cellular, nodular lesion and the collagen bands are two forms of the same disease occurring in different structures.

The acellular, collagenous forms are very retractile, and are the ones responsible for flexion of the metacarpophalangeal and interphalangeal joints. They lead to the formation of fibrous bands over which the skin of the palm remains thin, supple and mobile, except for a few millimetres proximal and distal to the flexion creases.

The nodular forms of Dupuytren's disease are quite different. Histologically, they present as a dense tissue, rich in cells and showing no organization. The picture is the same in the palmar and digital nodules, in the knuckle pads and in the plantar nodules of Ledderhose. In the hand, the round or oval shaped nodules adhere closely to the skin which becomes callous over the lesions. These are pretendinous and are usually found in areas where the fatty tissue is thick, between the interphalangeal creases, between the proximal I.P. and the M.P. creases, and only rarely proximal to this.

In our experience, as we shall see later, the nodules usually form in areas where no aponeurotic structure would be found in the normal hand. They are but slightly retractile. If after the removal of a large phalangeal nodule the finger still does not achieve complete extension, it is essential to look elsewhere (and we shall see where) for a band the severing of which will ensure a good result. Depending on the cases, any combination of these two distinct forms may be encountered. Some cases are

primarily nodular, and flexion is not pronounced. In other cases, the nodules are virtually absent and the strongly retractile bands prevail. Type D of the disease, described by Michon and Tubiana, is characterized by the presence of these bands which we shall describe later. And finally, there are cases where both forms are encountered, in equal or unequal proportions.

We believe that the bands and nodules do not represent two different stages in the course of the disease, but rather two distinct forms, which originate in different tissues. When the disease develops in a well defined sheet of aponeurosis, the lesion takes the form of dense, retracted fibrous bands, with abundant collagen and a paucity of cells. When it affects the adipose tissue, and spares the aponeurosis, the nodular form prevails. The fat disappears, the cellular fibrosis invades the deep layer of the skin and spreads inwards towards the underlying aponeurosis. In other words, we believe that the two forms which differ macroscopically (nodules or bands) and histologically, represent lesions in different tissues: in the fatty tissue for the nodules, and in the aponeurosis for the bands.

We agree with Hueston's view that the nodular lesion is independent of the aponeurosis. It shows a preference for the pretendinous zones where there is no aponeurosis, at the base of the phalanx, distal to the first interphalangeal crease, or the area between the proximal digital crease and the M.P. point. Much less frequently, a nodule may be found in the palm, proximal to the inferior palmar crease, between the skin and aponeurosis. Its adhesion to the aponeurosis is only secondary and comes from intimate contact with that structure.

When however, the disease attacks the aponeurosis, retractile bands are formed. These take their origin from the true aponeurosis and always follow a well defined course. We do not agree with Hueston when he says that they cannot be submitted to a rigid classification. The sclerotic process may selectively affect certain aponeurotic elements which we shall describe more fully later. Whether the lesions are found in frontal or sagittal, superficial or deep structures, the relations to the neurovascular elements are classical and anatomically predictable.

It is true that the proportions of the two types of lesions in one case may vary. There are essentially nodular forms, essentially retractile forms, and mixed forms with both nodules and retractile bands. Surgically the essential step is the excision of these bands which are responsible for flexion of the fingers. This excision is performed according to a technique based on the normal anatomy of the palmodigital aponeuroses.

This is why we think it is important to consider the anatomy of these structures. We studied these on the hands of adult corpses which we dissected with the utmost care. One of them happened to show Dupuytren's disease limited to the 5th finger. More than 50

colour photographs were taken at various stages of the dissection. The five figures shown in this article are drawings faithfully reproducing five of these photographs. They are neither compositions nor compound drawings. We have compared this study with our operative findings in more than 500 aponeurectomies.

1. ANATOMY OF THE MIDDLE PALMAR APONEUROSIS

By middle palmar aponeurosis is meant the central part of the superficial palmar aponeurosis, and excluding the aponeuroses of the thenar and hypothenar eminences. This middle palmar aponeurosis is triangular with the apex pointing proximally. It occupies the middle part of the palm between the two eminences. It is made up of two types of structures, the frontal and the sagittal.

The frontal plane (Figs. 2.1, 2.2) starts at the carpal annular ligament and is continuous with the terminal fibres of the palmaris brevis tendon. Proximally, it is narrow, dense and of uniform thickness. As it spreads out towards the root of the fingers, it gradually divides into four reinforced pretendinous strips. In the distal part of the palm, three frontal sheets with transverse fibres join the pretendinous strips, and close the angle of divergence of these strips. Their fibres, which constitute the superficial transverse ligament, are quite lax and run in a deeper plane than the vertical fibres of the pretendinous strips. Skoog stresses the point that the superficial transverse ligament is invariably spared in Dupuytren's disease, and we agree with this observation. The more proximal fibres of the superficial transverse ligament do not completely obliterate the angles of divergence of the pretendinous strips. In other words, these strips begin to diverge just proximal to the transverse fibres. There are triangular spaces filled by a more or less dense fatty tissue. These spaces with the sharp angle pointing proximally are limited laterally by the borders of the pretendinous strips, while their base is formed by the proximal border of the superficial transverse ligament.

The distal border of the superficial transverse ligament in the gaps between the pretendinous strips is always well defined. It forms the proximal border of a fatty zone which is lobulated, lax, slightly prominent and easily recognizable in the normal palm between the distal palmar crease, the axis of adjacent fingers and the commissures. In this fatty zone can be found the arteriovenous bifurcation and the two collateral nerves which more proximally were running under cover of the superficial transverse ligament.

In Fig. 1, the fatty lobules which have been left _in situ_ between the index and middle fingers conceal the artery and the internal collateral nerve of the index.

