Basic Thoughts on Dupuytren's Contracture

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3.1 Introduction

Dupuytren's contracture has been known as such for nearly 200 years. It is a common disease in certain areas of the world. Patients suffering from the disease can have normal life expectancy. The natural course of the disease should be well known. However, our understanding of Dupuytren's has grown little in the last 100 years despite the fact that it is easy to observe and over the years has been the subject of an enormous amount of data collection, research studies, and publications. Apparently, there is something wrong with our basic concepts, which bear reexamination.

3.2 Critical Remarks

There are problems with the understanding of nearly every aspect of Dupuytren's disease (DD).

3.2.1 Etiology

Heredity is the most plausible one, but no gene causing DD has yet been defined. Racial disposition is believed, but poorly understood: is there a genetic risk for, or a genetic protection against Dupuytren's? (Slattery 2010). As far as metabolic diseases are concerned, there is a higher incidence of DD among diabetics or patients with liver diseases but the incidence is not high enough to establish a firm relationship. An enormous amount of effort has been spent to prove a relationship to certain professions or to trauma. There are arguments in favor and against but not enough to solve the problem of trauma as etiology.

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3.2.2 Pathology

Pathologists classify DD as a fibromatosis: it starts with a proliferation of fibroblasts which produce collagen and ends with cords consisting of nonstructured masses of collagen. Although this classification is reasonable, the term fibromatosis does not tell the whole story. Other diseases classified as fibromatosis such as keloid, juvenile fibroma, or desmoid tumor do not have much in common with DD.

3.2.3 Anatomy

It was an important step to recognize that the disease is at the level of the palmar aponeurosis and not in the flexor tendons. Although publications on DD often describe and include an image of the palmar aponeurosis as the site of the disease, this is misleading. The disease occurs in areas where there is no defined fascia, such as the palmar side of fingers. Is this then an "ectopic" occurrence of the disease, or is our description of the palmar aponeurosis too narrow?

3.2.4 Dynamics

Does the occurrence outside of the palmar aponeurosis mean that DD is a kind of tumor of the connective tissue, and if so, why does it not occur in articular ligaments or in tendons other than the most distal segment of the palmaris longus tendon? Dupuytren and Goyrand questioned the argument of the palmar aponeurosis as site of origin of the disease in favor of the skin. Even Hueston attributed an unknown DD factor in the skin and hypodermis and hypodermis and based the dermofasciectomy on this consideration (Hueston 1985).

3.2.5 Histology

In many papers one can read: "DD was confirmed by histological examination." It is doubtful that any histologist just seeing one or two images of histological sections without knowing the macroscopic appearance and clinical data is able to establish a conclusive diagnosis.

3.2.6 Biology

The main question remains: What causes the contracture? The discovery of myofibroblasts by Gabbiani seemed to offer an easy explanation (Gabbiani and Majno 1972). Today, we know that the myofibroblasts are not specific for DD and occur in many different conditions with or without contractures (Hinz et al. 2007). The increased occurrence of type III collagen was regarded years ago as characteristic for DD but it turned out to be a common feature in wound healing in general. Many other factors described in connection with DD may have more to do with cell proliferation or collagen production than specifically with DD.

3.3 Functional Biomechanics of Palmar Fascia

The glabrous skin of the hands and feet of primates has a unique functional anatomy. It is fixed to the underlying layers in a way that skin folds are fixed, but also, ideally suited for both loose and firm grip. The subcutaneous fat tissue is compartmentalized within a firm network of collagen fibers. Pressure from the surface is transmitted to the connective tissue network around the fat lobules, which absorbs and diffuses pressure.

The baboon hand is very similar to the human. It has large fat lobules at the bases of the fingers – corresponding to the interdigital palmar monticuli of the human hand – and on the thenar and hypothenar eminence to provide an intimate "surrounding" contact with the object (Fig. 3.1).

This functional consideration helps to understand that the stiff subcutaneous connective tissue of the palmar side of the hand and fingers is a functional system which facilitates different mechanisms of grip. The palmar aponeurosis is only one part of this system (Millesi 1965). The connective tissue framework between dermis and the musculature is a functional unit which varies with location: the dorsum of the hand; the cranial skin; the palm of the hand, and the fingers. This system is called the superficial fascia in contrast to the deep fascia around and between the muscles. This anatomy affects the different distribution of early sites of DD.

