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The genesis of the palmar lesion

It is frequently stated that the primary lesion in Dupuytren's disease (DD) is the palmar nodule (Luck 1959; Gabbiani & Majno 1972; Hueston 1975). However, the term 'nodule', which is commonly used, is really a clinically descriptive term for a localized palmar thickening which although clearly palpable is not always clearly defined. The use of the term 'nodule' in this clinical context must be carefully differentiated from the biological or pathological use of the word 'nodule' in DD. The differences in the usage of the term 'nodule' in the clinical and pathological or biological sense have probably contributed to some of the differences of opinion about the aetiology or pathogenesis of DD. Few would argue that a palpable nodule — often localized to the proximal side of the distal palmar crease — is one of the most frequent and well recognized early clinical manifestations of the disease. Histological examination of one of these clinically manifest early 'nodules' from the palm shows that it is really a composite structure of thickened, coalesced, longitudinally running fascial fibres exhibiting patches or localised zones of intrafascicular cellularity collagenolysis and connective tissue remodelling. In reality, it represents a series of progressive changes which have been developing long before the thickened nodule became clinically apparent and which have gradually transformed the soft, plump, compliant palm of the child into the more fibrotic, inelastic and non-compliant material of the adult (Table 13.1).

In some circumstances, a clinically apparent nodule may appear to have developed locally without any obvious connection with a previously

Table 13.1 Biological events in Dupuytren's disease

Primary thickening of superficial palmar fascia, especially superficial longitudinal bands
Matting of longitudinal vertical and oblique fibres and loss of compliance
Loss of hyaluronate from fascial fibres?
Development of intrafascial cellular lesions
Secondary fascial thickening
Contraction — cellular and extracellular
Contracture — connective tissue remodelling

thickened band of fascia. However, the slowly developing fibrous plaques which are found in the region of the distal palmar crease are usually associated with more widespread thickening of the palmar fascial fibres and present either as a diffuse resistance to pressure on the palm or as a localized subcutaneous thickening in the distal palm. This type of localized subcutaneous thickening may also be associated with pits in the skin where the dermis is apparently tethered to the deeper fibres (Fig. 13.1). These dermal pits arising on the distal side of the distal palmar crease may be the first and only manifestation of underlying fibrosis.

Previous writers have described changes found in the dermis and palmar fascial fibres in samples of palm skin taken at operation from patients undergoing surgery for DD (Meyerding et al 1941; MacCallum & Hueston 1962; Chiu & McFarlane 1978; Bazin et al 1980; Ushijima et al 1984; Nezelof 1985). Whilst these samples have provided a fund of information about the nature and progression of the disease process they have not — because of their very nature — provided much information about the origin of the biological or

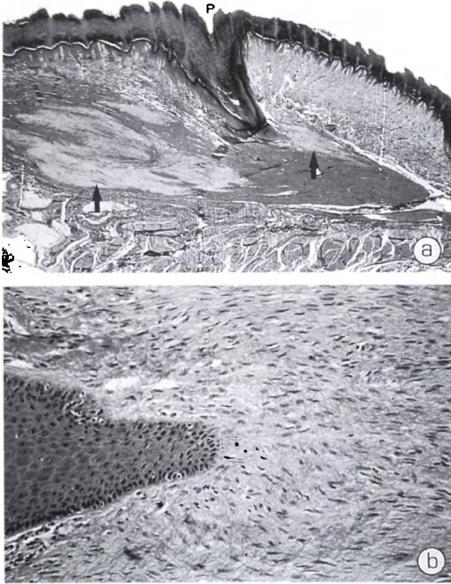


Fig. 13.1 Dermal pit. **a** Longitudinal histological section of thickened superficial pretendinous fascial band in distal palm showing pale staining intrafascicular cellular nodules (arrows). The proximal nodule is causing an invagination of the epidermis and overlying keratin, forming a dermal pit (P). **b** Higher power view of the indrawn tip of the invaginated epidermis in Figure 13.1a, showing cellularity of the contractile tissue of the nodule.

pathological changes preceding the clinical appearance of the disease. Some workers have occasionally been able to obtain biopsy samples of clinically asymptomatic palmar nodules at the time of operative treatment of some other lesion on the hand, such as a trigger finger (Chiu & McFarlane 1978). Although histological studies of these nodular biopsy samples describe changes which could justly be regarded as being earlier than those in clinically troublesome lesions, they do not necessarily describe the primal changes in the disease process.

However, the anatomical and histological studies of apparently normal, uninvolved hands, either by meticulous dissection of the surface

layers of the hand under the dissecting microscope (McGrouther 1982) or by the histological examination of serial longitudinal sections of palm skin and the underlying fascia from a wide variety of post-mortem subjects (Millesi 1959, 1985; Flint, unpublished observations — see also Chapters 10 and 28), have provided a better understanding of the microstructure of the apparently uninvolved palm and an opportunity to observe the development of changes within these normal structures which presage the later development of DD.

THE NORMAL PALM

In Chapter 2 the close interrelationship between connective tissue form and function has been stressed. Many features of this interdependent relationship are apparent in the specific adaptations to prehensile gripping of the superficial palm and its connective tissues.

The surface skin of the hand and fingers is covered by fine antiskid channels — the dermal ridges — the functional capability of which is enormously increased by the secretion of sweat from the apices of these ridges. Whilst the skin of the hand and fingers may undergo spontaneous hyperkeratosis as a self-protective mechanism when subjected to chronic localized frictional, torsional or pressure forces, the ability of the specialized integument to absorb these forces is enormously increased by its firm attachment to the underlying palmar aponeurosis by a honeycomb of interlocking fascial fibres. Grapow (1887) emphasized this relationship and noted that the palmar fascia 'secures grip through its intense surface connections with the overlying skin which might otherwise slide off the hand like a glove'. Millesi (1959, 1985) and Thomine (1965) also stressed the importance of this firm fibrous attachment in preventing avulsion of the skin during torsional and compressive movements. They pointed out that from a comparative anatomical viewpoint the palmar aponeurosis is a late phylogenetic development and is best developed in climbing animals. Millesi (1959) also noted the cushioning effect of the fat lobules laid down in the fibrous tissue network immediately beneath

the dermis in the superficial palmar connective tissue continuum.

LOAD BEARING AND MOBILITY

The load-bearing fibrofatty subcutaneous tissues of the palm, the heel and the plantar surface of the foot under the metatarsal heads are quite different from other subcutaneous fatty tissues (Gillard et al 1977). In load-bearing situations the subcutaneous fat cells are arranged in groups within fibrous compartments made up from collagen and elastic fibres embedded in a proteoglycan matrix, particularly rich in chondroitin sulphate (Fig. 13.2). The containment of the fat within relatively non-extensible fibrous boundaries makes it more efficient as a pressure

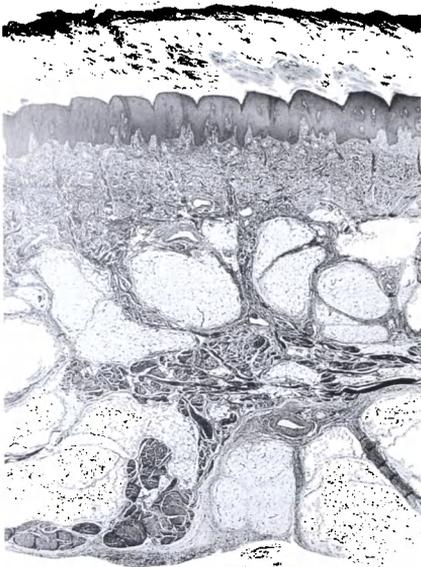


Fig. 13.2 Load-bearing skin of the heel showing the thickened keratin and epidermis and the loculated fibrofatty shock absorbers beneath the dermis.

absorptive structure. In the palm, anchoring of the skin is achieved by means of the interlacing three-dimensional fibrous network of 'the palmar connective tissue continuum' (see Chapter 2) which stabilizes the palmar skin during gripping and ensures that the skin does not fold or roll when subject to shearing or compressive forces. The hypodermal fat pads occupying the spaces within and around the three-dimensional fibrous network serve to absorb the energy of such forces.

