

### 3. HISTOLOGICAL ASPECTS OF DUPUYTREN'S CONTRACTURE

Christian Nézelof

If in many diseases microscopic examination gives a diagnosis and provides a number of data capable of indicating the aetiology of the disease, the histology of Dupuytren's disease only serves to confirm a clinically evident diagnosis without making any significant contribution as to its aetiology.

Our experience is based on the histological examination of 70-80 biopsies; these were taken at different stages of the disease in subjects from all age-groups. The biopsies were sent to us with an indication as to their topography, by our friend Raoul Tubiana.

They include:

- palmar nodules
- interphalangeal bands
- pieces of resected skin
- knuckle pads.

The histological picture shows a combination, in variable proportions, of fibroblastic proliferation and collagen accumulation.

It is possible roughly to distinguish two histological forms:

- (1) Lamellar thickening.
- (2) Nodular thickening.

(1) *Lamellar thickening* has a homogeneous texture with regular fasciculation.

Cellular density is low. The few recognisable cells are fibrocytes lying longitudinally between collagen fibres. The collagen fibres are wide, elongated, birefringent when seen through polarized light, and slightly arched. They are distributed evenly all through the biopsy, and here and there a strand is formed which runs into the fat and the adjacent tissues.

The more peripheral fibres seem larger and less arched. They often join to form a zone of hyaline sclerosis and then fail to take up the standard collagen stains. They are PAS negative.

The central fibres are thinner and more tortuous. They are PAS positive and only rarely metachromatic with toluidine blue. Silver impregnation shows the presence of a few argentophil fibres.

There are no elastic fibres.

(2) *Nodular thickening* is characterized by the presence of nodules of about equal size, roundish or oval shaped, and measuring 0.5 to 1 mm in diameter.

These nodules show a high cellular density. They consist of fibroblasts and fibrocytes arranged in an irregular, more or less concentric pattern.

The nodules are highly vascular. They are closely grouped together in the central part of the lesion. Rarely, they are found isolated near the periphery.

These nodules contain a thick network of fibres. The more easily recognizable components are argentophil fibres and young collagen fibres which are thin and tortuous, and stain readily with collagen stains and PAS. Between the fibres is the ground substance which is often metachromatic to toluidine blue. The fibres appear to originate from the nodules. As they leave the nodules, they unfurl and elongate, increase in size, and lie in criss-cross fashion, forming a dense complex mesh which, with the fibroblastic nodules, are the main components of the lesion.

(3) Between these two extreme histological forms, there are a number of intermediate forms, the frequency of which seems to increase the more sections one examines from the same biopsy.

In fact, the histological structure in Dupuytren's disease is very heterogeneous, lamellar and nodular lesions being encountered in adjacent areas. As a general rule, the palmar nodules are very cellular while the interphalangeal bands consist of dense hyaline fibrosis with only a scattering of cellular elements. The histological picture of the knuckle pads is more variable.

The two forms, lamellar and nodular, have been observed by most workers, some of whom believe that they have some prognostic significance. Meyerding was the promoter of this theory which he later defended.

It is difficult to agree with Meyerding about his conclusions on prognosis. From our studies it appears that the lamellar picture represents a late stage while the nodular lesions are indicative of a Dupuytren's of recent onset, or of a recurrence.

Apart from this observation, we failed to find a correlation between the histological picture, the stage of the disease, the age of patients and the presence of a particular susceptibility.

The examination of peripheral tissues provides some interesting information.

The subcutaneous fatty palmar pads situated anterior to the aponeurosis, are areas of hypervascularization. There is a dense network of capillaries and thick-walled arterioles, and one often sees normal segments surrounded by a proliferation of histiocytes and fibroblasts. Hueston believes that these areas are the starting point of the disease and of recurrent lesions.

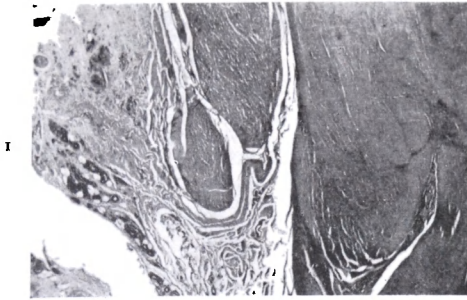


Figure 3.1  
Thickness of the band. Note its distinct superficial limit and the integrity of the dermis where sweat glands can be seen.



Figure 3.2  
Iron deposits around a capillary ( $\times 120$ ).

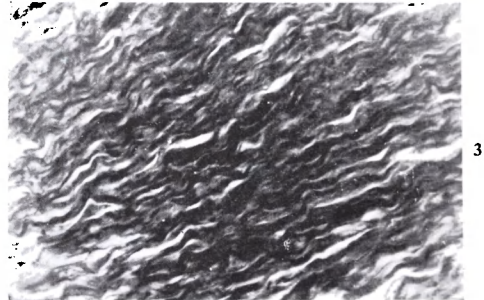


Figure 3.3  
Lamellar structure ( $\times 240$ ).

The sudoriferous glands and ducts are frequently surrounded by, and actually involved in, the fibrotic process.

But sub-fascial fatty tissue is always spared and marks the limit of spread of the lesions.

The Pacinian corpuscles are very hypertrophied, and yet this hypertrophy suggests no definite pathology.