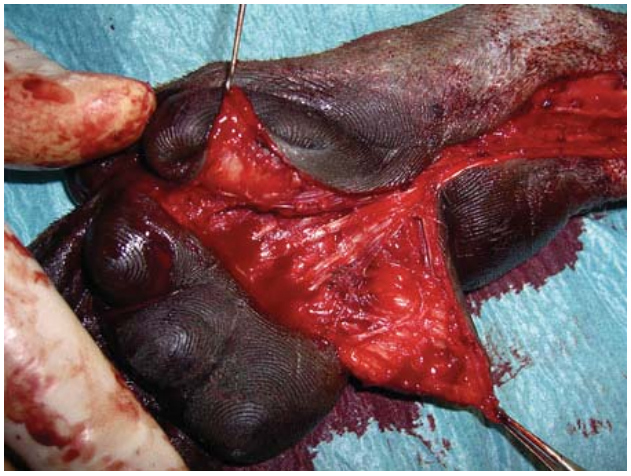


Fig. 3.1 Right palm of a baboon. The palmar connective tissue system functions to support grasping. Here, the skin of the palm is elevated toward each side. The pressure absorbing fat lobules with their connective tissue framework under the dermis at the incision lines can clearly be seen. In the center are the fibers of the palmaris longus tendon. With grasp, the palmaris longus contracts, and these fibers both flex the metacarpophalangeal joints and tighten the skin of the central palm. At the same time, the fat cushions on the radial and the ulnar side with their connective tissue framework close in around the object

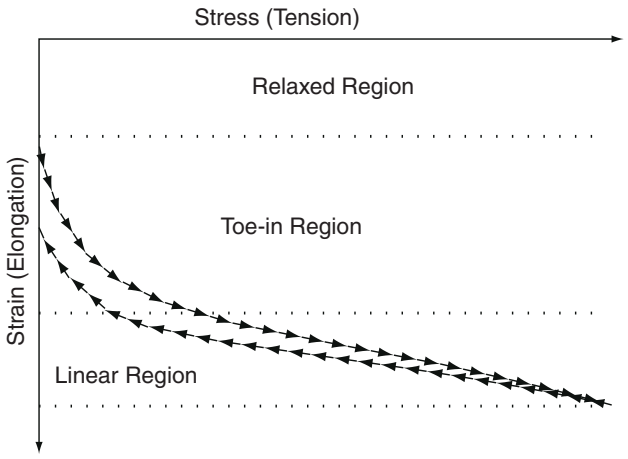


Fig. 3.2 Stress–strain test. The stress–strain test is a tool to define the viscous element in a viscoelastic system. *Stress* is the force applied; *Strain* is the length change from that force. Initially, a small force causes a significant lengthening as the wavy courses of the fiber bundles straighten (*up arrows*). Once this is complete, fiber bundle resist further elongation and the curve rises steeply. If the force is released, the fiber bundle returns due to its elasticity more or less to its original length (*down arrows*). De-elongation follows a different line as elongation. The original length is not reached but a small residual elongation remains. The residual elongation and the area between the two lines represent the viscous element of the system. An ideal elastic material without a viscous element would have no residual elongation

3.4 Viscoelastic Properties of Connective Tissue

All soft tissues and especially the connective tissues need the ability to change shape with movement and to return to their original shape when deforming forces cease. This is elasticity. *An ideal elastic material* like a steel spring deforms immediately if a deforming force acts and returns immediately to the original form if the deforming force is withdrawn: This is instantaneous elasticity. In biology such a material does not exist.

Elastic properties for connective tissues are provided by a combination of collagen fibers, elastic fibers, and ground substance. Collagen fiber bundles provide tensile strength, but have different mechanical properties depending on how much they are stretched. At rest, they are in a relaxed state with a wavy course. If longitudinal pull is applied, the undulations disappear and the fiber bundles become straight. This phase corresponds to the "toe-in" region of a stress–strain curve (Fig. 3.2). In this phase, fiber bundles lengthen with the application of a minimal force. Once the fiber bundles are completely straight, further lengthening requires movement of the ground substance between individual fibers and much more force is necessary for lengthening. This corresponds to the steeply raising "linear" region of the stress–strain curve (Fig. 3.2). If the stress is removed, elastic fibers recoil, reestablishing the length and the wavy course of the collagen fiber bundles.