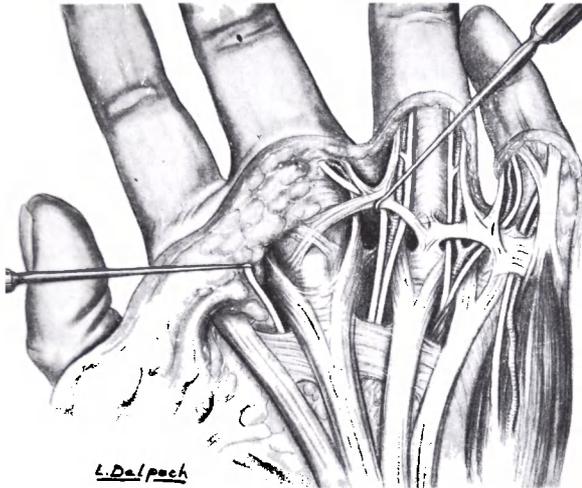Let us now consider the fate of the pretendinous strips

Figure 2.1
Palmar aponeurosis displayed after removal of skin and subcutaneous tissue. The pretendinous bands and their bifurcation as seen, the transverse superficial palmar ligament and the natatory ligament. Hooks retract the digital nerve and natatory ligament to show the course of the lateral and medial sheets to the middle finger.

distal to the superficial transverse ligament. This is mentioned in none of the anatomical descriptions that we found. In the classical diagrams, they are shown continuing down to the anterior aspect of the first phalanx. This, we think, is totally inaccurate. In fact, at the level of the metacarpal, the pretendinous strip divides V-wise into two strips which twist round, plunge inwards and run close to the lateral aspects of the capsule of the M.P. joint. A few transverse fibres come to form a small triangle, the apex directed proximally, which fills the angle between the strips.

No vertical fibres are found running in this angle. The floor is formed by part of the fibrous sheath of the flexor tendons. More distally, opposite the base of the proximal phalanx, there is a complex, transverse aponeurotic structure, the interdigital palmar ligament, which we shall describe later. But there is one point on which we insist: the pretendinous strip, a palmar reinforcement of the superficial aponeurosis, does not continue longitudinally and in a frontal plane from the palm to the palmar aspect of the first phalanx. There is at the most a very loose, felty fibrous covering over the anterior aspect of the flexor sheaths. This lack of continuity in the fibres of the pretendinous strips is not generally known,
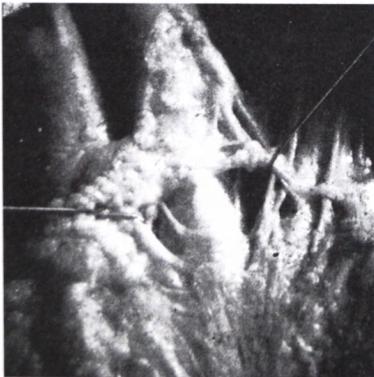

Figure 2.2
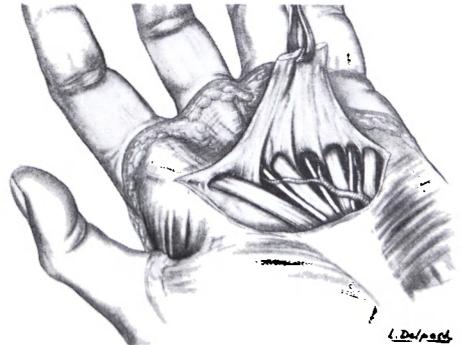Photograph from which Figure 2.1 was drawn.

Figure 2.3
The palmar aponeurosis is being dissected from proximal to distal and the superior borders of the septa of Legueu and Juvara are placed under tension and the 7 canals can be clearly seen.

but Thomine has described it accurately. There does exist, as he states, an 'aponeurotic hiatus' between the termination of the pretendinous strip and the palmar interdigital ligament, opposite the metacarpal head. According to Thomine, the fibres of the pretendinous strip end by 'being inserted in the dermis'. We do not agree with this. They terminate in fact by plunging into the depth of the hand after bifurcating. This does not exclude the existence of a few tracts between the dermis and the aponeurosis, such as are present all over the palm and especially at the flexion creases. We shall later consider the anatomy of the aponeuroses in this palmo-digital junction zone.

Let us now come back to the middle palmar aponeurosis of which we have only described the frontal structures. From the deep surface of the palmar aponeurosis sagittal lamellae run vertically and separate the tendons from the neurovascular compartments of the palm (Figs. 2.3, 2.4, 2.5). These are the classical 'partitions'

of Legueu and Juvara, formed by so-called perforating fibres which run from the deep surface of the middle aponeurosis to the anterior surface of the deep aponeurosis, which itself joins the metarcapals and covers the interosseous muscles. These structures are clearly identifiable on a transverse section (Fig. 2.9) going through the middle palmar crease. First one recognizes the metacarpals with, stretched in a frontal plane between their anterior crests, the deep palmar aponeurosis. In a more anterior plane, the flexor tendons are seen running in the axis of the metacarpals. Between the flexor tendons, anterior to the intermeta-carpal space, the nerves have already divided, but not the digital arteries and veins which divide much more distally, very near the commissure. In a more superficial plane is the middle aponeurosis. The fibrous partitions are eight in all, one on either side of the tendon to each finger. On the section, one recognizes the seven compartments limited anteriorly by the plane of the palmar
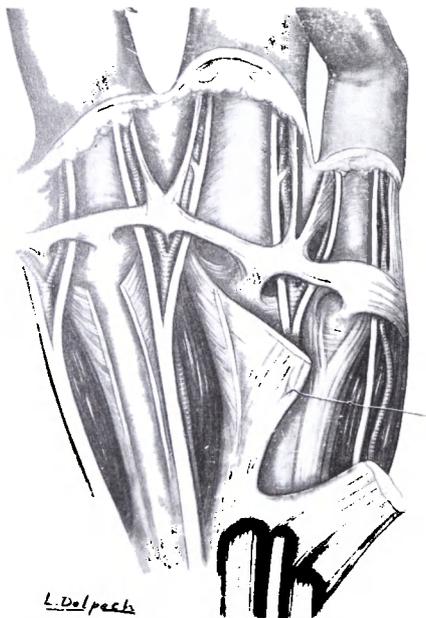

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Figure 2.4
The paratendinous septa of Legueu and Juvara have been divided on each side of the flexor tendons of the middle finger, so that the continuity of those septa with the lateral sheet of the finger is clearly seen. The important relation between the digital nerve and the fascia is shown with the lateral digital nerve to the middle finger passing anterior to this lateral digital sheet.