I believe that these fibrous attachments, particularly of the fine, superficial longitudinal and oblique fibres, and the surrounding fibrofatty compartmentalized shock-absorbers, hold the key to the early pathogenesis of Dupuytren's contracture and also allow the apparently divergent views of Millesi (1985) and Hueston (1985) or Dupuytren (1834) and Goyrand (1833) to be more easily reconciled.

The situation is compounded by the increased mobility of the fourth and fifth metacarpal rays, particularly around the metacarpophalangeal joints, which leads to increased excursion of and stress upon the skin, the subcutaneous tissue and the longitudinal fascial bundles on the ulnar side of the palm. In this region the superficial pretendinous longitudinal fibres inserting into the skin beyond the distal palmar crease form part of a complex fasciocutaneous anchoring system which controls the folding of the skin at the distal palmar crease during hand flexion. It is interesting that DD typically occurs in areas of the palm skin which are 'loose' in the resting flexed posture and where anchoring systems are more highly developed (McGrouther, personal observation).

Hueston (1977) has stressed that the transverse distal palmar crease is retracted and hidden when the fingers are flexed into a gripping position. However, if one marks the palmar skin whilst the fingers are flexed at the metacarpophalangeal and interphalangeal joints it will be observed that the contact areas lie proximal and distal to the distal palmar crease, exactly coincident with the site of development of palmar nodules (Fig. 13.3). Whilst I would agree with Hueston that the distal palmar crease retracts out of the zone of contact loading, it should be remembered that the distal palmar crease itself is rarely the site of development of the initial palmar nodule.

Millesi (1959) emphasized that by ordinary methods of dissection, the skin and subcutaneous fascia of the palm are removed together, revealing the so-called 'palmar aponeurosis'. As a result, the superficial longitudinal fibres are usually removed or lost and thus are ignored or only briefly described in most textbooks. By removing only a fine layer of corium from the palm and dissecting individual fat lobules, Millesi was able to reveal the whole of the 'binding tissue system' of the superficial palmar aponeurosis. By carefully dissecting hand specimens under water whilst viewing them through the dissecting microscope, McGrouther (1982) was able to show the course and attachment of the superficial longitudinal fibres and their relationship to the deep longitudinal fibres of the aponeurosis. He also

demonstrated their close blending with the deeper aspects of the dermis beyond the distal palmar crease. McGrouther's dissections enabled him to develop a much clearer understanding of the three-dimensional interrelationships of the longitudinal, vertical, oblique and transverse fibre components of the palmar fascia and relate them to the distribution of the known thickened cords of Dupuytren's disease.

SUPERFICIAL LONGITUDINAL PALMAR FASCIAL BUNDLES

A histological study of specimens of palm skin taken from the fourth ray of a large number of post-mortem subjects ranging from mid-term fetus to old age, which I carried out in Auckland, demonstrated that from a very early stage of fetal development it is possible to trace a constant band of fascial fibres branching from the superficial surface of the deep longitudinal pretendinous fascial bands. These branching fibres run obliquely distally and progressively superficially to blend with the deep dermis beyond the distal palmar crease (Fig. 13.4).

At birth, this superficial longitudinal fibre bundle is well defined approximately 30–50 μ wide, running in its own fine paratenon sheath between two layers of loculated fibrofatty pads (Fig. 13.5). The form of this arrangement was found to be so constant that for the purpose of description, the hypodermal fat pads lying proximal to the distal palmar crease may be regarded as a superficial and a deep group

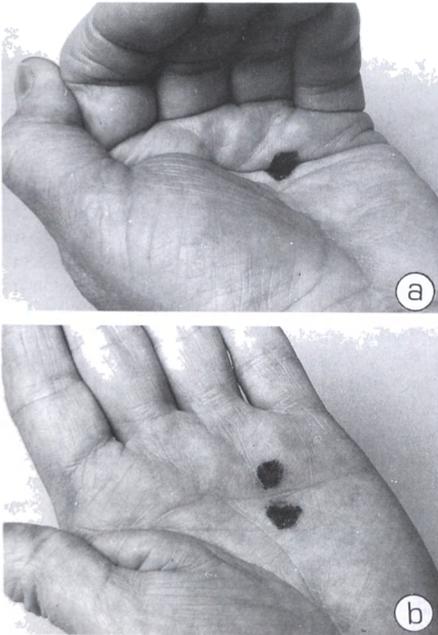


Fig. 13.3 a Ink spot marking impact area in fourth ray of palm of flexed hand. b Separation of spots on opening the hand demonstrate that the impact area in the flexed palm is coincident with the common sites of nodular thickening on either side of the distal palmar crease.

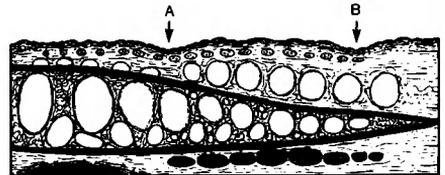


Fig. 13.4 Superficial and deep longitudinal pretendinous fascial bands. The fibres may separate more proximally at the level of the distal carpus. The subcutaneous fat pads lie superficial and deep to the superficial longitudinal bundle. (A) Distal palmar crease (B); proximal palmar crease.

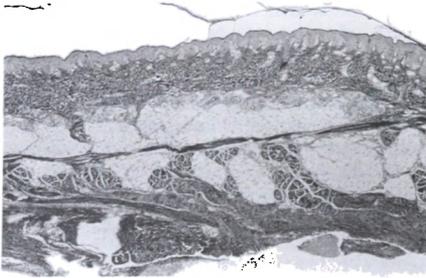


Fig. 13.5 Longitudinal section of palm skin of fourth ray of 1-day-old child showing superficial longitudinal fascial bundle running obliquely upwards towards the palmar crease, sandwiched between superficial and deep fibrofatty loculi.

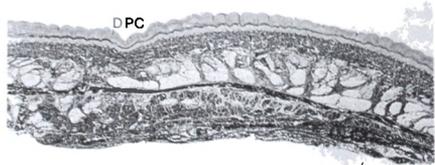


Fig. 13.6 Longitudinal section of adolescent fourth ray palm skin. Superficial longitudinal pretendinous fascial band separating from deep pretendinous fibres and passing forwards and upwards towards the distal palmar dermis. Note thickening of superficial longitudinal bundle and that the deep layer of fat pads proximal to the distal palmar crease (DPC) is not so apparent as the superficial group or as in Figure 13.5.

separated from each other by the obliquely running fibres of the superficial longitudinal fascial bundles (Fig. 13.6).

