THE PATHOLOGY OF DUPUYTREN'S CONTRACTURE*

By SIR PETER MacCALLUM AND J. T. HUESTON

Repatriation General Hospital, Heidelberg, Victoria

THE enigma of Dupuytren's contracture remains largely unsolved. For further progress in the study of its aetiology and treatment it is felt necessary to revise the present concepts of its pathology as it is almost universally held that Dupuytren's contracture is primarily a disease of the palmar aponeurosis.

After observation of more than 700 patients with Dupuytren's contracture, the operation findings in 200 fasciectomies and the histological study of specimens freely selected from these cases, we conclude that, although the palmar anponeurosis is intimately involved in Dupuytren's contracture, this represents only one aspect of a change which may occur in any part of the network of palmar connective tissue and is usually secondary to changes arising within the fibrofatty tissue on its superficial aspect.

MACROSCOPIC OBSERVATIONS

Because of individual differences in the natural history and in the operative findings in advanced cases, we believe that a study of the earliest detectable changes sheds most light on the true nature of the condition. Either one of two changes may be seen as the earliest sign of Dupuytren's contracture. In most patients there is first a palmar nodule (Fig. Ia) but, in a few, a short interphalangeal band producing flexion deformity of the proximal interphalangeal joint (Fig. Ib).

The palmar nodule is usually attached to the superficial aspect of the palmar aponeurosis in the line of the ring finger, is frequently tender on pressure and at times the site of spontaneous pain. In 10 per cent. of patients nodules were found in relation to the thumb, either overlying the radially-inserted intrinsic muscles or near the free margin of the first web space.

Surgical exposure of a palmar nodule demonstrates that the nodule is in the subcutaneous fibro-fatty layer (Figs. II, III, IVb). Its deep fixation is usually to the palmar aponeurosis but at the margins of the palm it is sometimes impossible to demonstrate deep fixation, such nodules lying wholly within the subcutaneous fatty layer (Fig. II).

In the early case with the nodule fixed to the palmar aspect of the aponeurosis, it fuses with it but never protrudes on the deep aspect of the aponeurosis. This constant appearance of the palmar nodule protruding from only the palmar aspect of the aponeurosis suggests a lesion applied to it rather than arising from it. The poorly defined margins of most nodules which during dissection demonstrate fixation at all points to the fibrous strands of the normal palmar subcutaneous layer, suggests an origin from the deeper elements of this layer nearest to the aponeurosis (Fig. III).

A distinctive palmar nodule occurs remote from the anatomical aponeurosis, at the ulnar border of the palm, overlying the tendon of insertion of abductor digiti minimi (Fig.

2

IVa, b). It is this nodule which may occasionally be demonstrated at operation to be free of deep fixation. Nodules of the dense poorly circumscribed fibroplastic tissue referred to hereafter as Dupuytren's tissue, have also been dissected from the subcutaneous tissue overlying the pisiform bone and flexor retinaculum. Such occasional freedom of deep fixation of the marginal palmar nodule supports the suggestion that the aponeurosis is not the essential structure responsible for this disease.

FIG. I (a).   Solitary palmar nodule in the line of middle finger with no band or flexion deformity.

The interphalangeal band is a much less common presentation of this disease. When interphalangeal joint flexion is the first sign, dissection usually reveals a dense, discrete band, 2 to 5 millimetres in diameter passing deeply from proximal to middle phalanges and intimately related to the digital neurovascular bundle. At times it is continuous with the extensor expansion or the joint capsule. These bands are fixed to the phalanges without any palmar connections and are free of nodules or local adhesions to the fatty tissue of the finger. The often tortuous course of the neurovascular bundle around these interphalangeal bands is due to their origin from the connective tissue supporting the neurovascular bundle, which is then displaced by the shortening of the developing bands.

Natural history

The palmar nodule may remain stationary and show only a skin callosity from pressure in a working man. Other nodules may appear in the palm or in the proximal segments of one or more fingers, the latter showing incomplete deep fixation in their early stages. Confluence of adjacent foci in the central
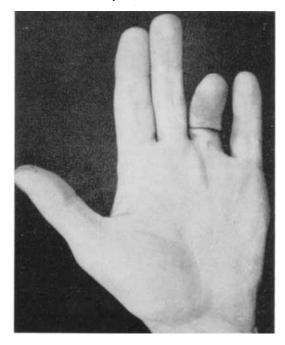

FIG. I (b).   Flexion deformity due to interphalangeal band.   The palm is clear.

palm may produce a raised plaque across which the flexure creases pass deeply and over which the dermis becomes fixed. This appearance is commoner in women and often does not progress to a flexion deformity, which may explain why fewer operations are required in women (Fig. V).

