

DUPUYTREN'S CONTRACTURE FOLLOWING INJURY

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DURING a discussion of the process which now bears his name, Baron Dupuytren (1833) made reference, in Paris, in the year 1832, to "the multiple causes that have been attributed to it (Dupuytren's contracture), and the variety of remedies with which it has been treated. It has been ascribed successively to a rheumatic or gouty affection, to an external injury, a fracture, metastasis of a morbid cause which sometimes occurs after inflammation of the sheaths of the flexor tendons or to a kind of ankylosis. We shall find out that all these causes are unfounded."

The number and diversity of the theories of aetiology which still command support would suggest that time has done little to resolve the enigma.

Trauma, as Luck (1959) has pointed out, has been proposed both as the major contributing factor and as the sole cause of Dupuytren's contracture. On the other hand, the part played by trauma has been dismissed as having little significance. McIndoe and Beare (1958) stated unequivocally: "We know of no evidence, pathological or statistical, to support the view that trauma plays any part in the pathogenesis of Dupuytren's contracture." Other writers are content to state that the aetiological factors are unknown. Skoog (1948) has suggested that "overstretching the longitudinal fibres (of the palmar aponeurosis) by hyperextending the fingers . . . can produce partial ruptures of varying size and irregular distribution." As a result of his work, he concludes: "Trauma is a causal factor in cases with a predisposition to the disease."

In view of these diverse opinions, the case history which follows may merit some interest:—

Mr W. H., aged 48, while at work on 14th October 1959, forcibly hyperextended the mid-finger of his left hand. He immediately developed marked tenderness throughout the length of the finger and well into the palm. Shortly after the accident, he noticed a point of tenderness and a little bruising about the level of the distal transverse crease opposite the mid-finger. The discomfort and pain were such as to prevent him from extending the finger to its full extent. He was seen on 3rd December 1959, at which time there was palpable a slight subcutaneous thickening at the point where bruising had been noted. It was felt that a tear of the palmar fascia had resulted in a small hæmatoma, which was undergoing organisation and contraction. There was no family history of Dupuytren's contracture and there was no evidence of involvement of the rest of the palmar fascia of this hand or the palmar fascia of his other hand, or of either foot.

His hand was opened on 14th March 1960, under general anaesthesia. A pneumatic tourniquet was used. When the skin was turned back there was an area in the tissues anterior to the metacarpo-phalangeal joint of the mid-finger which was translucent, greyish-white, oedematous, and thickened in a nodular fashion. This area was excised.

Further dissection revealed a rupture of the flexor sublimis tendon to the mid-finger.

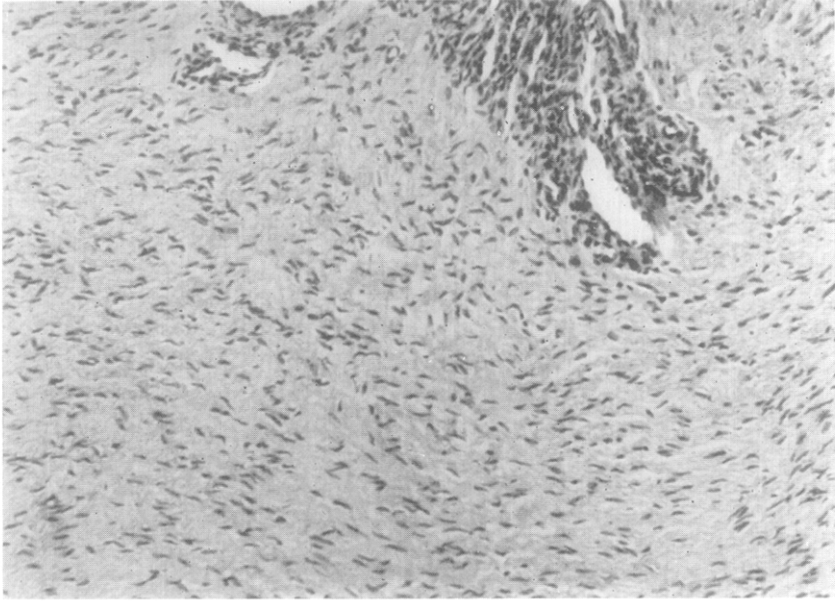


FIG. 1

Palmar fascia showing increased cellularity as the result of fibroblastic proliferation. At the top of the section a leash of small blood-vessels is present. (H. & E.)

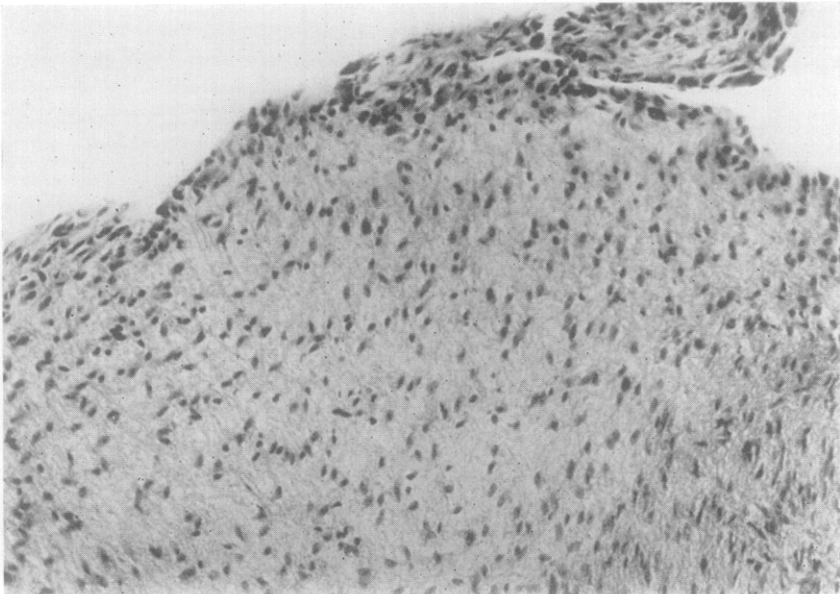


FIG. 2

The section shows part of the thickened tendon sheath from the inner aspect of which a number of rounded villous processes had developed. (H. & E.)

Histological examination of the excised tissues demonstrated the presence of a highly vascularised reparative process accompanied by areas of reactive fibrosis. As a result, both the tendon sheath and the more superficially located palmar fascia were considerably thickened. From the inner aspect of the tendon sheath a number of rounded villous processes had developed, their free margins poorly covered by synovial elements. In the palmar fascia the reactive process was characterised by a highly cellular fibroblastic proliferation which in some areas had assumed a distinctly nodular character. Striated muscle fibres which were involved in the proliferative reaction had undergone atrophy.

The histological features of the process within the palmar fascia were indistinguishable from those commonly encountered in the active phase of Dupuytren's contracture.

CONCLUSION

It would seem not unreasonable to regard the case recorded above as a clinical corroboration of the work of Skoog. While sweeping conclusions may not be drawn from a single case, the evidence suggests that traumatic rupture of the palmar aponeurosis may be followed by the development of a lesion similar, if not identical, to Dupuytren's contracture.

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