Figure 2.5
Photograph from which Figure 2.4 was drawn.

aponeurosis and laterally by the partitions of Legueu and Juvara. Four compartments transmit the tendons, and three transmit the neurovascular bundles and the lumbricals of the second, third and fourth interspaces. It will be noticed that the external neurovascular bundle of the index finger and the internal bundle of the 5th finger have no compartments of their own as they belong to the thenar and hypothenar regions respectively.

It is important to bear clearly in mind the inferior and superior borders of these partitions.

Proximally the falciform upper border begins well below the distal border of the annular ligament along a horizontal line which is about equidistant from the distal flexion crease of the wrist and the proximal digital crease. These partitions in fact never reach proximally beyond the superficial palmar arch and the tendinous origin of the lumbricals.

If the narrowed origin of the middle palmar aponeurosis is divided as proximally as possible, and if it is pulled down and freed from its connexions with the thenar and hypothenar aponeuroses, it is found that no deep attachments tie it down. Deep to it, one finds the superficial palmar arch and the division of the median nerve. Further down, folding of the middle palmar aponeurosis is restricted by the stretched sagittal partitions. The tendon and neurovascular compartments are exposed (Fig. 2.3). To lift the aponeurosis further, it then becomes necessary to divide the partitions down to their lower insertions. These insertions fuse with the pretendinous strips after their bifurcation on the lateral aspects of the capsules of the M.P. joints. We shall see later how the palmar aponeurosis, the interdigital palmar ligament and the digital aponeuroses become fused opposite the base of the first phalanx.

It is worth emphasizing the relationship of the tendons and the neurovascular bundles above and below the upper borders of the vertical intertendinous partitions.

Proximal to the origin of these septa the palmar aponeurosis forms a thick narrow sheet between the thenar and hypothenar eminences. The vessels and nerves are well superficial to the tendons so that when, at operation, this proximal segment of the aponeurosis is divided and the lateral margins freed, care is necessary to avoid the neurovascular bundles which completely conceal the flexor tendons. When the palmar aponeurosis is lifted the vessels tend to be lifted with it and, during the distal dissection, it is necessary to push them back to avoid injury in clearing the deep aspect of the aponeurosis.

More distally in the compartments between the septa the tendons diverge and the neurovascular bundles pass more deeply onto the same plane as the tendons.

2. THE DIGITAL APONEUROSES

The digital aponeuroses have been extensively studied over the years namely by Cleland (1878), Landsmeer (1949), Stack (1961), Thomine (1965). The most precise, the most complete and, to our mind, the most accurate is the description given by Thomine. Our observations differ from those of Thomine only in points of detail and our small disagreements are probably due to the fact that Thomine's studies are purely anatomical, while ours were influenced by our desire to explain the lesions observed in Dupuytren's

disease and to contribute to surgical technique.

A very skilful dissector, Thomine studied simultaneously a number of thin sections of the foetal fingers and described extremely fine sheets which are not identified at surgical exploration. For this reason, we think they are of little practical importance. This difference is particularly striking when we consider the palmar neurovascular bundle in the finger. Thomine attaches much importance to these structures, and we do not deny that microscopy can reveal the presence of a superficial fibrous sheet in front of the bundle, as well as a deeper sheet, these two sheets joining within the bundle and around it, and forming a sheath which is elliptical in cross-section. We remember that at one time a similar sheath was described for the sterno-cleido-mastoid muscle and for the muscles surrounded by the middle cervical aponeurosis. For the benefit of surgeons, Cunéo produced a more simple description which stressed the planes of cleavage of the neck rather than the actual aponeuroses which a good anatomist can always separate into relatively artificial layers.

Personally, as we always found this vascular sheath a rather weak structure, in the normal and in the diseased hand, we shall consciously neglect it.

For another reason, we shall not mention Cleland's ligament, a frontal structure which is described as running between the skin and the lateral aspect of the phalanges, and is supposed to separate a dorsal from a palmar compartment. We have never succeeded in identifying this ligament which, according to Thomine, can only correspond to the artificial association of distinct tissue structures.

In the middle of the palmar aspect of the first phalanx, between the proximal digital crease and the middle crease, (proximal I.P. joint) we only found a lobulated fatty tissue with scattered fibrous partitions and no organized structure. In a deeper plane, there does exist a clear plane of cleavage between this fatty tissue and the anterior aspect of the fibrous tendon sheath. Is this enough to claim the existence of a digital sheath which limits the fatty layer on the one side and forms the deeper aspect of the neurovascular sheath on the other? We think not. In other words, we do not believe in the existence of a cylindrical fascial sheath for the whole length of the finger. It takes much skilful and tricky dissection to demonstrate the palmar part alone.

According to us, there is in the finger only one distinct aponeurotic structure which we shall describe under the name of lateral digital sheet. Our description will differ but little from Thomine's description of the lateral digital band. This band, which is most distinct at the level of the second phalanx, we regard as a lamellar structure in a larger sagittal sheet which is continued along the lateral aspects of the finger, from the commissure to the third phalanx. We think that our description is simpler, more succinct, and more likely to explain the

relation between the anatomical structures and the pathological changes. Dissection of the lateral digital sheet is easy. One only has to remove the skin and then systematically to dissect out the fatty tissue. The lateral sheet is then exposed on the side of the finger, as can be seen from Fig. 6 which is an exact reproduction of a photograph. Horizontal sections made through the first phalanx and the proximal I.P. joint illustrate the description (Figs. 2.7, 2.8, 2.9).

Figure 2.6
Medial fascia of middle finger showing retinacular ligament and band along the side of the middle phalanx, both hiding the neurovascular bundle.
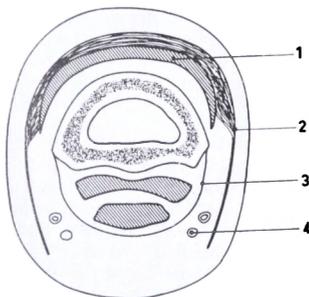

Figure 2.7
Transverse section through the proximal phalanx: (1) extensor apparatus; (2) lateral fascial sheet; (3) fibrous flexor sheath; (4) lateral digital nerve.

