Fascial Anatomy in Manual Therapy: Introducing a New Biomechanical Model

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ABSTRACT

Background and Purpose: Fascial anatomy studies are influencing our understanding of musculoskeletal dysfunctions. However, evidenced-based models for manual therapists working with movement dysfunction and pain are still developing. This review presents a synthesis of one biomechanical model and discusses underlying hypotheses in reference to some current trends in musculoskeletal research. **Method:** The author conducted principally a search of the health sciences literature available on PubMed for the years 1995 to 2011, and consulted published texts concerning this model. **Findings:** Some of the hypotheses proposed by this model have been investigated via anatomical dissections that have addressed the connections between deep fascia and muscles, the histology of deep fascia, and its biomechanical characteristics. These dissections have led to new anatomical findings. This model may also present new challenges for research in fields such as peripheral motor control and proprioception. **Clinical Relevance:** This information could introduce new perspectives for clinicians involved in the manual treatment of musculoskeletal dysfunctions.

Key Words: deep fascia, fascial anatomy, manual therapy, myofascial unit

INTRODUCTION

One tissue gaining increasing attention in manual therapy is the connective tissue known as fascia. While there is still ongoing discussion about how to categorize and name the various fascial layers[1] it is, nevertheless, possible to distinguish 3 different types of human fasciae, namely, superficial, deep, and visceral fascia. Each of these has its own anatomical and biomechanical characteristics and specific relationships to surrounding structures. Most studies concerning fasciae focus on the anatomy and pathology of specific areas, such as the thoracolumbar fascia,[2] abdominal fascia,[3] the Achilles tendon enthesis organ,[4] plantar fascia,[5,6] and the iliotibial tract.[7] While detailed studies pertaining to specific areas of fascia are important, they do not provide a vision of the human fascial system as an interrelated, tensional network of connective tissue. A few authors consider its 3-dimensional (3D) continuity[8-10] but these holistic models do not always provide specific indications for treatment. A functional model for the entire human fascial system that correlates dysfunctional movement and pain is in its infancy with regards to evidence-based investigations and studies.

This paper will examine a 3D biomechanical model for the human fascial system that takes into account movement limitation, weakness, and pain distribution during the analysis of musculoskeletal dysfunctions. While the interaction between all fascial layers is contemplated within this model, this paper will focus on the part that addresses the deep fascia, which appears to be principally implicated in musculoskeletal activity.

The model is the result of 35 years of study and clinical practice by Luigi Stecco, an Italian physiotherapist.[11,12] Developed specifically for manual therapists working with movement dysfunction and pain, the chief focus of this model is the relationship between muscles, deep fascia, and its components (epimysium, perimysium, and endomysium). More recently, this work has been supported by a series of extensive anatomical dissections of unembalmed cadavers. Histological, biomechanical, and functional studies have also been undertaken to verify some of the underlying hypotheses concerning the architecture of the fascia, its innervation, its relationship with muscle fibers, and the possible mechanisms of action of the manual technique itself.

Deep Muscular Fascia

Studies of deep muscular fascia support its role in epimuscular myofascial force transmission[13,14] although the degree to which it is involved in in-vivo muscle movements is still not clear.[15] Deep fascia is implicated in deep venous return[16] and its possible role in proprioception has been suggested.[17] Deep fascia is a well-vascularized tissue often employed for plastic surgery flaps,[18] and it responds to mechanical traction induced by muscular activity in different regions.[19] It has an ectoskeletal role and can potentially store mechanical energy and distribute it in a uniform manner for harmonious movement. The mechanical properties of the fascial extracellular matrix itself can be altered by external mechanical stimuli that stimulate protein turnover and fibroblastic activity.[20,21] These characteristics and the reported abundant innervation of deep fascia indicate that it could have the capacity to perceive mechanosensitive signals.[22]

The correct embryonic development of the musculoskeletal system requires the coordinated morphogenesis of muscle, muscular fascia, tendon, and skeleton. In the embryo, muscle tissue and its fascia form as a differentiation of the paraxial mesoderm that divides into somites on either side of the neural tube and notochord. The cartilage and bone of the vertebral column and ribs develops from the ventral part of the somite, the sclerotome, whereas the dorsal part of the somite, the dermomyotome, gives rise to the overlying dermis of the back and to the skeletal muscles of the body and limbs.[23] It is now known that muscular connective tissue is critical for the form and function of the musculoskeletal system, muscle development, and muscle regeneration in general. For example, in mammals, fetal connective tissue fibroblasts express the transcription factor Tcf4, which is essential for proper muscle development. Studies indicate that Tcf4-expressing cells actually establish a pre-pattern in the limb mesoderm that determines the sites of myogenic differentiation, thereby shaping the basic pattern of vertebrate limb muscles.[24] Other studies demonstrate that the absence of specific transcription factors in muscle connective tissue disrupts muscle and tendon patterning in limbs, and that to understand the etiology of diseases affecting soft tissue formation a focus on connective tissue is required.[25]