During late adolescence or early adult life, the fibre bundles were frequently more than a millimetre wide, and by the fourth or fifth decade were in some instances thickened and coalesced to 2–3 mm (Fig. 13.7). In such cases patches of intrafascicular cellularity or areas of apparent tissue repair or remodelling were sometimes evident within the fibre bundles. In some instances there was clear evidence of fibre rupture and connective tissue remodelling occurring within the substance of the 'microtendon' (Fig. 13.8). These regions of internal damage ranged from small discrete areas of change only discernible at higher magnification to large areas which were clearly discernible under the low power dissecting microscope (Fig. 13.9b). These larger zones were indistinguishable from classical Dupuytren's nodules.

As more specimens were examined, it became increasingly apparent that there was a progressive spectrum of change occurring within the fibres of the superficial longitudinal bands and that the thickened cords of fascial fibres containing the Dupuytren's nodules were generally anatomically coincident with the superficial longitudinal fibre bundles. This supports the concept that Dupuytren's cords and nodules developed in pre-existing fascial bands.

SUBDERMAL FIBROFATTY LOCULI

The importance of the attachment of the pretendinous fascial bands to the skin in maintaining the functional integrity of the palm has been stressed by a succession of writers (Grapow 1887; Legueu & Juvara 1892; Grodinsky & Holyoke 1941; Horwitz 1942; Skoog 1948; Iselin 1954a; Millesi 1959; McGrouther 1982), although few have recognized their role in the pathogenesis of the DD process. By contrast, there has over a corresponding period of time been much less mention of the subdermal fibrofatty loculi either in regard to their possible supportive functional role or in their relationship to the genesis of the palmar lesions.

In one of the earliest papers dealing with the anatomy of the palmar fascia, Maslieurat-Lagemard (1839) gave brief mention of these fibrofatty compartments, describing them as the 'soft pulpy tissue which contains a quantity of fat and numerous prolongations of fibres which come to adhere to the internal surface of the skin'. However, Madelung (1875), in what must now be considered to be a very enlightened work, graphically described the constancy and importance of the

'copious small fatty aggregations found in the palmar fascia of children and also of adults under the skin of the palm, ingrained as it were between the fibres of the connective tissue of the palmar fascia and the

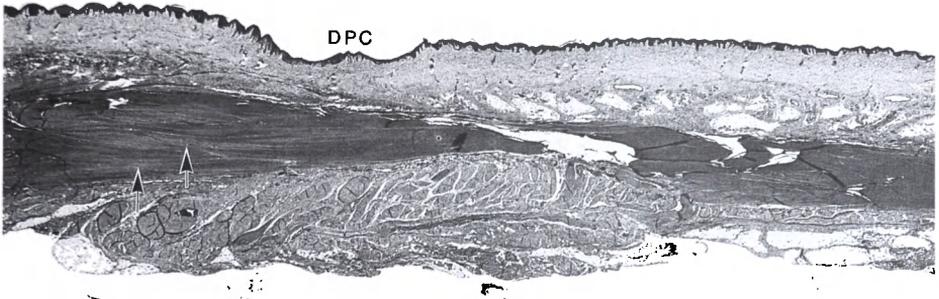


Fig. 13.7 Longitudinal histological section of palm skin of fourth ray of 47-year-old male showing marked thickening of superficial longitudinal band throughout its length. Distally note lighter streaks of intrafascial cellular reaction at site of small intrafascial ruptures (arrows). Note decreased size of fibrofatty loculi as compared with Figures 13.5 and 13.6. DPC = distal palmar crease.



Fig. 13.8 Van Gieson stained longitudinal section of thickened superficial longitudinal pretendinous band showing zone of fibril discontinuity, collagen disaggregation and remodelling forming early cellular nodule.

numerous fibres ascending from it to the skin. Every interstice is filled with them, every bending of the hand causes shifting of these fatty particles within distinct limits'.

Madelung then clearly identified the probable function of the fatty pads, stating: 'it is clear that here as well as in other parts of the body, the function of the fat is to moderate pressure to which tissues of that part of the body are exposed and to

distribute it over a larger area'. Since that time there have been few brief mentions of the existence of these subdermal fatty loculi, notably by Skoog (1948), Iselin (1955) and Thomine (1965). Millesi (1959), having meticulously picked out the small fatty globules from between the fine fibres of the superficial palmar fascia, was obviously well aware of their presence and indicated that they had a protective cushioning function.

Napier (1965) described the main fat pads of the palm under three headings — the thenar, the hypothenar and the metacarpophalangeal — and their function in accommodating the hand to grip. However, he specifically mentioned that the area of the palm overlying the palmar aponeurosis was relatively fat-free and formed the central anchorage for the skin and the rest of the palm. It may be that his observations were based on dissections of older hands in which the subdermal fat had disappeared. Certainly Madelung (1875) had also noticed the marked loss of subcutaneous fat in the mid-palm of older subjects; with great foresight he suggested that its absence could lead to increased stress on the more prominent fibres of the palmar fascia. In contrast to this, Iselin (1955) considered that the subcutaneous fatty pads were replaced as a *secondary* event by infiltration