Over three or four years the palmar changes become slowly more discrete and the tissue becomes firmer and whiter than the early poorly defined greyish nodule. This

shrinkage of the diffuse palmar nodules to denser more discrete bodies, leaving the intervening palmar tissues once more apparently free of disease, is not regarded as a disappearance but rather as an aggregation of the formed tissue.

FIG. II. A dense discrete nodule of Dupuytren's tissue at the ulnar border of the palm, lying entirely within the subcutaneous fat without any deep fascial or aponeurotic fixation.

No definite instance has been observed by us of a palmar nodule disappearing, although this has been reported by careful observers including Gordon (1948) and Iselin (1954). Such observations must be of considerable importance when the aetiology is considered.

Flexion deformity has not been observed in the presence of a palmar nodule alone. The band is responsible for the principle feature of Dupuytren's contracture but a nodule precedes the appearance of such a palmar band. In progressing cases, an opaque fibrous thickening may be seen on the palmar aspect of the aponeurosis for one or 2 centimetres around the palmar nodule. In the line of the relevant digit, this roughened aspect of the aponeurosis becomes raised up to be continuous with the developing band. An early band is usually well developed and tendinous proximal to the nodule but distally it is opaque with fat lobules incorporated in its surface and although this aggregation of palpable thickened tissue usually extends to the proximal or middle phalanx it is often difficult to define by naked eye from the adjacent palmar fibro-fatty tissue. The developed band, continuous with the aponeurosis, stands forward several millimetres and lies close beneath the skin (Fig. VI).
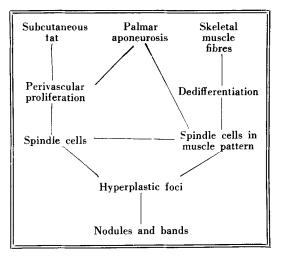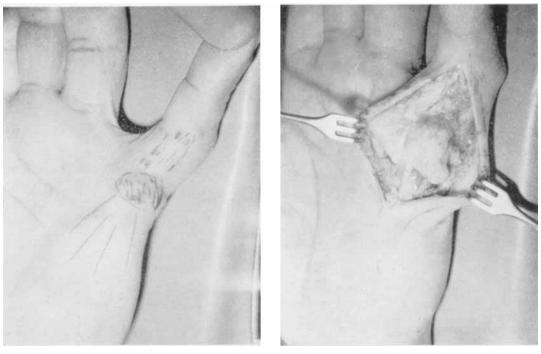

FIG. III. A palmar nodule and developing band to little finger. The palmar nodule can be seen applied to the superficial aspect of the palmar aponeurosis and the ill-defined new fibrous tissue of the developing band is clearly within the subcutaneous fibro-fatty layer.

Considerable tension is developed in a band when the finger is forcibly extended. On incising such a band from its superficial aspect the ends spring apart and retraction makes the cut ends concave. This sudden separation of the cut ends of the band is sometimes found to occur before the whole thickness has been divided. It is significant that the remaining deepest part of the band, which was clearly not contributing much to the contraction, can be seen to be the plane of the original palmar aponeurosis. This indicates that the principal site of contracture is not aponeurosis itself but the newly-formed superficial mass of tissue.

TABLE 1

PALMAR CONNECTIVE TISSUES

Subcutaneous fat	Palmar aponeurosis	Skeletal muscle fibres
Perivascular proliferation		Dedifferentiation
Spindle cells		Spindle cells in muscle pattern
	Hyperplastic foci	
	Nodules and bands	

(a)   (b)

FIG. IV (a) and (b). A nodule overlying the insertion of abductor digiti minimi. When exposed, the nodule is clearly a development from the overlying subcutaneous fibro-fatty tissue.

The deep aspect of the palmar aponeurosis remains smooth and free from the diffuse changes and attachments of its palmar surface except for the deep inter-tendinous septa which pass to the metacarpals and support the common digital neurovascular bundles. These septa, which are the principal skeletal attachment of the palmar aponeurosis, become very thickened in those advanced cases with flexion deformity of the fingers. No nodules have been observed in these septa. Discrete dense tendinous bands continuous with the distal end of a thickened inter-tendinous septum are sometimes found passing from the neck of the metacarpal to the proximal phalanx, grossly displacing the neurovascular bundle in some cases and causing a flexion deformity to persist after excision of the more obvious pretendinous band. In appearance and structure these deep bands resemble the inter-phalangeal bands of the fingers. Bone is occasionally found in the insertions of an old band into the phalangeal margin.