On the side of the proximal phalanx, it forms a sheet with a free anterior border, and a posterior border which bears a close relation to the extensor apparatus which, at this level, consists of the common extensor tendon plus the tendinous expansions of the interossei. Most of the fibres in this structure run vertical to the axis of the finger. Some run obliquely downwards and posteriorly to form a superficial strap over the extensor apparatus. Superficially, the skin over the lateral aspect of the finger opposite the sheet is thin and supple. At this level, there is but little fat, and very fine fibrous tracts tie the skin down to the sheet.
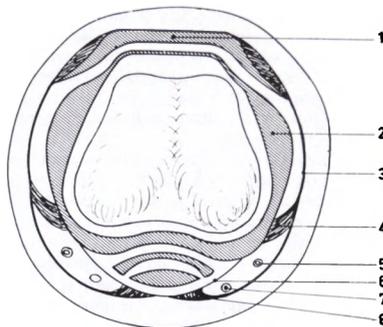

Figure 2.8
Transverse section of proximal interphalangeal joint: (1) extensor apparatus; (2) capsule and collateral ligament; (3) lateral fascial sheet; (4) expansion of lateral fascial sheet to capsule; (5) digital artery; (6) fibrous flexor tendon sheath; (7) digital nerve; (8) expansion of lateral fascial sheet to fibrous flexor sheath (origin of retinacular ligament).
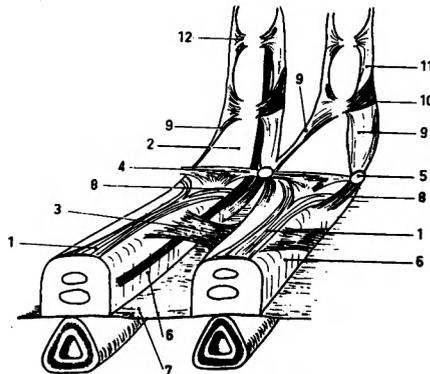

Figure 2.9
Diagram of fascial elements: (1) pretendinous band of palmar aponeurosis; (2) fibrous flexor sheath; (3) superficial transverse ligament of palm; (4) natatory ligament; (5) chiasma of natatory ligament; (6) septa of Legueu and Juvara; (7) deep transverse ligament; (8) origin of lateral digital fascial sheet, fusion of bifurcation of pretendinous band and distal extremity of septum of Legueu and Juvara; (9) lateral digital fascial sheet; (10) insertion of this sheet into the pulley of flexor sheath; (11) digital band at middle phalangeal level; (12) insertion of digital sheet on the pulley of the fibrous flexor sheath at distal interphalangeal level.

Finally, we note that this sheet has a uniform density along the distal three-quarters of the phalanx. Along the proximal quarter, at the origin of the sheet, near the commissure, the anterior border is markedly thicker than the posterior segment facing the extensor tendons. On a lateral view of the finger, the sheet completely

conceals the neurovascular bundle which lies between the deep surface of the sheet and the anterolateral aspect of the flexor sheath. At this point, we must stress the close relation of the sheet to the nerve. Opposite the proximal phalanx, the sheet separates the lateral teguments from the neurovascular bundle. But a capital change occurs opposite the proximal interphalangeal joint. Fibres leave the sheet and find a strong insertion in the fibrous elements of the capsule of the joint. From the deep aspect of the sheet, fibres are given off which become imbricated with the lateral ligaments. From the anterior border, a bunch of fibres run across, and superficial to, the neurovascular bundle towards the middle of the flexor sheath where they find an insertion. On a face view of a dissected finger, the nerve, which is visible at the level of the proximal phalanx, is hidden opposite the I.P. joint by expansion of the sheet to the flexor sheath (Fig. 2.8). This expansion has been described by most authors, but some believe that it is inserted on to the bone. This, to us, seems impossible as, in a deeper plane, it could only reach the capsule laterally or the tendon sheath anteriorly. Being anterior, the insertion must be on the fibrous sheath. At that level, the neurovascular bundle does go through a tight fibrous tunnel formed posteriorly by the anterolateral aspect of the proximal I.P. joint and the flexor sheath, and anteriorly by the articular expansion of the lateral sheet. The slit is closed posteriorly by the capsular expansions of the sheet, and anteriorly, near the axis of the finger, by the expansion which crosses over the nerve and becomes closely adherent to the axis of the finger at the fibrous tendon sheath.

We now have to describe the lateral sheet opposite the 2nd phalanx, or in other words consider the fate of the fibres which have become inserted deep into the fibrous capsule of the proximal I.P. joint. The deeper ones, those which adhere to the capsule, run vertically along the side of the second phalanx and again become adherent to the lateral aspect of the next joint, i.e. the distal I.P. joint. There again there is a continuous strand of fibres which form a posterior cover for the extensor tendon. The density of this stand is quite different on the side of the finger and on its posterolateral aspect. On the side of the finger it forms a strong, solid band stretched between the lateral aspects of the I.P. joints like the string of a bow, the arc of which is formed by the concave border of the second phalanx. Thomine's description here is perfect in every way. There does exist a strand which joins the posterior border of the band to the border of the lateral strips given off by the extensor tendon. It is so thin, so tenuous that we can easily understand that it has been overlooked. But there are also more superficial fibres. They come off the anterior expansion of the strand which is adherent to the flexor sheath, opposite the

proximal I.P. joint; from there they run a distal and posterior course, the first ones running very posteriorly while the others run a more oblique course at about 45° to the axis of the phalanx—the whole structure forming a triangular sheet. Its summit lies anteriorly and is fixed to the fibrous sheath; it covers the lateral aspect of the second phalanx and, on the back of the same phalanx, it forms a strap which runs superficial to the extensor apparatus to which it is adherent. On the posterior aspect of the finger, the two triangular sheets meet in the midline after covering the medial and lateral aspects of the finger. These oblique fibres which strap the extensor tendon are stronger than those at the base of the first phalanx, though they occupy a comparable position. Although they belong to the lateral sheet, their oblique course makes them appear separate and they can be separated with a scalpel. In this way, the retinacular ligament of Landsmeer is artificially created. And having been thus created, it is assumed to have some physiological function which we think extremely doubtful. A number of authors have given confusing descriptions of this ligament; this comes out from the very summary diagrams which appear in a number of publications. In our figure, copied from a photograph, it appears as a distinct structure, as clearly as in the photograph published by our colleague Rabischong (Fig. 2.6).