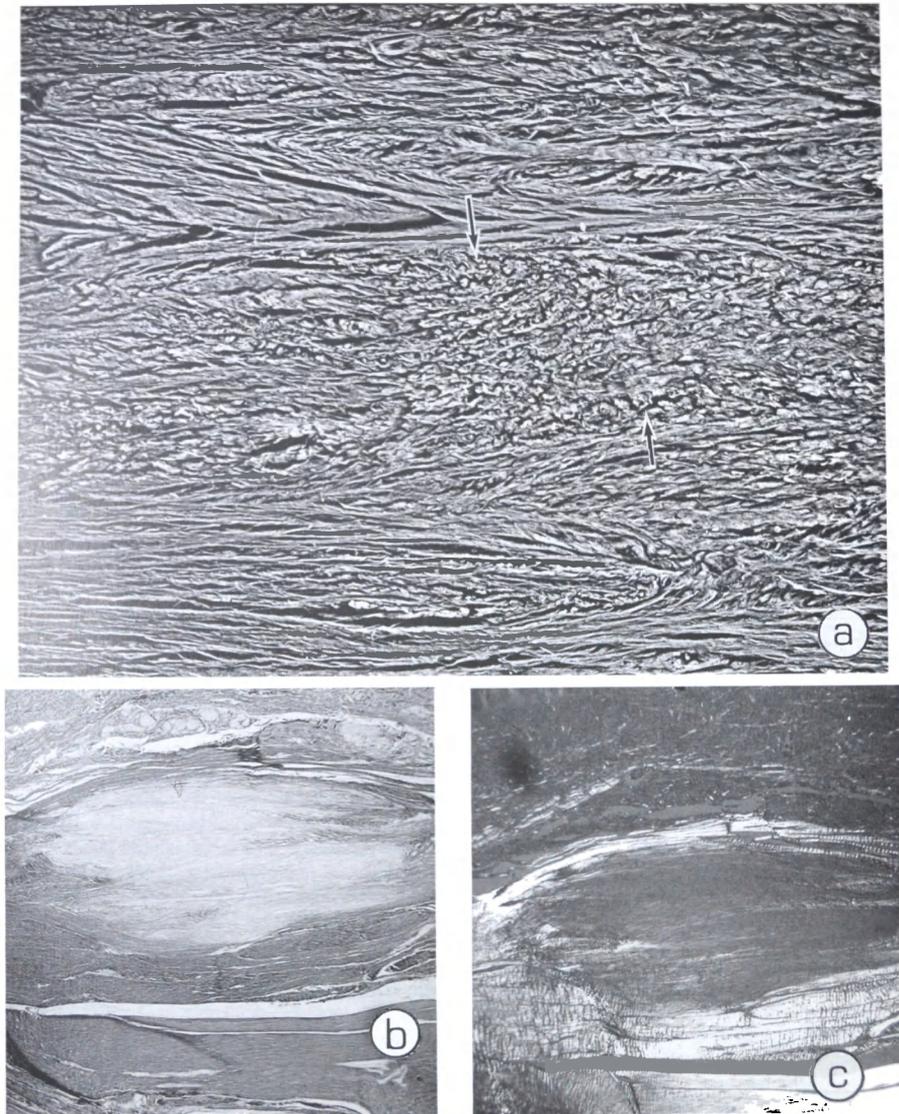


Fig. 13.9 a Small areas of cellular proliferation and fibril discontinuity observed in scanning electron microscope study of clinically unaffected longitudinal pretendinous fascial bands. b Longitudinal section of intrafascial cellular zone in longitudinal pretendinous band demonstrating fibril discontinuity across the lesion but fibril continuity peripherally. c Polarized view of same specimen showing fibril discontinuity within the central cellular zone but peripheral continuity.

of the Dupuytren's lesion extending superficially from the volar surface of the fascia. This idea is also expressed in many other writings dealing with the pathogenesis or histopathology of Dupuytren's disease (Meyerding et al 1941; Iselin 1954b; Mac-Callum & Hueston 1962; Nezelof 1985).

During the course of a comparative histological study of weight-bearing and non-weight-bearing skins (Gillard et al 1977), marked differences were observed in the size, shape, extent and distribution of the subdermal fibrofatty loculi in the palmar skin. At first it was thought that these differences were age-related, but further study demonstrated that this was not so. However, these studies indicated that there was an inverse relationship between the thickness and integrity of the subdermal fat and the thickness and compaction of the superficial longitudinal fibre bundles. In other words, loss or absence of the fat pads seemed to be associated with an increased thickness or compaction of the subdermal connective tissues and particularly of the superficial longitudinal pretendinous fascial bands.

The frequency of these histological observations prompted a more intense study of the structure of the superficial palm occupying the zone between the under-surface of the epidermis and the volar aspect of the thick, deep, pretendinous longitudinal fascial bands. In Chapter 2 this zone was referred to as the 'palmar connective tissue continuum' to emphasize the functional and structural integrity of the subepidermal and subcutaneous layers.

In surveying the vast literature that has accumulated on DD over the past few decades we have been struck by the paucity of information about the biology or even the morphology of the tissues within this important functional layer. There are numerous papers dealing in minute detail with the palmar fascia and the mode of its involvement in Dupuytren's disease (Luck 1959; Gosset 1967; Stack 1971; McFarlane 1974, 1985; Tubiana et al 1982; Gosset 1985; Strickland & Bassett 1985), but most of these, with the exception of papers by McGrouther (1982) and Millesi (1959, 1965, 1985), pay scant attention to this superficial layer of skin, fat and fibres, whilst providing exhaustive descriptions of the pretendinous fascial bundles, their extensions into the fingers and the effect of these

on the development of deformity in DD. This is understandable because clinicians tend to be more interested in the treatment of the deformity and thus in the anatomical basis of the contractural bands. However, to understand the enigma of DD it must be appreciated that whilst the disease may sometimes start on or within the volar surface of the deep pretendinous bands of the palmar fascia, it probably more frequently has its beginnings in the connective tissues lying between these deep pretendinous bands and the epidermis, i.e. in the 'palmar connective tissue continuum' (see Chapters 2 and 28).

INTERRELATIONSHIP OF FASCIAL FIBRES AND FIBROFATTY LOCULI

The underside of the dermis is normally pegged into the underlying subcutaneous tissue by a honeycomb of fibrous compartments which on longitudinal section of the palm appear as fibrous bands running between the dermis and the pretendinous bands (Figs 13.6 and 13.10). Some of these vertical and oblique bands form septae between the fibrofatty compartments of the hypodermis and provide the hydroelastic shock-absorption system protecting the palmar connective tissues and the deeper neurovascular bundles and tendons from excessive compressive loading and shear stress. These fibrofatty compartments are particularly well developed in the palm proximal and distal to the distal palmar crease.

In longitudinal sections taken along the line of the fourth metacarpal ray, the fibrofatty pads proximal to the palmar crease are seen to be round or ovoid and packed closely together, being separated from each other by vertical or oblique fibrous septae (Figs 13.10 and 13.11). In fortunate planes of histological section of these palmar skin samples it is possible to observe that the fibrofatty compartments are separated into a superficial and deep group by the superficial pretendinous longitudinal fascial band (McGrouther 1982; Figs 13.6 and 13.11).

Distal to the distal palmar crease the superficial longitudinal band becomes progressively more superficial and blends with the dermis. Throughout its length this fascial bundle is surrounded by a

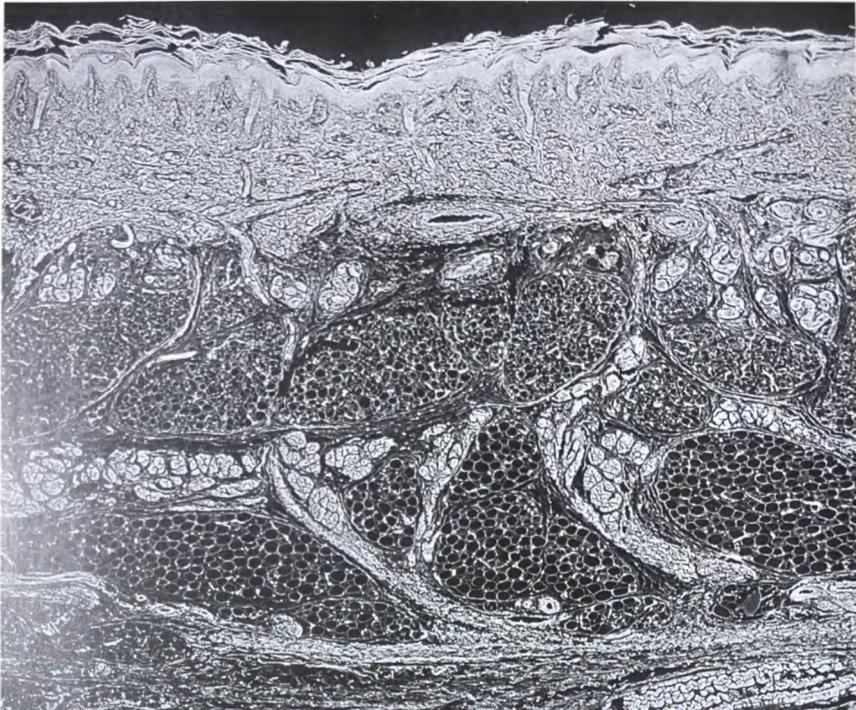


Fig. 13.10 Scanning electron microscope photomicrograph of longitudinal section of 1-day-old palm skin showing fibrofatty loculi, fibrous septae and attachment of the dermis to the longitudinal fascial strands by oblique and vertical fascial bundles.

thin paratenon-like sheath, indicating that there is normally a range of movement of this microtendon during flexion and extension of the hand and fingers (McGrouther 1982; Figs 13.5 and 13.11). Distal to the distal palmar crease, the fibrofatty compartments are often much larger, more ovoid and less compacted than those of the proximal group. The fibrous septae between them are also derived from vertical and obliquely running fibres extending between the dermis and the volar surface of the tendon sheath or the deep longitudinal fibres of the palmar fascia (McGrouther 1982; Millesi 1985).