An ideal elastic material will return to the original length along exactly the same line as the elongation. This is not so with living material. The return to the original state follows another line. The area between the lines is proportional to the viscous component. The original length is not immediately reestablished. There remains a residual elongation. This is also proportional to the viscous component. This combination of an elastic property with a viscous element is *viscoelasticity* (Fig. 3.2).

Another important aspect of viscoelasticity in biologic systems is *mechanical recovery time*. If a

stress–strain experiment is performed and then immediately repeated, the stress–strain curve is less stiff and less force is necessary to achieve the same strain. This is due to the fact that the individual fibers remain temporarily rearranged after the first experiment. If the experiment is repeated after enough time for the fibers to fully recover, the stress–strain curve follows again the original pattern. This period of time is called the mechanical recovery time.

3.5 Experimental Studies of Palmar Fascia Mechanics

3.5.1 Studies Comparing Viscoelastic Properties of Fiber Bundles of Flexor Tendons and Fiber Bundles of the Normal Palmar Aponeurosis

In a stress–strain experiment, a significant difference between normal flexor tendons and normal palmar aponeurosis can be detected. The stress–strain curves of flexor tendons are much stiffer: much more organized to transmit force. The rise of the straight segment of the fiber bundles of normal palmar aponeurosis is significantly flatter: much less force is required for elongation. The recoiling effect is the same. This means that the fiber bundles of the palmar aponeurosis are more extensible with the same ability to return to the original length: They are more "elastic" (Fig. 3.3).

What is the functional significance of this "elasticity"? The fiber bundles *store energy*. An example is the plantar aponeurosis which forms the base line of a triangle with the middle foot and the calcaneus. Bearing weight widens the angle between the middle foot and the calcaneus. The elastic stretch of the plantar aponeurosis stores this mechanical energy, which is released later as it assists lifting the foot (Fig. 3.4).

In a similar way the energy storing effect of the palmar aponeurosis supports the hand during different gripping activities.

The energy storing effect is also important for the pressure absorbing function of the superficial fascia of the palm. The loose fiber network of this system absorbs the pressure by deformity and return to the original shape. Since the pressure is not high and distributed to a wide surface, a loose network with a few collagen fibers is sufficient.

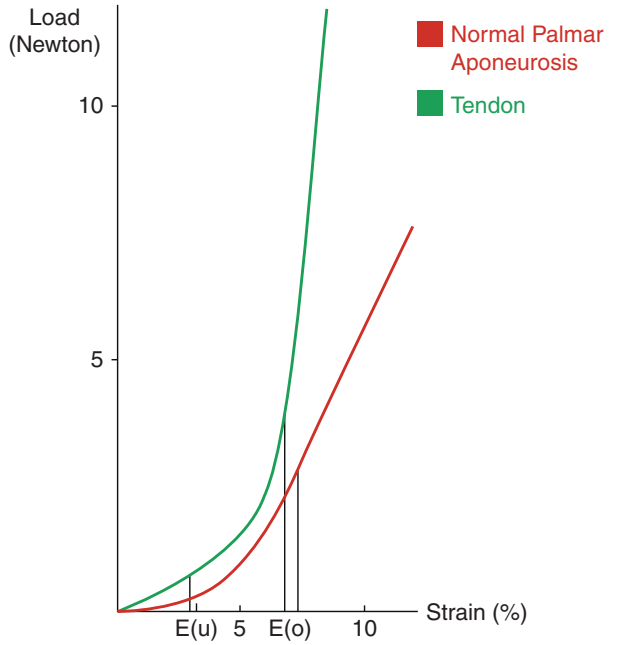


Fig. 3.3 Stress–strain test of flexor tendon and of palmar aponeurosis. Comparing the stress–strain test of fiber bundles of flexor tendon (*green*) and fiber bundles of palmar aponeurosis (*red*). The stress–strain curve of fiber bundles of flexor tendons is much stiffer, better at resisting force and transmitting stress. The curve of the fiber bundles from palmar aponeurosis is significantly less stiff. They can be elongated with much less force. They are more "elastic"

3.5.2 Stress–Strain Experiments with Different Stages of DD as Compared to Palmar Aponeurosis of Normal Individuals

We performed stress–strain tests with contracture bands and fiber bundles of palmar aponeurosis of individuals without DD. The tested tissue was elongated by 10% and the residual strain was measured. In normal palmar aponeurosis, the residual strain was well under 1 per mill. In contracture bands it was an average of 30 mm/m.