As muscle cells differentiate within the mesoderm, each single muscle fiber is progressively surrounded by endomysium, groups of fibers by perimysium, whole muscles are enclosed by epimysium and deep fascia encloses groups of muscles. The connective tissue that accompanies the development of muscle fibers and nerve components facilitates the different innervations and functions of the muscle fibers within each muscle belly. Furthermore, the fascia unites all of the fibers of a single motor unit that are often distributed throughout a muscle in non-adjacent positions, allowing for synergy between recruited fibers and separation from nonrecruited fibers. Fascia can therefore adapt to variations in form and volume of each muscle according to muscular contraction and intramuscular modifications induced by joint movement.

This fascial-based organization allows each single muscle fiber to slide somewhat independently from its adjacent fibers. In addition, deep muscular fascia has significant characteristics that allow it to perceive muscle fiber tension. Many muscle fibers attach directly onto fascia,[26] and it also connects with muscle fibers via intermuscular septa, fascial compartments, and tendon sheaths. Histological studies of deep fascia in the limbs show that it consists of elastic fibers and undulated collagen fibers arranged in layers. Each collagen layer is aligned in a different direction and this permits a certain degree of stretch as well as a capacity to recoil.[27] Fascia can also be tensioned, as it connects with bone through periosteum.

Even though this strict relationship between muscle fibers and their surrounding fascia is characteristic of all muscles, the role of the fascia in musculoskeletal function has only received attention in the last decade. In fact, the number of studies about how muscles work is still significantly higher than studies investigating the possible functions of deep muscular fascia.

THE BIOMECHANICAL MODEL

In order to analyze the fascial system more effectively, Stecco[11(p 28)] divides the body into 14 functional segments: head, neck, thorax, lumbar, pelvis, scapula, humerus, elbow, carpus, digits, hip, knee, ankle, and foot (Figure 1). Each functional segment is comprised of a combination of portions of muscles, their fascia, and the joint components that move when these muscle fibers contract.

Myofascial Unit

Six myofascial units (MFU) are considered to govern the movement of the body segments on the 3 spatial planes. An MFU is described as a functional unit composed of motor units innervating monoarticular and biarticular muscle fibers, the joint that they move in one direction on one plane, the deep fascia that unites these fibers, and the nerve components involved in this movement. One example is the MFU for knee extension where fibers from medial and lateral vasti are the monoarticular components and fibers from the rectus femoris provide the biarticular component (Figure 2). Myofascial units are considered to be the functional building blocks of the myofascial system. In this model, it is postulated that deep fascia is a potentially active component in movement coordination and peripheral motor control and that, due to its innervation, the fascial component of each MFU is a possible source of directional afferents that could contribute to proprioceptive information.

Center of coordination

Within the deep muscular fascia of each MFU, a specific small area called the center of coordination (CC) is identified. A CC is defined as a focal point for vectorial forces produced by monoarticular and biarticular muscle fibers of an MFU acting on a body segment during a precise movement and are often situated within the deep fascia overlying a muscle belly. In reference to the MFU for knee extension mentioned previously, the CC is located between the vastus lateralis and rectus femoris, halfway on the thigh (see Figure 2).

Through clinical observation and studies comparing acupuncture points, myofascial trigger points, and the sum of the vectorial forces involved in the execution of each segmental movement, Stecco[12 (pp 325-326)] noted that impeded gliding of the deep fascia commonly occurs at these intersecting points of tension. The term center of coordination is used to infer the possible involvement of deep fascia in monitoring movement of a related segment via its connections to muscle spindles, Golgi tendon organs, and other mechanoreceptors.