3. THE PALMO-DIGITAL JUNCTION

What we are most interested in at this junction is the continuity between the palmar and digital structures, a continuity which can be demonstrated by deep dissection. It is because these palmodigital fibres lie deep on the sides of the metacarpophalangeal joints that the retractile fibrosis leads to the typical flexion deformities of Dupuytren's disease.

But before following these longitudinal fibres, we must consider in more detail the superficial, transverse palmar interdigital ligament which we have not yet described (Figs. 4 and 5).

Its fibres draw a long curve, slightly concave proximally, from the lateral border of the index finger to the medial border of the 5th finger. From its more superficial fibres, small fibrous strands run up to the dermis. As it crosses the base of each phalanx, its deep fibres become closely attached to the anterior aspect of the tendon sheath by a tough band. But these same deep fibres as they run across each commissural space, form an arch under which the neurovascular bundles run down to the fingers. At these arches there is an intricate mingling of fibres between the longitudinal fibres of the aponeurosis, the lateral digital sheet and the palmar interdigital ligament. As an example let us consider in detail this imbrication or 'chiasma', at the third interspace between the middle finger and the ring-

finger (Figs. 2.10, 2.11). Opposite the neck of the third metacarpal, the medial branch of the bifurcated pretendinous thickening is twisted on its axis and runs deep into the hand. Its deep aspect is closely adherent to the capsule of the M.P. joint. The branch of the pretendinous thickening fuses with the inferior part of the partition of Legueu and Juvara. Together they run as a bundle of fibres under the transverse palmar ligament to form the medial laterodigital sheet of the middle finger. Thomine is quite right when he says that the superficial fibres of the transverse palmar ligament arising from the middle of the commissure,
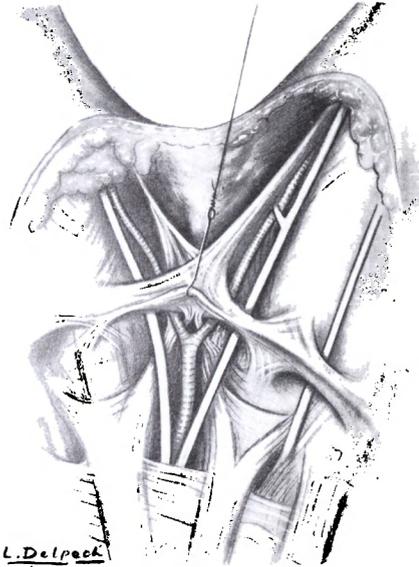

Figure 2.10
In the web space the origin of the lateral fascial sheets of the finger and, crossing between the digital nerves and the aponeurosis, the chiasma of the natatory ligament.

become divergent distally, and run one branch towards the anterior border of the medial digital sheet of the middle finger, and the other to the anterior border of the lateral digital sheet of the ring finger. Thomine has suggested that the fibres running from the transverse palmar ligament to the lateral digital sheets are in fact the proximal origin of these sheets. We do not agree with this view and regard these fibres as an accessory structure. The true origin of the lateral digital sheets is constituted by the deep fibres which are the common continuation of the bifurcated pretendinous band and of the intertendinous partition of Legueu and Juvara.

Figure 2.11
Photograph from which Figure 2.10 was drawn.

We must remember that they adhere closely to the capsule of the M.P. joint and are continuous with the anterior border of the deep aponeurosis. Although the anterior border of the digital sheet does give off a few fibres which mingle with the transverse palmar ligament, the majority of the fibres of the digital sheet run deep under the transverse palmar aponeurosis which have regrouped opposite the metacarpophalangeal joint.

At this level, it is essential that we describe accurately the relations of the nerves with the aponeurotic bands.

Figure 2.12
A clear demonstration of the digital nerve passing anterior to the aponeurotic expansion as it forms the proximal end of the lateral digital fascial sheet.

From a surgical standpoint, these relations are extremely important. If they are not clearly understood, aponeurectomy becomes a hazardous procedure.

On a transverse section of the hand, going through the distal third of the metacarpal, the sagittal partition can be seen lying between the tendons and the neurovascular bundle. Relative to the axis of the finger, the nerve is more peripheral than the partition. On a section of the finger at the level of the proximal phalanx, the digital sheet is more lateral than the nerve, again relative to the axis of the finger. These structures must have crossed at some point, and this point is in the line of the M.P. joint. The aponeurotic sheet after running longitudinally along the metacarpal, is deviated off the axis of the finger by the protruding metacarpal head. The lateral digital nerve crosses over it and continues along the side of the phalanx. More distally the divided branch of the digital artery follows a similar course. Figs. 2.10, 2.11, 2.12, especially 2.12, drawn from a specimen, show this crossing more accurately than any verbal description.

4. LESIONS AND MECHANISMS OF FLEXION IN DUPUYTREN'S DISEASE

We have said earlier that there are two types of lesions in Dupuytren's disease—the nodules and the bands.

The nodules superficially are very adherent to the skin, and deeply may become attached to the aponeurosis when this is present. But in a deeper layer, there is always a plane of cleavage between the nodule and the subjacent structure, provided this structure is not an aponeurosis. This is most obvious when a nodule is present in the midline, between the metacarpophalangeal crease and the neck of the proximal phalanx.

One usually has no difficulty in finding a plane of cleavage between the deep aspect of the nodule and the anterior aspect of the fibrous flexor sheath. But superficially, there usually exists a continuity between the nodule and the dermis. The nodule is always situated along a metacarpodigital axis. We agree with Hueston when he says that the nodule is formed in the fatty tissue, and at first develops independently of the aponeurosis. For the palmar nodules, this is still debatable. There is no palmar nodule without retractile sclerosis of the pretendinous aponeurotic band, and one is entitled to believe that the lesion started in the aponeurosis and later spread superficially to the subcutaneous fat and to the dermis. But at the level of the proximal phalanx, it is different: the nodule always lies on the midline where there is no recognizable aponeurotic structure. There the nodule develops in the subcutaneous cellular layer.

Proximally, every large mid-line phalangeal nodule is continued into a flattened, fibrous, ribbon-like strand towards the bifurcation of the pretendinous strip. More distally, the nodule is always adherent to the expansions of the lateral digital sheets which converge and become inserted in the midline, proximal to the tendon sheath insertion and to the proximal phalangophalangeal joint line (where the retinacular ligament finds its origin). It is at this level that excision of phalangeal nodules is most difficult and presents the risk of causing injury to the collateral nerve.