The fat on either side of a band shows varying degrees of fibrous replacement, ranging from slight in the proximal palm to almost complete in the region of the nodule which is found to have attachments in all directions, not only proximally to the skin and distally into the digital skin and skeleton, but dorsally to the metacarpophalangeal joint capsule and radially in many cases across the web spaces to adjacent fingers. A nodule may thus be seen as the centre of a field of radiating fibrous bands and septa. The nodule overlying the abductor digiti minimi tendon is often a spectacular example of this centripetal fibrosis and contraction as it puckers the skin by dermal involvement, flexes the proximal interphalangeal joint by its longitudinal fascial attachments, abducts the little finger when adherent to the abductor digiti minimi tendon and finally may hyperextend the terminal interphalangeal joint by involvement of fibres passing into the extensor expansion.

Nodules arising within a finger are found, on dissection, to be merging freely into the subcutaneous fat of the finger. It is often difficult to demonstrate any discrete fascial attachment of the digital nodules except in the advanced case.

Skin pits are common in Dupuytren's contracture being directed proximally with the apex fixed to a palmar nodule or band. Although no true contraction of the skin occurs, some fixation of the skin is usual over palmar nodules. Pits are commonest near the distal palmar crease but occur elsewhere if the band has a strong dermal attachment. After subcutaneous fasciotomy the skin resumes its normal dimension showing that it has been only passively included in the contracture.

Knuckle pads overlying the dorsum of the proximal interphalangeal joints of the fingers were present in over 40 per cent. of patients requiring fasciectomy. On the few occasions when excision of these lesions was required, a plane of dissection was found between the knuckle pad and the extensor tendon (Fig. VII), indicating its origin from the paratenon and not from the tendon itself.

FIG. V. A diffuse plaque of Dupuytren's tissue which has been in the central palm of a woman for years without producing bands or contracture.

Recurrence of Dupuytren's contracture after excision is common in those patients who require operation while the disease is rapidly progressing and particularly if they have a strong family history. Such recurrences may arise in the cleared area of the palm and fingers despite meticulous excision of aponeurosis and abnormal tissue (Hueston, 1961). Recurrences in the finger require secondary excision more often than those in the palm and at operation have macroscopic features no different from a virgin case. These findings in recurrent Dupuytren's tissue indicate that, as it could not have arisen from any pre-existing anatomical fascial bands, it must have developed from the fibro-fatty tissue remaining in the region. The relative freedom from contracture of palmar recurrences suggests that normally contracture occurs through involvement of the anatomical longitudinal fibres of the aponeurosis. The proximity of recurrent digital tissue to tendon sheaths and phalanges may account for the continued tendency for a flexion deformity to be produced.
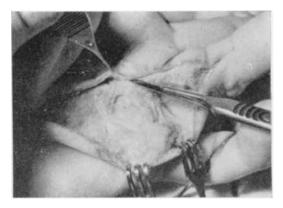

FIG. VI. Operative dissection of a band to ring finger demonstrating that the band has formed within the subcutaneous tissue and lies anterior to the palmar aponeurosis plane. The dermis is firmly adherent along the surface of the band.

Conclusions from macroscopic observations

The earliest observed change, the palmar nodule which may be multi-focal, appears to arise within the palmar connective tissues immediately overlying and adherent to the palmar aponeurosis.

Centrifugal fibrosis from the nodule through the network of palmar fibrous structures produces fixation to both skin and skeleton.

Contraction is preceded by the formation of bands which are raised in the line of maximum tension on the palmar nodule and by irregular involvement of the anatomical network of palmar fascia.

The appearance and behaviour of recurrent Dupuytren's tissue is consistent with its origin from fibro-fatty tissue rather than remnants of the palmar aponeurosis.

lagenous material and showing no mitoses at all. The appearance of these nodules on section consequently varies enormously in the same specimen, sometimes within a very short
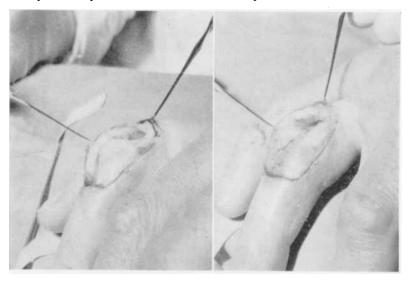

FIG. VII. Dissection of a knuckle pad demonstrates the freedom of direct Involvement of both dermis above and extensor tendon beneath.