There is considerable variation in the size and distribution of these fibrofatty compartments. In some instances the layer of fat pads may be twice or three times as thick as the dermis, whilst in others the dermis may equal or exceed their thickness. Initially, in our studies, it was thought that the difference in the size of the fibrofatty compartments was associated with ageing, but study of a large number of samples demonstrated that the differences were not age-related and that it was possible to have a very thick layer of fat pads in old people or alternatively pads that were almost completely obliterated in young people. We were



Fig. 13.11 Scanning electron microscope photomicrograph of longitudinal section of 1-day-old palm skin showing superficial longitudinal bundle (SLB) approximately $100\ \mu$ wide running between superficial and deep layers of fatty loculi within its paratenon sheath.

surprised to find the extent to which the fat pads were sometimes almost totally replaced by fibrosis in the palm and by the fact that this could happen over a wide age range.

At birth, or shortly thereafter, the fat pads are arranged like a closely packed honeycomb (Fig. 13.10). However, in some instances by early adult life or even in adolescence, the fibrofatty compartments in the specimens we examined had apparently been replaced by progressive fibrosis which appeared to have its origins in the loose connective tissue around the fatty loculi. This fibrosis was generally preceded by increased vas-

cularity within the fatty loculi and by marked perivascular round cell response. Figure 13.12 shows the dramatic differences which were found between birth and early adolescence, with marked disruption of the fatty loculi and loss of the normal fibrous configuration. Figure 13.13 shows the increasing fibrosis and the gradual obliteration of the fatty loculi.

Fibrotic replacement

In many instances there was obvious fibrotic replacement of part or all of the deep or superficial

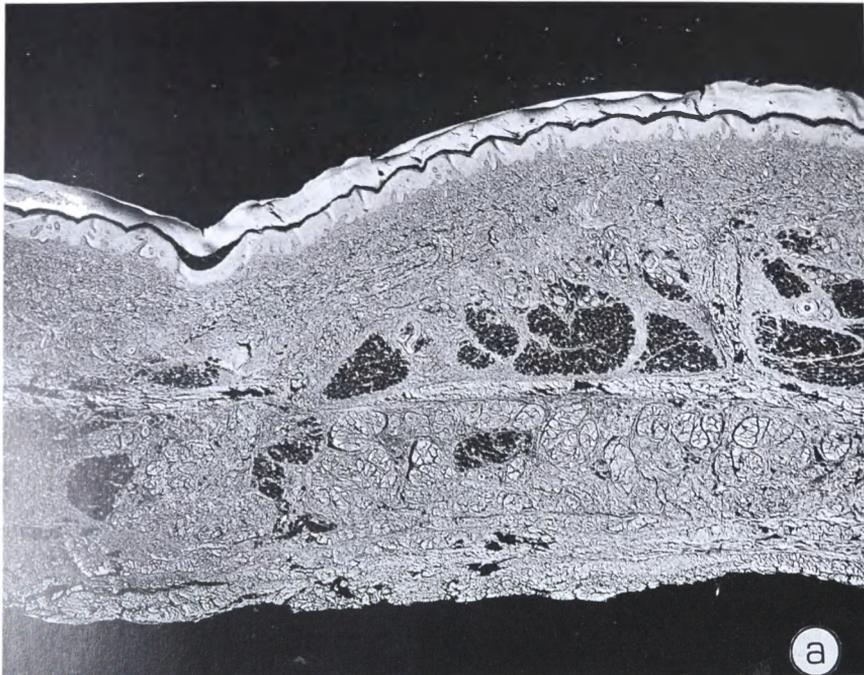


Fig. 13.12a Scanning electron microscope photomicrograph of longitudinal section of palm skin of a 15-year-old male. Note the marked relative decrease in size and integrity of fibrofatty loculi as compared to Figure 13.10. b Scanning electron microscope photomicrograph of longitudinal section of palm skin of an 18-year-old male. Note the marked fibrosis of palmar connective tissue and loss of fat as compared to Figure 13.10 at the same magnification.

layer of the fat pads proximal to the distal palmar crease. Examination of more than 250 samples of palm skin demonstrated a pattern of fibrous replacement which appeared to begin in the deeper layer just proximal to the distal palmar crease and which progressively extended backwards, i.e. proximally, throughout the whole of the deep layer. Fibrotic replacement of the deep layer of the proximal group of fat pads often appeared to be followed by fibrotic replacement of the superficial layer, progressing in a similar manner proximally from the distal palmar crease (Fig. 13.13).

Although it is not possible to be certain that

these progressive changes always took place in this order, it was unusual to find isolated fibrotic replacement of the proximal or superficial fat pads without involvement of those adjacent to the distal palmar crease. It was also unusual to find replacement of the superficial layer if the deep layer fat pads were still intact.

Increased vascularity

The fibrotic replacement of the fibrofatty compartments appeared to be preceded by, and associated with, increased microvasculature of the