The nodules are usually what worry the patients most. In fact it is a relatively accessory lesion. The nodule does give rise to sclerosis and to cutaneous retraction, but it never leads to progressive flexion of the fingers. This flexion is due to the retraction of the aponeurotic structures forming bands, the situation of which depends on the anatomy of the aponeurosis.

This retractile sclerosis may be localized, or it might be extensive, or may be restricted to a palmar or a digital segment.

A large number of clinical forms can be distinguished. We shall only describe the most common ones.

(a) RETRACTION OF THE PRETENDINOUS PALMAR STRIPS

This can exist without nodule formation. In this case, adhesions to the skin are scanty: they are limited to a few millimetres here and there, proximal and distal to the point where the pretendinous strips cross the distal palmar crease. Such a lesion only results in flexion of the M.P. joint as, through its bifurcate insertion into the capsule of the M.P. joint, the shortened strip pulls on the base of proximal phalanx of the finger. The palmar skin is then often found to be thin and supple. In these cases, a simple subcutaneous fasciotomy will sometimes result in complete extension of the finger, which shows that metacarpophalangeal flexion comes exclusively from retraction of the pretendinous palmar strip. But in such cases, we usually restrict simple fasciotomy to old patients, female patients, rheumatic patients and patients in poor physical or mental health. We much prefer fasciectomy, but we only mention fasciotomy because it provides a good experimental demonstration of the pathogenesis of digital flexion in the pure palmar forms.

In the course of fasciectomies performed in the severe, purely palmar cases, affecting possibly two adjacent fingers, one may observe that the pathological process affects not only the pretendinous strip but also the intertendinous partition of Legueu and Juvara.

The latter is then found to be tough, thickened and difficult to cut with scissors. Sometimes, in its distal part, it ends as a cylindrical cord 2 to 3 millimetres across, which runs deep and is crossed over by the collateral nerve. Obviously the nerve must be dissected out before the cord is divided. It is easy to understand why a fasciectomy limited to the frontal fibres of the aponeurosis will be inadequate unless the excision is

extended into the intermetacarpal space to include the strongest cord that pulls on the phalanx. This cord in fact constitutes the lower end of the partition of Legueu and Juvara after its fusion with the bifurcated branch of the pretendinous strip. In such a case, a single transverse incision across the pretendinous strip will hardly increase digital extension. In other quite common cases, one may find a combination of a palmar band (with or without palmar nodule) and a large prephalangeal midline nodule. Operation must then include palmar fasciectomy plus excision of the phalangeal nodule. As this nodule is attached proximally to the pretendinous strip and distally to the retinacular ligament of Landsmeer, it is easy to understand why it may lead to flexion of the middle phalanx relative to the proximal phalanx. Such flexion is never severe and usually yields readily after aponeurectomy and removal of the nodule. If it does not improve, there is usually something else: for example a diseased lateral digital sheet. In Dupuytren's disease it is this sclerotic and retractile lesion in the digital sheet which gives rise to the most severe forms, those which are included in group D of Michon and Tubiana.

(b) RETRACTION OF THE LATERAL DIGITAL SHEET

In the majority of cases, this retraction which leads to the formation of a lateral digital band is associated with a pretendinous palmar band. There are cases (relatively rare) where one finds a combination of palmar band, phalangeal nodule and lateral digital band. It is then difficult clinically to detect the lateral digital band before operation. One must always look for it at operation, and it will always be discovered when full extension is not restored by fasciectomy and removal of the phalangeal nodule. Quite often when there is a unilateral (on one side of the finger) or bilateral (both sides of the finger) digital band, there is also a palmar band but no phalangeal nodule. On palpation of the forcibly extended finger, the midline prephalangeal fatty tissue feels soft, yielding and normal. But on the sides of the finger one can feel a thin, taut cord which corresponds to the anterior border of the lateral digital sheet.

Surgically, one begins with the dissection, and the lifting of the palmar aponeurosis starting with its proximal end. This reveals a thickened, sclerosed, retracted pretendinous band which is not continuous on the palmar aspect with any abnormal tissue beyond the proximal digital crease. But it continues on the side of the metacarpal head and of the base of the proximal phalanx as a solid sheet which corresponds exactly to what we have described as the proximal attachment of the lateral digital sheet. But before coming to the latter, one must identify the nerve from the palmar side and follow it as it seems to emerge from the depths, and is crossed by the lateral prolongation of the pretendinous

strip. If the M.P. joint is in pronounced flexion, the anterior border of this band becomes superficial as it stretches from the palm to the side of the root of the finger. The band then lies at the same superficial level as the frontal plane of the palmar aponeurosis. It lifts the collateral nerve which is easily dissected out if one is aware of this pathological relation which, contrary to what has been claimed by some authors, does not represent an anatomical anomaly. Further away from the axis of the finger, the collateral artery crosses over the origin of the lateral sheet. Like the nerve it must be separated, identified, and treated with respect.

At this stage of the operation, there is no difficulty in dividing the lateral digital sheet at its origin, but the deeper fibres often escape the knife and are not easy to cut through as they lie trapped between the sides of adjacent M.P. joint. The surgeon then passes on to the finger where a Z-incision, with a midline vertical branch, is made on the proximal phalanx. We want to stress this point, as one is easily tempted into making a lateral or posterolateral phalangeal incision when faced with a lateral digital band. This type of incision which we have used ourselves on a number of occasions has little to recommend it. It gives a poor exposure of the distal portion of the digital band which is in the midline opposite the line of the proximal I.P. joint, at the origin of the retinacular ligament. It opens first on to the lateral band when in fact, before tackling this band, one must identify and protect the artery and nerve which are nearer the midline. Finally, the Z-plasty of the first phalanx has the immense and irreplaceable advantage of making possible closure of the palmar incision without tension and without a graft.

The edges of the Z are then raised. On the sides of the phalanx where the band runs, the lateral skin of the finger is very adherent to the sheet. The band must be freed right through to the lateral border of the interosseous tendon. But before going further, the nerve and collateral artery must be freed on the internal aspect of the lateral band (i.e. internal relative to the digital axis). Half way down the phalanx, this is easily done as the neurovascular bundle is perfectly free and is never included in a fibrous mesh. But it must then be followed right down to the proximal I.P. joint, it must be freed during its passage through the tunnel formed by the palmar expansion of the lateral sheet. Finding this tunnel and widening it must be done with great care and prudence.