MICROSCOPIC OBSERVATIONS

The histological picture typical of Dupuytren's contracture is a combination of fibroblastic proliferation and collagen formation in which these 2 phases of activity may be freely intermingled or separated into hyperplastic cellular foci and dense collagenous bands. The distribution of the fibroplastic activity within the one specimen may vary from large areas within a nodule to scattered patches along the length of a palmar band or islands among the fat loculi of the distal palm and fingers.

The structure of a hyperplastic cellular area is characteristic, consisting of fleshy nucleated fibroblasts arranged in parallel or in elliptical foci merging peripherally with collagen bundles but often very clearly demarcated from these by intervening vascular septa. The cellular elements of the bundles vary from closely set, relatively short, plump, parallel spindles with oval nuclei, very little intercellular collagen and sometimes fairly numerous mitotic figures, to elongated narrow nuclei widely separated by much col-

space. On the assumption that the signs of proliferative activity precede those of differentiated maturity, maturity means age. A repetition of the process over several months or years gives rise to the mixed picture seen in this disease.

The collagen deposition is mainly in the direction of the principal band and in many specimens the plane of the original palmar aponeurosis is preserved intact on the deep aspect of each palmar specimen so that although its structure is found to become incorporated into the main mass of new tissue, this has been formed on its palmar aspect, there remaining on the deep aspect of most specimens a plane of uninvolved loose fatty and areolar tissue. This is not seen in the distal palmer nor digital specimens where the limit of the change is poorly defined in all directions. The peripheral changes are seen in most cases to pass into the fatty tissue on the palmar and lateral aspects and often to include areas of fat within the distal part of the dissected operation specimens. This only confirms the macroscopic appearance.
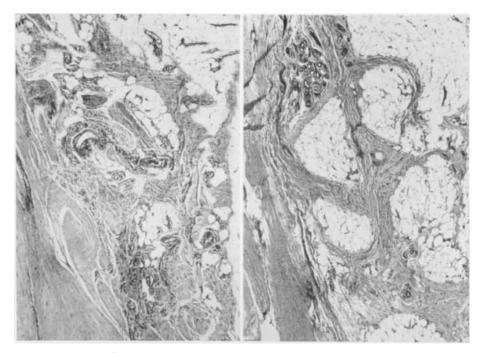
FIG. VIII (a). Increased vascularity and perivascular infiltration in the subcutaneous fat at the margin of a palmar nodule. (x 50)

FIG. VIII (b). Progressive replacement of subcutaneous fat by new fibrous tissue adjacent to palmar band. (x 50)
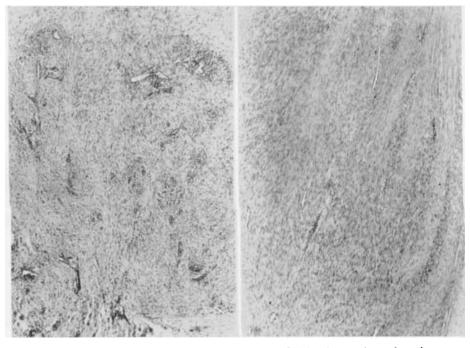
FIG. VIII (c). A young hyperplastic cellular area showing the perivascular arrangement of the new tissue. No fat remains. (x 50)

FIG. VIII (d). A maturing palmar hyperplastic cellular area showing orientation along the line of digital tension. (x 50)

The change of greatest interest in the adjacent and included fibro-fatty tissue is an increased vascularity (Fig. VIIIa). The new vessels are small calibre arterioles and capillaries. They are frequently thick-walled and sclerosed and lie between the fat loculi where they are surrounded by sheaves of small round cells and new fibroblasts so that a gradual fibrous replacement of fat loculi by peri-vascular proliferation can be observed (Fig. VIIIb).

Within the hyperplastic foci the new fibroblasts are arranged around sheaths of branching blood vessels (Fig. VIIIc). As the fibroblasts mature the nuclei become elongated and separated by increasing amount of collagen and orientate themselves parallel to the major surviving vessels which are found mainly in the line of longitudinal stress (Fig. VIIId) but may run with the vessels in abruptly differing directions. The directions of the fibres is most disturbed where there is nodule formation.

Although the perivascular infiltration may form small round cell collections, this is consistent with the normal picture of tissue proliferation and does not signify an inflammatory response. No giant cells were seen. No polymorphs were found nor any eosinophils to suggest an allergic factor, although the general pattern of change was more consistent with a local allergic response than any other type of change.

