The palmar fibromatosis or the loss of flexibility of the palmar finger tissue. A new insight into the disease process of Dupuytren's contracture based on clinical and anatomical findings

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Today it is generally accepted that Dupuytren's disease starts as a nodule somewhere within the palmar soft tissue of the hand – the fibro-fatty-layer between the skin on one side and the aponeurosis and the fibrous tendon sheaths , respectively, on the other side. Nevertheless, the traditional view of the disease process is still using the picture of a contracion of pre-existing fibrous bands. Yet the key to a consistent pathogenesis can only be found if the structural and functional mix of tissue is taken into account that the fibromatosis creates in the subcutaneous tissue. Already more than 50 years ago Vernon Luck had focused on the interaction between the proliferating nodule and the fibrous bands, and he developed a concept of the pathogenesis that in its fundamental view is still valid today. The concept presented in this paper is based on Luck's concept.

The basic biomechanical processes in the living hand are instrumental in understanding the disease process. The soft palmar tissue above the aponeurosis and the fibrous tendon sheaths consists of skin anchoring fibers, enclosed fat and skin. When fingers are moved this area is subject to very significant changes in length. Stationary tissue areas only exist in the mid palm and in the fingertips. The interposed soft tissue above the finger skeleton is compressed and folded up when the fingers are flexed and unfolded when the fingers are extended. This requires a complex structure of the skin anchoring fibers to allow for tension free tissue movements. When being used and in their relaxed position fingers are more or less flexed and thus the compressed, folded tissue is the dominant tissue formation. The proliferating nodule of fibromatosis now occupies the subcutaneous tissue predominantly in its flexion formation, an effect that so far has not sufficiently been taken into account. So there is no need for implicating a contracting tissue process. The fibromatosis occupies the shortened, compressed tissue. The fat is being displaced and a conglomerate develops that consists of proliferating tissue, enclosed skin anchoring fibers in their flexion position, and attached structures of deeper tissue, like the aponeurosis or the vertical septa. When the fingers are extended this complex becomes subject to tension which has an effect on morphogenesis and

histogenesis of the Dupuytren tissue. The fibromatosis "freezes" the flexion formation of the palmar tissue and the flexed fingers cannot be extended freely anymore, thus creating the well known extension deficit. The since 400 years postulated and never ultimately proven concept of contractio digitorum thus becomes redundant. The pulling tension acting on Dupuytren tissue can explain the transformation of fibroblasts into myofibroblasts, the tendiniform alignment of the tissue conglomerate and the reactive hypertrophy of the attached fiber structures. And the structural remodeling of the subcutaneous fibers can interpret the frequently observed displacement of the finger nerves. These typical findings are illustrated in detail using clinical and anatomical in situ pictures. And the therapeutic consequences of this contraction free concept are discussed and include a minimized resection at fasciectomy and a long-term use of static night splinting.