The digital sheet has now been freed on two aspects. During the anatomical description, we said that its posterior border gave rise to fibres which form a covering for the extensor aponeurosis. This must now be divided vertically close to the border of the extensor apparatus. Further down the finger, opposite the proximal I.P. joint, one must also divide the band which bridges over the nerve and fuses in the midline with the

fibrous sheath and the longitudinal fibres which are closely adherent to the capsule reinforced by the lateral ligaments. Inadequate posterior dissection can well spoil the result of the operation. If the neurovascular bundle has been well isolated, the nerve is not at risk; the artery is more vulnerable and must be spared. With the lateral band held with strong Ombredanne forceps, the dissection is continued proximally and in depth to divide the last fibres from the deep aponeurosis. While dissecting the proximal origin of the lateral digital sheet, we have been able to confirm that this origin lies in the intermetacarpal space as we described. The main origin does not appear to be from the palmar ligament or the commissure as Thomine suggests. It is easy to understand how retraction of this band, which starts at the metacarpal head, anterior to the axis of the phalanx, and is fixed on the fibrous sheath in front of the proximal I.P. joint, can cause strong flexion of the second phalanx on the first. As its posterior fibres fuse with the lateral strips of the extensor tendon, it is also quite normal that severe retraction would result in hyperextension of the distal phalanx.

In some cases, rarely concerning the middle or the ring fingers, the disease may affect the digital sheet and spare the palmar aponeurosis. The phalangeal Z-incision then makes possible a resection of the lateral digital sheet in the same way. But division of its deep proximal origin may be more laborious than with the combined palmar and digital approach.

In Dupuytren's disease of the 5th finger, there is characteristically a band on the lateral side, alone or associated with palmar lesions. The digital sheet on the ulnar origin is complicated. It arises from the anterior aspect of the terminal tendon of the abductor of the 5th finger. Its fibres cross the terminal, superficial internal fibres of the transverse palmar ligament but come up much higher on to the hypothenar aponeurosis. They are in no way related to the middle palmar aponeurosis. Here again, we are in favour of a Z-incision over the proximal phalanx, the Z being continued vertically along the ulnar border of the hand. The Z is done as follows: a midline incision on the anterior aspect of the phalanx, a branch of the Z starts from the distal end of the incision and comes up the radial side of the proximal phalanx of the 5th finger. The other arm of the Z runs from the proximal end of the midline incision towards the medial side of the 5th finger. From this end of the Z a 2–3 cm vertical incision is made along the ulnar side of the hand. Resection of the lateral band of the 5th finger is then performed with no difficulty.

We must now describe another form of Dupuytren's disease which for lack of a better name we shall call the commissural Y-bands. In these cases, a pretendinous palmar band continues as a double phalangeal band, one on the side of the finger corresponding to the palmar band the other on the side of the adjacent finger. In all

these cases, it is difficult to separate the affected fingers (there is often intertrigo of the commissure) and the palmar interdigital ligament is itself thickened, and retracted in the commissure to which it is adherent. The exact anatomy of the lesions was at first difficult to define. It is in fact a Y-shaped band. The Y-band arises from a single partition of Legueu and Juvara. A cord is formed which gets thicker at the commissural chiasma where two digital bands are given off one to each adjacent finger. It is in such cases that the relations between the nerves and the aponeurotic bands appear most confusing to the inexperienced surgeon.

All this we think is the information one can derive from the anatomical study of the palmodigital aponeurosis and its pathological modifications. In order to keep this study short, we have avoided on purpose the thenar forms. These are of no particular anatomical interest.

A resection of the aponeurosis performed according to strict surgical rules will almost always result in full extension of the affected fingers. But in old cases, the fingers may remain in flexion in spite of a perfect aponeurectomy. What then are the remaining obstacles? One is well known—it is the retraction of the lateral ligament and of the anterior capsule of the proximal I.P. joint. This, we believe, is the result of an ordinary retraction from prolonged immobilization in flexion, and is not due to extension of the disease process to the capsule itself. A bilateral and anterior capsulectomy may reduce this residual flexion. But we always found the results very mediocre, the more so as there is another cause for this persistent flexion which no author seems to have mentioned: that is retraction of the flexor muscles of the finger. In one case of Dupuytren's disease which we considered incurable, we had warned the patient that we would amputate the little finger. Yet we performed a palmar aponeurectomy to free the ring finger which was also affected. We then carefully resected a large band on the internal side of the 5th finger with no improvement of flexion. Then, we proceeded to amputate the finger and to divide the flexor tendons. This resulted in a considerable gain in extension, showing how severe the retraction of the flexor muscles of the 5th finger can be. The disarticulated finger still remained flexed at the proximal I.P. joint. An anterior capsulectomy at this joint did not relieve the tension in the lateral ligaments. The finger could be straightened but would spring back into flexion. It was only when we divided the lateral ligaments that the finger recovered its normal suppleness. In those cases where there has been persistent flexion for months or years, leading to retraction of the muscles and ligaments, amputation seems to be the only reasonable therapeutic solution.

Erosion and flattening of the volar aspect of the condyles of the proximal phalanges were found in

fingers amputated from another patient. Thus it is vain to hope for useful functional recovery in certain long-standing forms of Dupuytren's contracture. If, after adequate resection of a volar nodule and of the lateral phalangeal bands the finger cannot be easily extended, we believe that it is useless to seek further extension by anterior and lateral capsulotomies of the proximal interphalangeal joint. Such a capsulotomy will seem at first to have been successful because the finger can be straightened, sometimes easily, sometimes with detectably abnormal tension of the flexor tendons. However, in the next few weeks, the greater tension of the flexors over the extensors will pull the finger again into flexion. Moreover in these obstinate cases extension can only be obtained at the risk of upsetting the circulation of the fingers. In two cases secondary amputation of the ring finger has been necessary because of progressive ischaemic necrosis of the two terminal phalanges. Since we seek a functional result it is admitted that the most perfect aponeurosectomy will be inadequate in some severe cases with secondary joint changes. In these very rare cases (less than 1 per cent) hand function is better restored by amputation than by preserving a stiff, straight finger. When amputating the little finger a large dorsal flap, taken as far down as the proximal interphalangeal joint, is preserved for use in closing the palmar defect.