The fibroblastic invasion and replacement of the palmar fat is regarded as the clue to the production of Dupuytren's tissue. The sequence can be followed from vascular invasion and perivascular cellular proliferation to maturing nodule and finally to a relatively acellular dense atrophic tendinous band. The centrifugal spread of this proliferative process from an initial palmar nodule along pre-existing septa to the skin and along the palmar aponeurosis and through the fibro-fatty connective tissue of the palm into the digits is accompanied and followed by a maturation which slowly produces contraction of the newly-formed elements. Only when these have fixed skeletal attachments does actual deformity follow. Atrophy is a later stage.

Examination of mature palmar bands and the dense interphalangeal bands revealed a scattered fibroblastic process between heavy collagen bundles (Fig. IX) which clarify the enigma of band production without any obvious preceding nodule formation referred to in the macroscopic appearance. Slit-like spaces (Fig. IX) are present in many hyperplastic foci and bands and although some contain blood cells there are many which do not contain blood and are not lined by endothelium but may be tension artefacts of contraction.

FIG. IX. Longitudinal section of a palmar band showing scattered hyperplastic foci and slit-like spaces better developed than usual. (x 50)

Longitudinal section of a palmar band occasionally reveals a most instructive picture, a deeper layer of dense acellular collagen with a diffusely hyperplastic zone extending from this layer to the plane of dermal dissection (Fig. X). The deeper layer represents the original aponeurosis often much thickened. The superficial hyperplastic zone has replaced the layer of fibro-fatty tissue normally present in this situation.
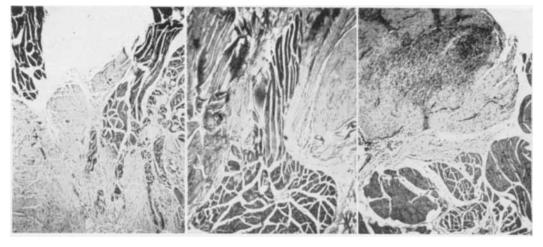

FIG. X. Longitudinal section of a palmar band showing the hyperplastic recently formed layer overlying the more mature formed layer of the palmar aponeurosis. (x 25)

The earliest macroscopic nodules have been described as sometimes free of adhesion to the aponeurosis and the tissue of origin is seen histologically to be the heavily vascularized fibro-fatty tissue of the palm immediately overlying the aponeurosis. The relation of the newly-formed tissue to the skin is consistent with the replacement rather than displacement of the subcutaneous and dermal elements. Thus the newly-formed tissue may pass directly into the vascular dermal papillae overlying a nodule, surrounding Pacinian corpuscles and sweat glands which may be separated from the overlying dermis by 2 or 3 millimetres of newly-formed tissue.

vascular control and, with this vascular enlargement, further enquiry may confirm a functional relationship.

Micro-haemorrhages or small deposits of iron pigment are seen in about 1 specimen in 10 of Dupuytren's tissue, usually in the hyperplastic foci. The pigment may be intra- or extra-cellular and is interpreted as evidence of past interstitial haemorrhage. These findings do not represent anything more than the fragile nature of any recently formed, as yet undifferentiated area of fibroplasia and need not be seriously considered as an aetiological factor in the development of Dupuytren's contracture. They are to be

FIG. XI. (a) Specimen from operation seen in Fig. IV. showing loss of normal staining properties of fibre bundles within surface layer of abductor digiti minimi. (x 50)
(b) Same specimen showing further dedifferentiation of skeletal muscle to fibrous tissue. (x 50)
(c) A typical hyperplastic nodule of Dupuytren's tissue on the surface of the same muscle. (x 50)

Broadbent (1955) has drawn particular attention to the conspicuous enlargement up to 3 or 4 times normal size of the layered Pacinian corpuscles which are part of the sensory afferent division of the sympathetic, for pressure perception, proprioception and deformation sensation. These enlarged corpuscles lose their myelin sheaths. Corresponding to the enlargement of the Pacinian corpuscles there is an accompanying regional thickening and sclerosis of the arteries and arterioles supplying the affected area. This suggests a derangement of the reflex for local

regarded not as the cause but as the result of the recent fibroplasia.