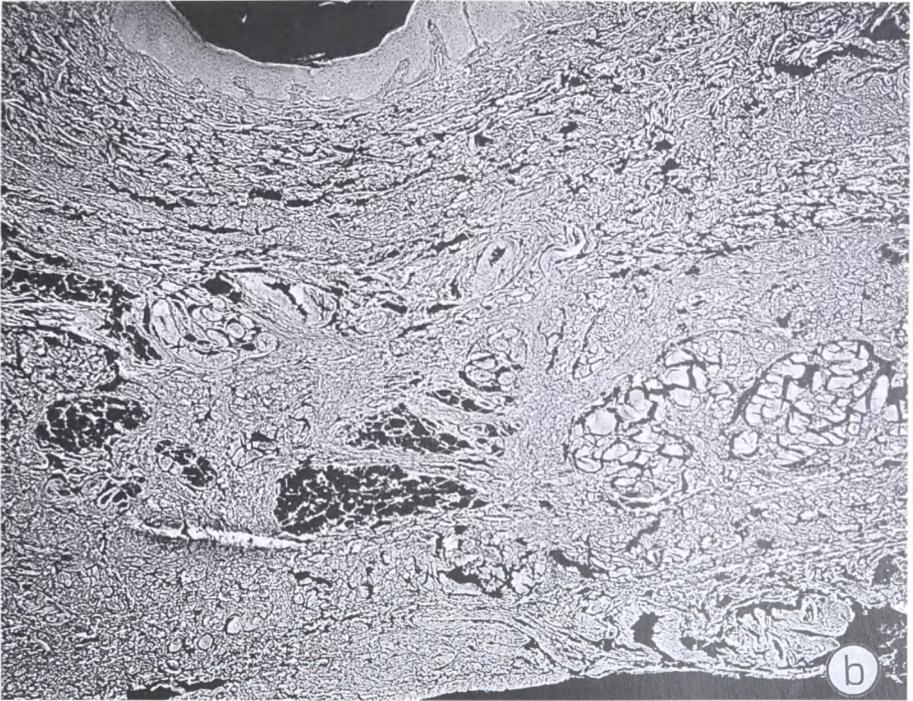


Fig. 13.12b

connective tissues within and between the fatty compartments and by perivascular fibroblastic activation (Fig. 13.14). Sometimes the replacement was associated with perilobular fibrosis and there often appeared to be periacinar and periductal fibrosis spreading centrifugally from the sweat ducts. The increased vascularity and perivascular reaction observed at the onset of the fibrotic replacement agreed with the observations of Meyerding et al (1941) but was at variance with the suggestion of Kischer & Speer (1984) that the excessive fibrosis of Dupuytren's contracture and hypertrophic scarring is due to, or associated with, tissue anoxia, occurring as a result of microvascular occlusion (Kischer et al 1982).

In ideal circumstances the young fibrofatty loculi exhibited well formed fat cells with intact

microseptae surrounded by a thicker peripheral fibrous boundary composed of collagen and elastic fibres (Figs 13.10 and 13.11). The progressive fibrosis of the shock-absorbing system in the palm skin was associated with disaggregation, fragmentation and clumping of the elastic fibres in the fibrous septae between the fatty loculi and also with changes in the microarchitecture of the collagen fibres. Scanning electron microscopic photographs of specially prepared paraffin-embedded sections of palm skin graphically demonstrated marked disaggregation of the fibrous walls and septae of the fatty loculi and dense compaction and thickening of the collagen fibre bundles (Fig. 13.15). These findings support the observations of Millesi (1985) of compaction and changes in the viscoelastic properties of some of

the collagen fibres in the superficial palm before the onset of any of the classical features of Dupuytren's disease.

Consequence of fibrosis

Although no biomechanical studies have been

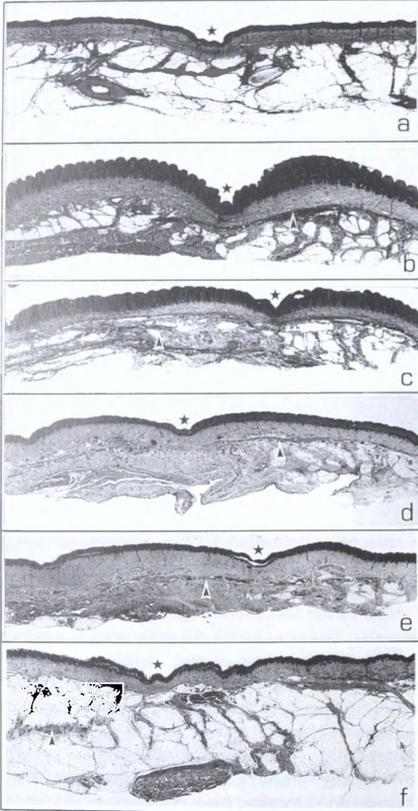


Fig. 13.13 Variations in distribution of fibrofatty loculi in longitudinal sections of palm skin taken along the fourth ray of individuals of various ages: a 17-year-old female; b 18-year-old male; c 16-year-old male; d 83-year-old male; e 80-year-old female. Star = Distal palmar crease; arrow = superficial longitudinal bundle, where discernible. f Intact fibrofatty loculi in an 83-year-old female, showing that the loss of fibrofatty loculi does not inevitably occur during ageing.

done to assess the resilience or compliance of fibrotic palms as opposed to those containing normally intact fat pads, it is likely that the progressive loss of the fibrofatty shock absorbent layer would be followed by progressive alterations in the distribution and dissipation of pressure and shearing forces in the palm. These changes would be compounded by the loss of free movement of the fascial microtendons within their paratenon sheaths which would follow their involvement in the progressive fibrotic process (McGrouther 1982). Compressive, or more particularly, compressive shearing forces applied to regions of the palm unprotected by an efficient elastic recoil system, especially in regions of greatest anteroposterior mobility, as in the fourth and fifth rays, could produce marked alterations in the longitudinal fibrous elements of the palmar connective tissue continuum.

As long as the protective insulation of the fat pads remained relatively intact and the longitudinal fascial fibres were still relatively mobile, increased compressive or shearing load on the palm would induce increased tensional loading on the longitudinally running fascial fibres leading to their compensatory thickening and hypertrophy. On the other hand, once the compressive absorptive capacity of the fat pads had been more profoundly affected by the increasing fibrosis, the longitudinally running collagen fibres would become less compliant and less able to slide or deform to accommodate the applied stress. This would be compounded by the glueing together and compaction of the fibre bundles which follow inflammatory oedema. These various factors would subject the fibre bundles to localized areas of high stress loading. The biological effect of this is discussed more fully in Chapter 26.

Although collagen fibres are extremely strong in transmitting or withstanding tensional forces they are notoriously weak when subjected to compressive or shearing stress (Parry 1988). It is for this reason that the collagen fibres in cartilage are embedded in a proteoglycan-rich matrix which absorbs and dissipates the compressive loads. If the longitudinally running collagen fibres in the superficial pretendinous bands are deprived of their protective fat pads, or the concentration of the glycosaminoglycan hyaluronate within the fibre bundles decreases (Flint et al 1982), making them

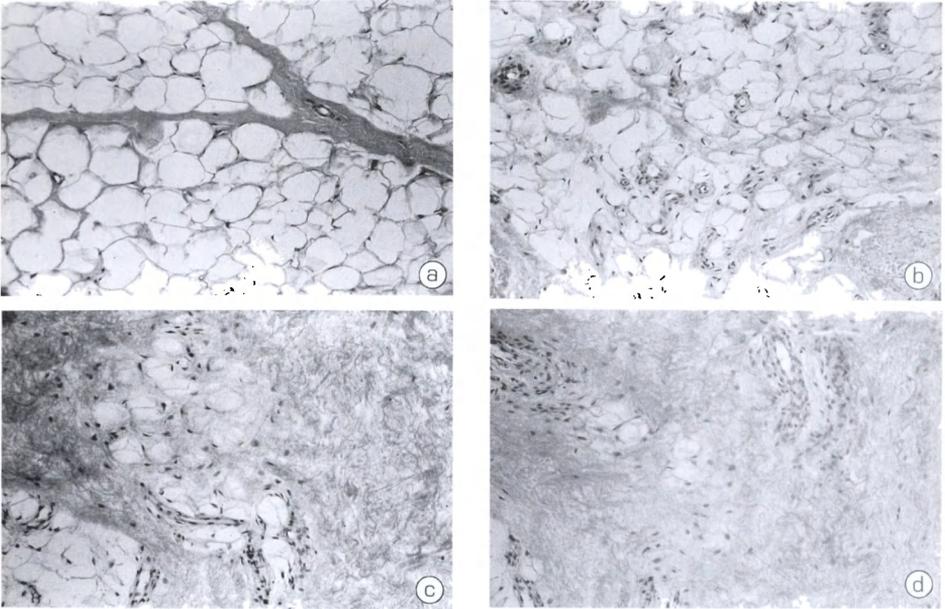


Fig. 13.14 Vascularity and fibrosis of fatty loculi. **a** Normal fatty loculi; **b** increasing vascularity amongst subdermal fibrofatty loculi in the palm of a 15-year-old male; **c** perivascular cellularity and fibrosis in fatty loculi in another area of the same palm; **d** area of fibrous replacement with faint ghosts of fatty loculi.

less compliant and stiffer, they become more vulnerable to these vertically applied compressive and shearing loads. As a consequence they are more likely to sustain stress or fatigue fractures and cracks, especially when the longitudinal bands are repeatedly flexed by the impact loading.