We then repeated the experiment with a lesser degree of elongation: 5% and 2.5%. The residual strain was less than with 10% elongation but the difference between contracture bands and normal palmar aponeurosis was still highly significant.

We tested also abnormally thickened fiber bundles of palmar aponeuroses without nodule formation and without contracture. We observed a significantly

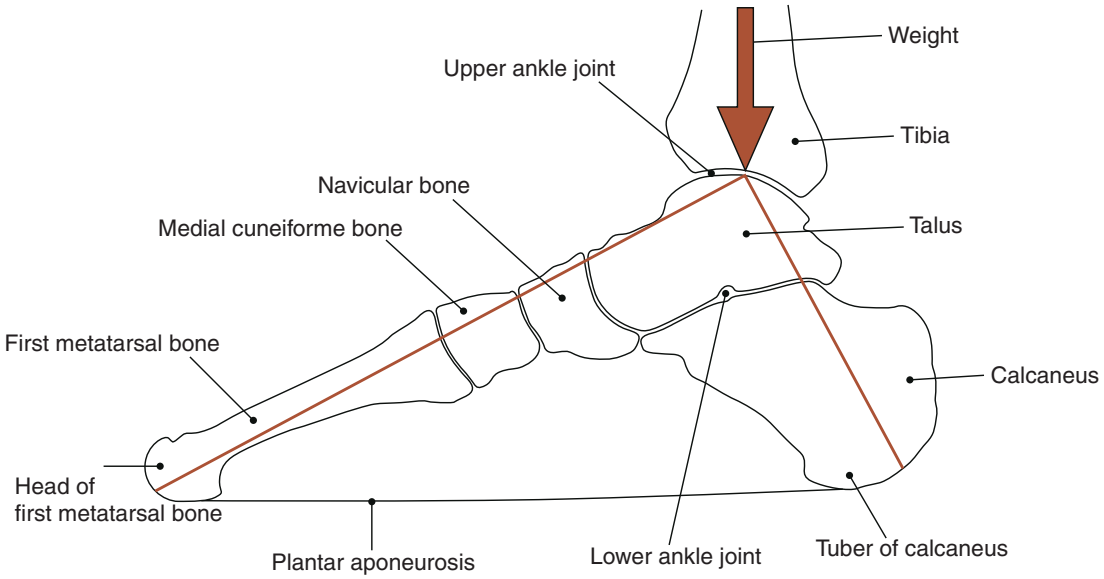


Fig. 3.4 Energy storing function of the plantar aponeurosis. The plantar aponeurosis forms the base line of a triangle with the middle foot and the calcaneus. Bearing weight widens the angle between the middle foot and the calcaneus. The elastic stretch of the plantar aponeurosis stores this mechanical energy, which later assists in lifting the foot

increased residual strain. Finally, we tested specimens from palmar aponeuroses from patients suffering from DD, which had normal appearance by macroscopic and microscopic view. After 10% elongation these specimens showed a significantly greater residual strain between 2 and 2.5 mm/m. The difference to normal palmar aponeurosis was not significant after 2.5% and 5% elongation (Table 3.1).

These results suggest that, in patients with DD, changes of the viscoelastic properties occur before other changes, such as nodules, with cell proliferation appear.

3.5.3 Recovery Time in Different Stages of DD as Compared to Normal Palmar Aponeurosis

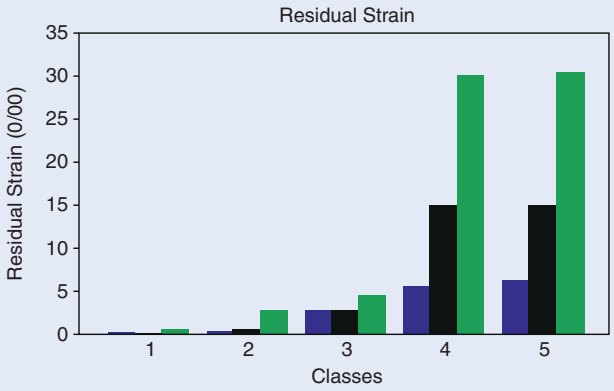
The mechanical recovery time for normal palmar aponeurosis for persons with or without DD on average was 10 min. It was not increased in specimens with fiber thickening only. The recovery time was longer in contracture bands but it was enormously increased in tissues of active contractures. It extended up to 3 h as compared to normal values of 10 min (Table 3.2).