Finally, examination of specimens in which the nodule at operation was adherent to and merging with formed skeletal muscle masses such as palmaris brevis, abductor digiti minimi or the abductor pollicis brevis, has demonstrated an interesting series of changes which can be interpreted as the conversion of striated muscle fibres to Dupuytren's tissue. The transformation from muscle to connective tissue is first seen in a change of the staining

character of the muscle. In the middle of the muscle bundle the fibres alter in their characteristic staining, with Masson's stain from brown to blue; with Van Gieson from yellow to red; with haemotoxylin and eosin from red to very pale pink and with phosphotungstic acid from purple to light brown. Intermediate mixtures of these stainings are also seen with each stain. Simultaneously with the change there occurs a loss of cross-striation which seems to be the first differential character to disappear (Fig. XIa).

There follows one of two lines of dedifferentiation, one an extensive laying down of dense colagenous tissue between connective tissue nuclei with pointed ends and wavy outline (Fig. XIb) and the other, the proliferation of cells with ill-defined margins and plump rounded nuclei showing one or more nucleoli with hyperchromatic mitotic figures (Fig. XIc). These also lay down collagen and the nuclei ultimately assume the same form as those of the other group. In each case there is an increase in bulk of tissue, the fibres swell and fuse and the bundles become in fact characteristically staining connective tissue bundles. About the cells collagen is deposited in greater or less amount. The cells straighten out and become parallel, widely separated, narrow and relatively fewer and bands begin to form as the stress is taken up. The rest is shrinkage and deformity, producing increasing stress and work hypertrophy of the band as suggested by Luck (1959).

Conclusions from microscopic observations

A sequence can be demonstrated from increased vascularity of fibrous network in the palmar fat, with perivascular cellular proliferation replacing fat loculi, to the production of hyperplastic foci and finally to the deposition of mature collagen in bands.

In Dupuytren's contracture such a process commences on the palmar aspect of the palmar aponeurosis and extends centrifugally along the formed fibrous elements of the palm, including the aponeurosis with its intertendinous septa, the normal fine fibrous septa to the palmar skin and into the fingers.

Maturation leads to shrinkage and contracture. The deep bands in the fingers and palm appear to have scattered fibroplasia rather than aggregation into nodules and their close relationship to the digital skeleton explains the production of early and severe contracture even in the absence of palmar nodules.

The transformation of superficial striated muscle fibres into dedifferentiated connective tissue and even into Dupuytren's tissue is suggested as a further facet of the general pattern of connective tissue behaviour in Dupuytren's contracture.

The presence of iron pigment is noted as an incidental finding. There is no evidence of inflammation, traumatic or infective.

DISCUSSION

The evolution of the knowledge of the structural changes in Dupuytren's contracture has been in 3 phases. Originally regarded as a contracture of the flexor tendons and termed "crispatura tendinum", the tendons were cleared of blame and the palmar aponeurosis indited by Astley Cooper (1822) and Dupuytren (1832). However, since then the origin of the condition has frequently been placed in the palmar tissues anterior to the palmar aponeurosis. Goyrand, as early as 1834, suggested this alternative to Dupuytren's explanation and also refuted the traumatic theory of origin but so great was Dupuytren's prestige that this opposing view was given scant attention.

Langhans (1887) first described the microscopic structure of Dupuytren's contracture and stressed the progressive replacement of the surrounding palmar connective tissue. This hyperplastic process in the tissue anterior to the aponeurosis was confirmed by Nichols (1899).

Although Anderson (1891) accurately described the pathology as an "inflammatory hyperplasia" commencing in the subcutaneous tissue of the palm, involving the skin and fascia secondarily and replacing the adipose connective tissue, this was ignored until restated by Meyerding, Black and Broders (1941). Nevertheless there is no current text which does not dismiss the pathology of Dupuytren's contracture as "a contracture of the

palmar aponeurosis" and it has been felt necessary to present again the structural changes in order to place correctly the seat and mode of progress of this condition.

The present study has demonstrated that the change in Dupuytren's contracture is primarily one of progressing perivascular fibrous replacement of the fatty palmar tissue overlying and ultimately directly involving the palmar aponeurosis. The contracture occurs principally in this newly-formed tissue but involves the palmar aponeurosis and the fibrous strands or septa associated with this structure. It has seemed a paradox that continued hyperplasia should be associated with shrinkage but coincident fibrosis in many areas of the new tissue accounts for the tension developed and the deformity occurring when this force acts across joints. As flexion occurs it seems from the slit-like spaces that the tissue planes of the band and nodule slide upon each other and the volume of the thickened, shortened band may be little more than the uncontracted band.