5. SOME PRACTICAL CONCLUSIONS

So far we have operated on about 610 cases of Dupuytren's contracture and as a result of this experience certain technical points can be laid down.

(a) We have made it a rule only to operate when there is at least 30 degrees finger flexion. We advise patients against operation when there are only nodules or weak palmar bands not producing flexion deformity. No benefit will be obtained by operating at stage o when there is no functional loss. Moreover we cannot predict the natural history of Dupuytren's contracture and it may be 5 or 6 years before the onset of digital flexion deformity. On the other hand it has been found that, when a limited palmar aponeurosectomy has been performed, extension of the retraction process has occurred elsewhere in an unexpected situation, particularly in the proximal phalangeal segment of the fingers. Another operation is then necessary.

(b) In such cases we try to slow down the development of the pathological process by corticoid injections into the aponeurosis. Eight injections per month are given in two courses within a year, with strict aseptic precautions. By giving the injection from the dorsal aspect of the hand this is quite painless, the needle passing between the two metacarpals to reach the palmar aponeurosis where the depth of the needle is checked by palpating the palm.

There is no danger from this treatment and it seems useful without us being able to give statistics on its results.

(c) In all cases with moderate or advanced flexion deformity we advocate aponeurosectomy to remove at least the proximal part of the palmar aponeurosis and its extension overlying the third, fourth and fifth metacarpals. All digital lesions are excised at the same time. Is it still necessary to condemn forced extension of the fingers without surgery? It is a blind manoeuvre which can only stimulate the process of retraction and can produce rupture of the adherent overlying skin.

Simple subcutaneous fasciotomy seems only justified in very rare cases with supple skin and a fine palmar band producing metacarpophalangeal flexion only in one finger without any digital lesion.

(d) General anaesthesia and a pneumatic tourniquet are routine except when regional anaesthesia is advised to lessen the risk in very old cardiac patients. A wrist block is straightforward, infiltrating the median and ulnar nerves at the level of the carpal tunnel and passing subcutaneously over the dorsum of the wrist. It is worth noting that the pneumatic tourniquet is quite well tolerated on the forearm by some patients, despite being above the zone of anaesthesia, and these patients do not remember any pain from such a procedure. We do not have much confidence in brachial plexus blocks, intravenous regional anaesthesia or axillary blocks.

(e) Postoperative complications. We find haematomas are rare. Nerve lesions are the result of clumsiness. Prolonged disturbances of sensation are seen in some patients without having had the slightest nerve lesion but in whom the dissection to free the digital nerve at the level of the proximal interphalangeal joint has been difficult and presumably has inflicted a mild contusion.

Necrosis of the proximal border of the transverse palmar incision of McIndoe seems to be our commonest complication. This necrosis is negligible if it only involves the marginal millimetre or two of the wound. Occasionally however, a triangular slough with 1 cm margin requires excision, but, after this, spontaneous epithelialization always occurs. These secondary scars leave a little induration. If it were possible to predict those cases where skin necrosis will occur, a split skin graft would certainly be used. We have not yet had to use this but it will doubtless be warranted in some cases.

Twice we have seen necrosis of both flaps of the Z-plastics over the proximal phalanx with exposure of the flexor tendons but a cross-finger flap has given an excellent result in each case.

Postoperative stiffness of the entire hand is the most serious complication of all. It occurs in those rheumatic patients with atrophic tendencies and abnormal vasomotor responses in the hand. Persistent postoperative oedema of the hand is sometimes helped by local corticoid therapy.

Careful active postoperative re-education of the hand reduces the incidence of these complications. They cannot be totally avoided, particularly the functional loss from stiffness preventing full active flexion of the fingers.

Let us insist on the importance of active postoperative movements. Without this the best operation will be left with a functional deficit. Fifty per cent of the result of the operation seems to depend on this regime of active movement which can only be delegated to carefully instructed physiotherapists.

The most upsetting forms of this complication are in women and particularly in those with a rapid development of the disease, pains radiating up the arm and other rheumatic manifestations. The others are men with horny hands, stocky and already with an element of oedema in the palmar and dorsal tissues of the hand.

Postoperative infection is very rare. A meticulous preoperative toilet of the web spaces is necessary in stage IV cases with several fingers flexed because often some intertrigo is present.

Finally frost-bite, chilblains or Raynaud's disease must make us very hesitant to operate—even to the point of refusal.

With regard to the syndrome described by Hueston— Dupuytren's diathesis—associating Dupuytren's contracture with knuckle pads and plantar lesions, this seems to be very rare in France.

(f) Recurrences. We have not seen true recurrence, i.e. reappearance of retractile bands in an operated area, after the technique that we use (except in two cases when the aponeurosectomy had been imperfectly performed). Some recurrences have been seen for instance when, during excision of a phalangeal nodule, the surgeon has left behind an associated lateral phalangeal band which was the real cause of the proximal interphalangeal deformity.

On the contrary it is possible by a limited resection to accelerate the appearance of new lesions outside the operated area in fingers quite clear at the time of the first operation. This is actually an extension rather than a recurrence of the disease.

In severe forms when there are already joint changes, one finger, usually the fifth finger, tends to flex again. This result is so troublesome as to amount to a recurrence, but in fact it is due to fibrous retraction of the skin and the capsular elements of the proximal interphalangeal joint.

SUMMARY

The author first reminds us of the two types of lesions found in Dupuytren's disease: the nodules and the retractile bands. They differ histologically and clinically, only the latter causing severe digital flexion. It is not possible to correlate the localization of nodules with the anatomy of the palmar and digital aponeuroses. The bands however, are known to result from a retractile sclerosis of the aponeurotic structures. It is essential therefore to know the anatomy of these aponeuroses when resecting these bands. This anatomy is described in detail and its peculiarities are stressed.

Conscious of these anatomical points, the surgeon can perform systematic adequate, efficacious aponeurectomies without endangering neurovascular trunks, the course and relations of which relative to the pathological structures is always easy to understand.

The author considers in detail the lateral digital aponeurotic sheets and the bands that develop therein. He also describes the anatomy of the retinacular ligament of Landsmeer.

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