3.6 Discussion

I hold the view that the cellular proliferation is preceded by a stage of fibrosis with thickening of the preexisting collagen fibers. The trigger might be the fact that fibers exposed to longitudinal stress do not relax completely after elongation and lose their wavy (undulated) course. This may be a stimulus for collagen production. In this case, the fault has to be attributed to the elastic fibers that fail to achieve complete recoilment (Millesi 1965). Longitudinal tension is certainly a factor stimulating collagen production. The thickening of the fibers changes the viscoelastic properties, causes atrophy of the loose peritendinous tissue and leads to fusion of neighboring thickening fibers to form major units. This has again an effect on the viscoelastic properties. At the same time the elastic fibers between the individual collagen fibers are changed as far as distribution and morphology are concerned. They are not anymore tiny, equally distributed fibers but show fragmentation and thickening. Frequently they are collected en masse in certain spots.

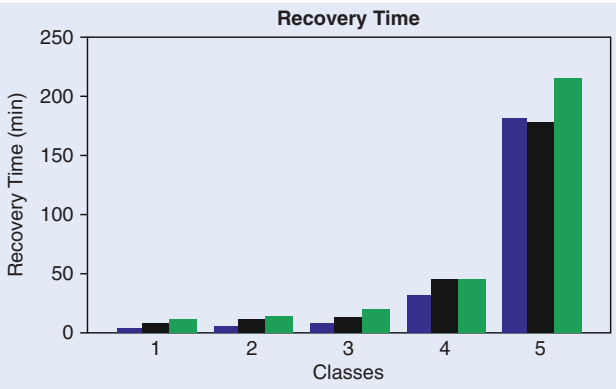
At the beginning of the disease, we see a disintegration of the sensible equilibrium that is the basis of the whole system of the energy storing devices. This consideration explains why the specially designed system

Table 3.1 Results of stress–strain test of different tissues and after different levels of elongation



Tested tissues:
1. Palmar aponeurosis of patients without DD
2. Apparently normal segments of palmar aponeurosis of patients with DD
3. Thickened fiber bundles from Palmar aponeurosis of patients with DD before formation of cellular nodules
4. Contracture band with cell proliferation
5. Residual contracture bands
The stress–strain tests were performed using elongations of
2.5%: black column
5%: white column
10%: green column
Stress–strain tests of fiber bundles of palmar aponeurosis of patients without DD showed a minimal residual elongation only at all three levels of elongations
Apparently normal palmar aponeurosis of patients with DD showed a minimal residual elongation only at 2.5% and 5% elongation. After 10% elongation however, the residual elongation of palmar aponeurosis with normal appearance of patients with DD revealed a significantly elevated residual elongation
The residual elongation was increased after elongation of all three levels in thickened fiber bundles without cell proliferation (Group 3)
It was very much increased at all three levels in groups 4 and 5

Table 3.2 Mechanical recovery



If after a stress–strain experiment with elongation and return to the original strain the experiment is immediately repeated the stress–strain curve is less stiff and less force is necessary to achieve the same strain. This is due to the fact that the individual fibers remain arranged according to the stress of the first experiment and do not need to be arranged again. If the experiment is repeated after some time and the original, less organized state has been reestablished; the stress–strain curve follows again the original pattern
The mechanical recovery time was determined for the same groups of tissue as in Table 3.1 (1–5) after elongation of 2.5% (black column), 5% (white column), and 10% (green column). The recovery time did not differ in Group 1, 2, and 3. It was somewhat elevated in group 4 (bands with cellular proliferation) but enormously elevated at all three levels in group 5 (contracture bands of the residual stage)

of the superficial fascia at the palmar side of the hand and fingers with the palmar aponeurosis as an essential part falls ill with DD and the flexor tendons – only a few mm apart and also exposed to trauma and stress – never develop a similar disease.

3.7 Conclusion

The results of these studies are the basis of the following conclusions:

DD is a systemic disease of the superficial fascia of the palmar aspect of the hand and fingers and the plantar side of the foot and toes. A function of these tissues is pressure absorption and energy storage. They have more pronounced elastic properties than other similar tissues. The pathology starts with changes of the viscoelastic properties. Wrong distribution and reduced efficiency of the elastic fibers may play a role in the initial stages of the disease. At this level hereditary factors are involved. The natural course and the treatment are not the subject of this chapter.

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