Trauma was blamed for this condition by Dupuytren and although this has been questioned frequently by careful observers since his time, particularly by Keen (1881), Anderson (1891), Black (1915), Corlette (1948) and Herzog (1951), nevertheless this is still the most widely held theory of aetiology. There is nothing in the histology to support a theory of reparative granulation tissue producing the hyperplastic nodules as was propounded by Skoog (1948) in his erudite but polemic publication. The presence of iron pigment is consistent with minor vascular ruptures occurring in the softer hyperplastic nodules during the normal stresses of everyday work and it is significant that there is rarely any evidence of inflammatory response in relation to this pigment. These micro-haemorrhages are to be regarded not as the cause but as the result of the recent fibroplasia. The description and illustrations used in Skoog's work to support the theory of fibre rupture are due to the diverging fibres which sweep around or through a nodule being divided obliquely in cutting the sections. No simple rupture of dense bands has been found and great force must have been used to produce such artefacts in Skoog's study.

Recent studies of the incidence of Dupuytren's contracture by Herzog (1951) confirm that manual workers are not more often affected than sedentary workers. Indeed there is evidence that diminished hand activity allows the appearance and increases the rate of progress of Dupuytren's contracture. In a study of Sudek's atrophy, which invariably is in a disused hand, Plewes (1956) found most patients developed thickening of the palmar tissues and if Dupuytren's contracture had been present before the injury it made rapid progress. Such observations along with the increased incidence observed in chronic invalids and alcoholics (Wolfe, Summerskill and Davidson, 1956; Hueston, 1960) make a theory based on palmar trauma untenable. On the other hand the increased palmar vascularity and perivascular proliferation described in the present paper are consistent with these incidence studies. It is not unreasonable to expect a change in the vasomotor control of disused hands with increased vascularity of the soft tissues as this has been long recognized radiologically in the osteoporosis of the skeleton of a disused limb.

There is no evidence of a true inflammatory basis either infective or traumatic and the histology is consistent only with a progressive fibroplasia from the peri-vascular cellular cuff. On the basis of extensive cadaver dissections, it has been suggested by Millesi (1961) of Vienna that the elastin, which has been noted as absent from the newly-formed Dupuytren's tissue, may be lost earlier also in the aponeurosis where nodules then form, possibly as a response to this change in aponeurotic structure. This is an interesting line of investigation at present in progress.

The theory of Krogius (1921) that the new tissue arises from residues of embryonic muscle in the palm has been recently revived by Stein, Wang, Macomber, Rajal and Heffernan (1960) on the basis of "bundles of spindle cells which take tinctorial shadings suggestive of muscle" in operative specimens. Although the observations of these workers provide no real evidence of the existence of this hypothetical muscle tissue, they do place the primary lesion of Dupuytren's contracture in the fibro-fatty tissue immediately anterior to the aponeurosis and help to substantiate the present concept. Dedifferentiation of

normal striated muscle fibres in the superficial palmar musculature is seen leading ultimately to its taking part in the production of Dupuytren's tissue. In order to appreciate the relationship between the pathology of skeletal muscle and the changes seen in Dupuytren's contracture it is necessary to go back to the basic observations of embryologists and histologists so admirably reviewed by Adams, Denny-Brown and Pearson (1954) †

Histologically similar hyperplastic tissue is common in the knuckle pads and plantar fascia of patients with a strong Dupuytren's diathesis. No contracture has been observed in these ectopic sites. The high proportion of hyperplastic tissue in these sites may be related to this difference in behaviour. No explanation is available for the location of this ectopic tissue which has been observed also in the tunica albuginea of the penis, the medial side of the great toe, the fascia lata and the flexor aspect of the wrist.

Recurrence of Dupuytren's contracture after fasciectomy has been blamed upon inadequate removal of the palmar aponeurosis and its extensions (Kanaval, Koch and Mason, 1928) but, particularly in the fingers, it is common to find recurrence within a field which has been thoroughly cleared of aponeurosis and Dupuytren's tissue (Hueston 1961). The only explanation for histologically identical tissue being reformed in an

---

†It is of interest that Dupuytren's contracture is not mentioned in that work. In the early stages of embryological development it is difficult to distinguish myoblasts from connective tissue and other mesenchymal cells. Even haemopoiesis may be met with in muscle in infants. Surgeons are familiar with recurrences of Dupuytren's tissue after "total" fasciectomy and this may possibly represent the further local development of tissue with that potential even though differentiated muscle may not be demonstrable. Nor would the development of retrogression with formation of a tendinous type of fibrous tissue with retractile properties of an intensity greatly exceeding those of normal fibrous tissue seem altogether strange. The collagenous fibrils of the connective tissue of muscle wind between the muscle fibrils and are characteristically elastic. The tendon connective tissue is quite rigid, even retractile and exhibits resistance to stretching exceeding that of bone or muscle. The peculiar properties of the fibrous tissue of Dupuytren's contracture are thus possibly accounted for.

adequately cleared area is that it has arisen from the locally remaining fibro-fatty subcutaneous tissue. No recurrence has been observed after Wolfe graft replacement in areas totally cleared of fat. The theories involving injury to fascial structures can no longer be put forward to explain the recurrence of Dupuytren's contracture, whereas the present findings of its structure and mode of development are supported by the very circumstances of recurrence.