I believe that the palmar lesions of DD arise as a direct consequence of one or other of these biological responses. It has been well documented in other biological situations that the application of increased tensional loading to longitudinally running collagenous structures such as tendons inevitably leads to concomitant progressive increases in the thickness of the tension-transmitting collagen fibres (Buck 1953; Viidik 1967, 1968; Tipton et al 1967, 1970, 1975; Arem & Madden 1976). Such progressive thickening of the longitudinal fibre bundles was very evident in our studies. Figure 13.16 shows the increase in thickness from the normally very thin superficial pretendinous band

of the child to the thickened palmar cords of the adult. It is apparent from our studies that the thickened cords run in the same direction and between the same anatomical layers as the thin superficial pretendinous bands and that they are in reality variations of one and the same structure. The observation of cellular nodule formation within these thickened bands (Figs 13.1 and 13.9) demonstrates that the DD process, on occasions at least, has its origins within abnormally thickened, but normally occurring, anatomical structures (Stack 1971; McFarlane 1974, 1985; Tubiana et al 1982).

Histopathological nodule development

In previous chapters it has been noted that the nodular areas which develop in the thickened fascial cords appear to be similar in biochemical content and cellular morphology to hypertrophic



Fig. 13.15 Scanning electron microscope photomicrograph of the palm of an 18-year-old male showing loss and disaggregation of most fatty loculi and marked fibrosis of deep layers of the palm.

scars. Careful consideration of all the available data indicates that they are likely to be areas of reparative response or at least active tissue remodelling which develop as the result of a wound healing repair *within* the microtendon-like structure of the fascial bundle. How do these changes come about?

I believe that the changes in the physical characteristics and biomechanical properties of the palm — either by loss of the protective fat pads or by changes in the physicochemical and biomechanical properties of the fat contained within the fibrofatty lobules — will lead to an increased loading of the longitudinally running strands. In some instances, the increased intermittent tensional loading may simply stimulate

collagen synthesis and collagen accretion by giving rise to increased fibril and bundle diameters (Parry et al 1982). However if the physical properties of the collagen fibres are less than ideal, perhaps due to changes in nutrition, changes in intrafascial hyaluronate levels, the effects of frictional heating (Harkness 1961, 1979, Rigby 1977) or the local accumulation of free radicals (Murrell et al 1987a, b; Outhwaite et al 1988), or if the increased tensional loading is more sudden and acute, there may be localized tearing or shredding of some of the collagen fibres within the substances of some of the longitudinally running bundles. If the tearing or shredding produces complete separation of the fibres, leaving a gap between the fibre ends such as is found following division of a tendon, one

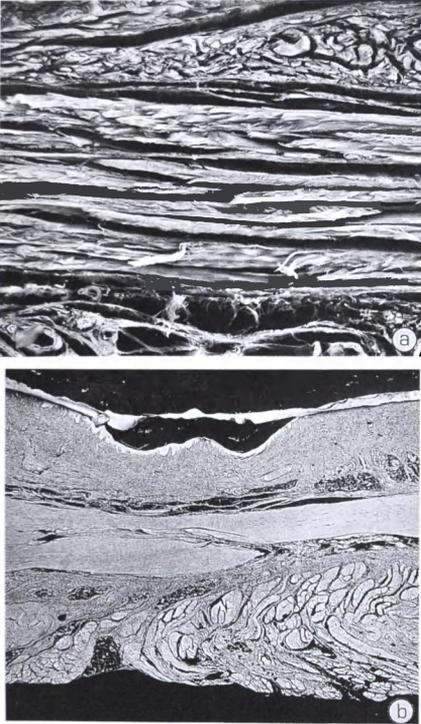


Fig. 13.16 a Scanning electron microscope photomicrograph of 3-month-old child: superficial longitudinal fascial strands approximately $100\ \mu$ thick lying within paratenon sheath. **b** Thickened superior longitudinal fascial band approximately 2 mm thick in 67-year-old male.

would anticipate that the normal processes of tendon repair would be set in motion with division and migration of cells from the paratenon-like structure covering the fascial strand. In this way the fibril continuity would be re-established without nodule formation, under the stimulation and guidance of the tensional forces (Fig. 13.17).

However, if the tearing of the fibres within the bundle was only partial, leaving an intact outer rim of fibres around an internal tear (Figs 13.9 and 13.18), then the repair process would be subject to entirely different forces. Cells coming into the reparative area would not then be subjected to

longitudinal tensional forces and as a result would tend to form a diffuse cellular mass which would gradually increase in size to produce an obvious thickening within the fibre bundle. As the tensional forces would be still transmitted along the intact fibres in the periphery of the damaged bundle, the cells in the centre of the divided repair area would be deprived of the necessary controlling sensory input. In fact, they would probably be subjected to increased *compressional* forces induced by the pressure and constraint of the intact outer fibres as they were placed under tensional load (see Fig. 13.19). As a result, a cellular hypertrophic area would develop within the cord reminiscent of the hypertrophic callus which develops around a poorly immobilized limb fracture. As the developing wound repair lying within the substance of the palmar fascial bundle would be deprived of uniaxially directed tensional forces, the cells within their reparative area would continue to divide (hence their dense aggregation), and would continue to synthesize increased amounts of glycosaminoglycans, particularly chondroitin sulphate, and type III collagen. This situation would be continually self-perpetuating unless it was interrupted by changes in the physical loading of the tissue, or by treatment such as triamcinolone injections aimed at halting or reversing the cellular reaction (Ketchum 1971).

Research in other areas has indicated that tendons or skin wounds which are subjected to uniaxial longitudinal tension along their length, with little or no distractive tension from either side, are likely to contain lesser amounts of total glycosaminoglycan and lower levels of chondroitin sulphate (Flint et al 1980). The fact that the nodular areas of Dupuytren's contracture have quite high levels of total glycosaminoglycans with a greatly increased proportion of chondroitin sulphate adds weight to the suggestion that the repair tissue within the nodule is not subject to longitudinal tensional forces as one might expect, but is subject to laterally applied compressional forces.