All the skin biopsies examined came from pathological areas. They showed superficial keratosis and fibrosis

in the dermis, changes which, we believe, are extensions of the connective tissue lesion rather than lesions developing independently.

Histological records give little information on the starting point of the lesions. It is clear that the main changes occur in the anterior part of the aponeurosis and that the pathological process spreads superficially rather than in depth. As Skoog and Lagier reported, the transverse fibres of the palmar aponeurosis are spared for a

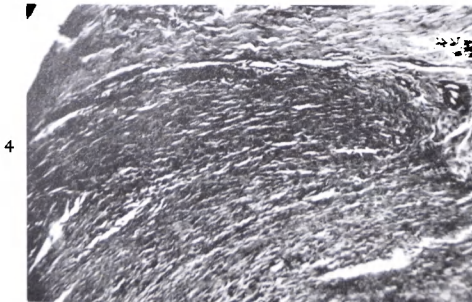


Figure 3.4  
Nodular structure; note the cellularity ( $\times 120$ ).

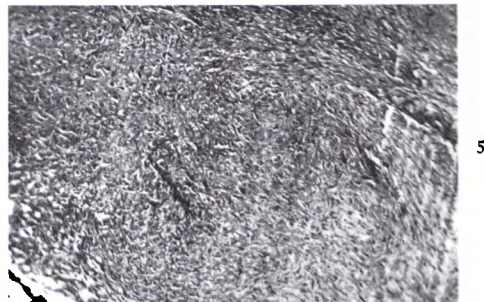
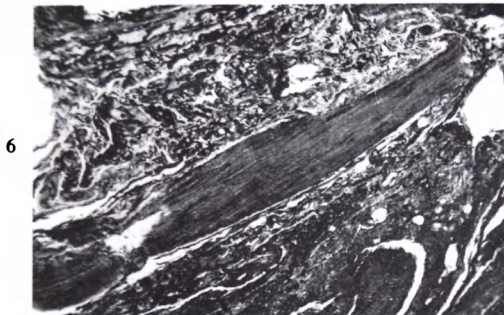
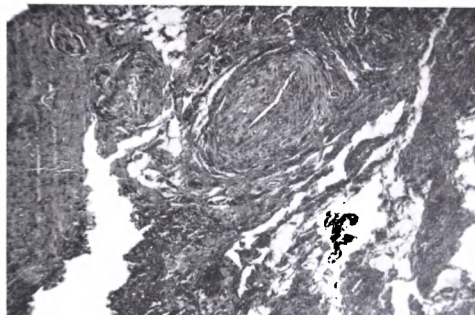


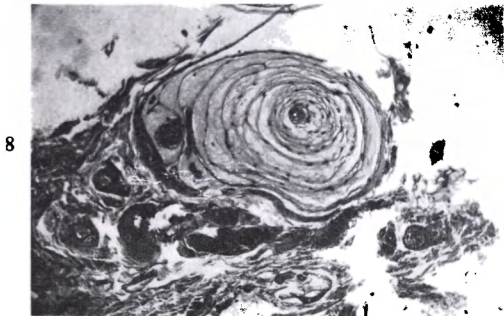
Figure 3.5  
Nodular structure ( $\times 120$ ).



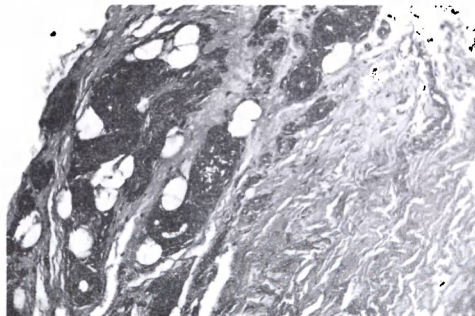
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Figure 3.6  
Shows absence of elastic fibres in the thickened aponeurosis  
and the abundance of areolar tissue (Wergert  $\times 60$ ).

Figure 3.8  
A Pacinian corpuscle in the dermis ( $\times 100$ ).

Figure 3.7  
Thick walled arteries.

Figure 3.9  
Deep dermal structure showing sweat glands adjacent to nodular  
thickening.

pathological changes, and we believe that the main lesions lie in the preformed connective tissue of the hand. The absence of elastic fibres in the pathological lesions seems to prove it.

Except in a small number of cases, we failed to demonstrate the presence of iron pigments. Unlike Skoog, we do not think that they are of any great significance or that they suggest micro-ruptures in the fibres.

On the whole, we can conclude that the contribution of histology in Dupuytren's disease is extremely disappointing. It does little else than confirm what was suspected clinically and observed at operation, namely the presence of *fibrosis*. It gives no clues as to the starting

point of the fibrosis, its histogenesis and its aetiology, long time. But it is difficult to agree entirely with Hueston's theory which claims that Dupuytren's disease is primarily a disease of prefascial adipose tissue which later extends to the palmar aponeurosis.

In our opinion, the hypervascularization, the venous stasis, and the presence of collections of round cells represent a reaction of the tissues rather than the initial

The more interesting observations are probably the absence of an inflammatory reaction, the absence of metamorphic changes in the ground substance, and the rarity of pigment deposits. These negative observations neither prove nor disprove existing aetiological theories.

## REFERENCES

- NEZELOF, C. & TUBIANA, R. (1958). La maladie de Dupuytren. Étude histologique. *Semaine des Hôpitaux de Paris*, 34 (18), 1102.