CONCLUSIONS

Any of the palmar connective tissues may become involved in the progressive hyperplasia and fibrosis of Dupuytren's contracture. The palmar nodule formed by progressive perivascular spindle cell proliferation becomes the centre of a centripetally contracting field of newly-formed collagen.

The palmar aponeurosis is a major factor in the production of contracture only as it becomes involved in this process of collagen shrinkage. The French term "maladie de Dupuytren" is preferable to "Dupuytren's contracture" as it is a disease which may never proceed to the stage of contracture.

The transformation of subcutaneous fat to fibrous tissue is a prominent feature both macroscopically and microscopically and explains both the method of progress of Dupuytren's contracture and of its recurrence after excision of the normal aponeurosis and fascial system.

It is also suggested that the striated muscle fibres of the palmaris brevis and on the surface of the hypothenar and thenar muscles may become converted to spindle cells and then to Dupuytren's tissue.

The hypothesis is propounded that in the production of Dupuytren's contracture all the palmar connective tissues may, by dedifferentiation to spindle cells, contribute to the fibrous hyperplasia.

The enlargement of the Pacinian corpuscles associated with the vascular hypertrophy and arteriolar sclerosis is possibly of aetiological significance and should be further investigated.

ACKNOWLEDGMENTS

REFERENCES

ADAMS, R. D.; DENNY-BROWN, D. and PEARSON, C. M. (1954), "Diseases of Muscle"; New York, Paul B. Hoeber.

ANDERSON, W. (1891), *Lancet*, vol. 1, page 1.

BLACK, K. (1915), *Brit. Med. J.*, vol. 1, page 326.

BROADBENT, T. R. (1955), *Rocky Mtn. Med. J.*, vol. 52, page 1087.

COOPER, A. (1822), "A Treatise on Dislocations and Fractures of the Joints", page 524, first edition, London.

CORLETTE, C. E. (1944), *Med. J. Aust.*, vol. 2, page 177.

DUPUYTREN, G. (1832), "Lecons orales de clinique chirurgicale faite a l'Hotel Dieu de Paris", vol. 1, page 1.

GORDON, S (1948), *Canad. Med. Ass. J.*, vol. 58, page 543.

GOYRAND, G. (1834), *Mèm-Acad. Roy. de Mèd.*, vol. 3, page 489.

HERZOG, E. G. (1951), *Lancet*, vol. 2, page 1305.

HUESTON, J. T. (1960), *Med. J. Aust.*, vol. 2, page 999.

—— (1961), *Plast. Reconst. Surg.*, vol. 27, page 569.

ISELIN, M. (1954), *Rev. Rhum.*, vol. 21, page 203.

KANAVEL, A. B.; KOCH, S. L. and MASON, M. L. (1928), *Surg. Gynec. Obstet.*, vol. 48, page 145.

KEEN, W. W. (1882), *Philad. Med. Times*, vol. 12, page 370.

KROGIUS, A. (1920), *Zbl. Chir.*, vol. 47, page 914.

LANGHANS, P. (1887), *Zbl. Chir.*, vol. 14, page 497.

LUCK, J. V. (1959), *J. Bone Jt. Surg.*, vol. 41A, page 635.

MEYERDING, H. W.; BLACK, J. R. and BRODERS, A. C. (1941), *Surg. Gynec. Obstet.*, vol. 72, page 582.

MILLESI, H. (1961), personal communication.

NICHOLS, J. B. (1899), *Amer. J. Med. Sci.*, vol. 117, page 285.

PLEWES, L. (1956), *J. Bone Jt. Surg.*, vol. 38B, page 195.

SKOOG, T. (1948), *Acta. Chir. scand.*, vol. 96, suppl. 139.

STEIN, A.; WANG, M. K. H.; MACOMBER, W. B.; RAJAL, R. and HEFFERNAN, A. (1960), *Ann. Surg.*, vol. 151, page 577.

YOST, J.; WINTERS, T. and FETT, H. C. (1955), *Amer. J. Surg.*, vol. 90, page 568.