Our previous research work on tendons and sesamoids (Gillard et al 1979) demonstrated that applied compressional forces encourage the development or persistence of round chondrocytic-like cells which synthesize increased amounts of glycosaminoglycans, particularly chondroitin sul-

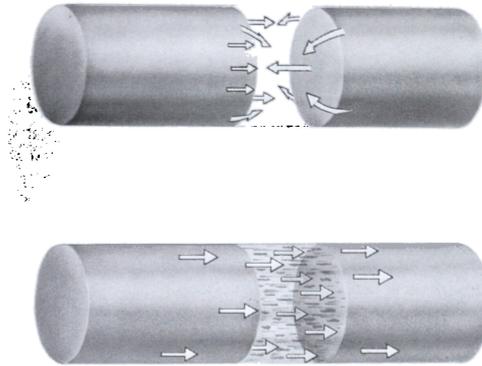


Fig. 13.17 Complete division of tension-transmitting fibres stimulates cellular organization and repair of the gap (top) which becomes organized and aligned by uniaxial tension forces (bottom).

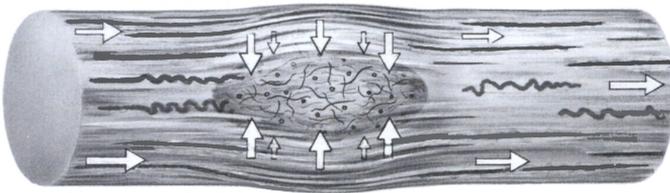


Fig. 13.18 The result of an incomplete intrafascial (or intratendinous) central tear or fibre division: fibres are intact peripherally. The central repair would lack the uniaxial tensional forces necessary for cellular and scaffold alignment and would be subject to additional lateral compressional forces from the intact peripheral longitudinal fibres.

phate, with the result that there is less compaction and organization of the collagen fibres (Merrilees & Flint 1980). In the Dupuytren's nodule the greatest increase in glycosaminoglycan is in the chondroitin sulphate fraction. This and the chondroid morphological features of the cellular response support the suggestion that the tissues within the nodule are subjected to lateral com-

pressional forces as a result of the longitudinal tensional forces still exerted on the peripheral fibres (Fig. 13.20).

Previous research has demonstrated that the red staining of collagen fibres of mature intact cords by the Masson trichrome procedure indicates that they are subject to longitudinal tension (Flint & Lyons 1975; Flint et al 1975) whereas the retention

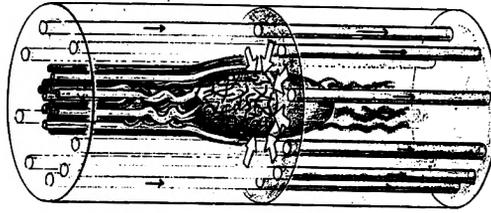


Fig. 13.19 Centripetal pressure forces acting on the intrafascial repair will perpetuate the lack of longitudinal orientation of cells and collagen fibres and stimulate continued synthesis of chondroitin sulphate and type III collagen.

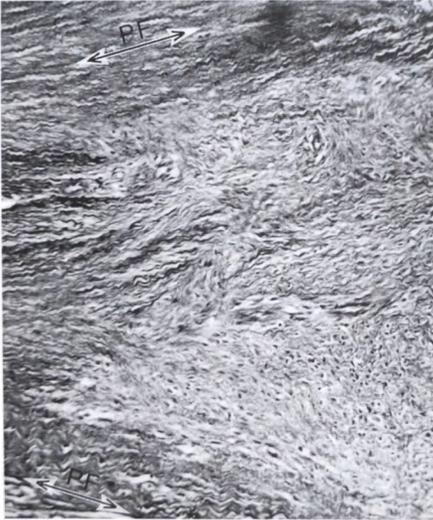


Fig. 13.20 Photomicrograph of longitudinal section of edge of intrafascial lesion in superficial longitudinal band showing peripheral fibre (PF) continuity, central fibre discontinuity and chondroid-like cells within the reparative zone.

of the green dye of the Masson trichrome procedure by collagen fibres indicates that they are not subject to these forces. In many Dupuytren's nodules and bands the red Masson staining of the

mature fibrous cords confirms their tensional state whilst the green staining of collagen fibres within the nodules or in the bands adjacent to the nodule supports the concept that these fibres have been severed at the level of the nodule and demonstrates that there is little or no uniaxial tensional force acting upon them. Polarized light studies also demonstrate the disaggregation and separation of collagen fibres in the centre of the nodules with the maintenance of a rim of old intact fibres around the periphery (Fig. 13.21).

It is now over 40 years since Tord Skoog postulated that the cellular nodular areas developed in regions where the collagen fibres had been disrupted (Skoog 1948, 1963, 1967). Unfortunately, his ideas and those of supporting workers such as Larsen (Larsen et al 1960) have been severely criticized (Hueston 1975, 1985) and almost rejected. It is now over 100 years since Madelung (1876) suggested that as a result of the loss of the subdermal fat,

single regions of the palm would become more exposed to a constant pressure, become more easily injured, especially those which are situated opposite the firm part of the bony structure of the hand, e.g., the heads of the metacarpal bones and the flexor tendons. The frequent injurious exposure evidently excites the now unprotected tense connective tissue of the palm to a state of chronic inflammation, leads to hyperplasia of the normal fibres and finally to their shrinking with consequent permanent bending of the

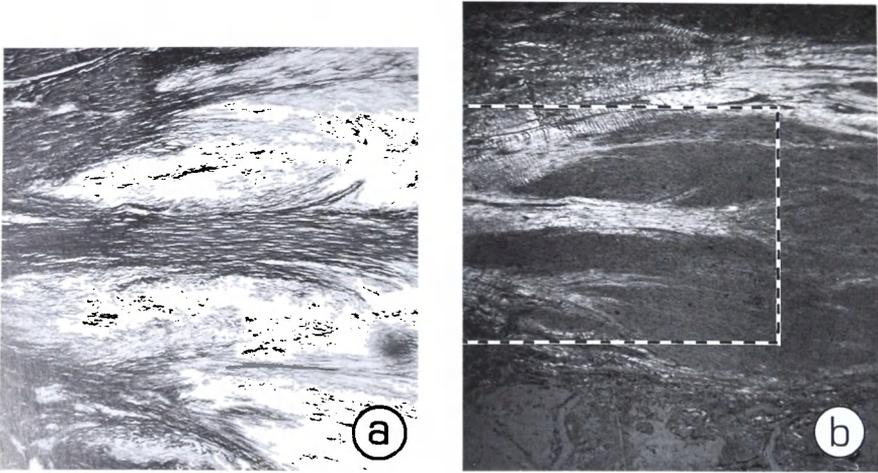


Fig. 13.21a Photomicrograph of Van Gieson stained histological section of the superficial longitudinal band showing a composite intrafascial lesion partly divided by a band of fibres. These fibres probably previously served as the peripheral fibres of two adjacent lesions which have become confluent as the result of secondary fibril rupture. b Adjacent section, viewed through crossed polarizing prisms, shows the composite nature of the lesion with residual birefringent collagen fibres surrounding the island of frustrated repair. The field demonstrated in Figure 13.21a is indicated by dashed lines on Figure 13.21b.

fingers . . . and that the numerous consequent exposures to injury will act more and more on the individual diseased part and incite it to further change. (Madelung, 1876).

Whilst we acknowledge that DD may have a multifactorial basis, we feel that it is now essential to reconsider the role of mechanical factors in the induction of the palmar lesion. We believe that

with the application of biological principles to an understanding of DD we are now able satisfactorily to integrate the observations of Dupuytren, Goyrand, Madelung, Meyerding, Skoog, Hueston, Millesi, McFarlane, McGrouther and Flint into a more meaningful and understandable integrated montage.