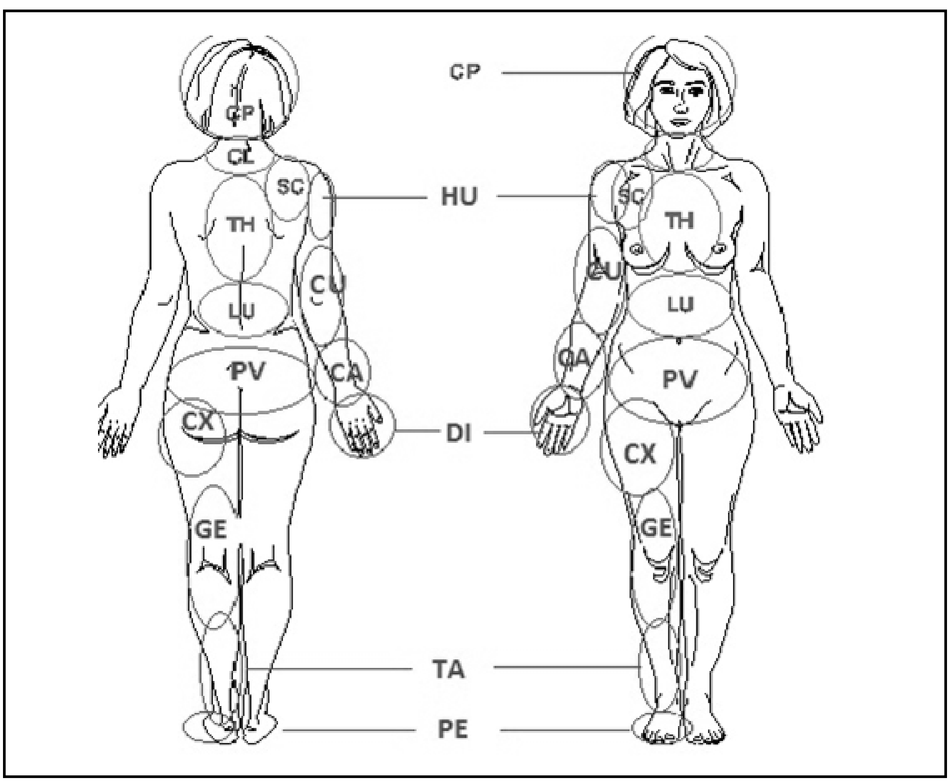

Figure 1. Fourteen body segments. CP: Caput, CL: Collum, TH: Thorax, LU: Lumbar, PV: Pelvis, SC:Scapula, HU: Humerus, CU: Cubitus, CA: Carpus, DI :Digits, CX: Coxa, GE: Genu, TA: Tarsus, PE: pes. Each segment comprises joint(s), portions of muscles that move the joint(s), the fascia surrounding these muscle fibers. Latin terms are used to distinguish these segments from simple joints.

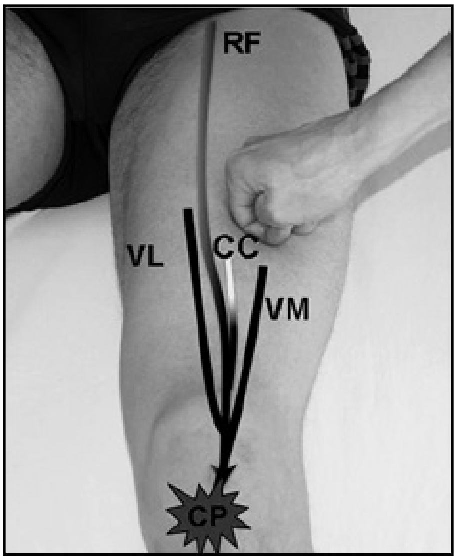

Figure 2. The MFU (Myofascial Unit) for knee extension comprises monoarticular components (vastus lateralis VL, medialis: VM, and intermedius), and biarticular components (rectus femoris: RL). The CC (center of coordination) for this MFU is situated midway on the thigh over the deep fascia between vastus lateralis and rectus femoris and the CP (center of perception) is located in the anterior knee joint.

Center of perception

For each MFU, a circumscribed area around the joint is described. This is where traction exerted during muscle fiber activity of this MFU is perceived on the joint capsule, tendons, and ligaments. This circumscribed area is called the center of perception (CP); and according to Stecco,[11 (p 23)] when any given MFU is malfunctioning, then pain is felt in its corresponding CP. For example, in the MFU for knee extension the CP is located in the anterior knee joint (see Figure 2).

Any impeded gliding between collagen fibers within the deep fascia of an MFU is thought to cause anomalous tension, resulting in firing of afferents from embedded mechanoreceptors within the fascial component of the MFU. Subsequently, disturbed motor unit recruitment could then produce incongruent joint movement, resulting in conflict, friction, inflammation of periarticular soft tissues, and sensations of pain or joint instability over time.

Fascial Mediation of Agonist-antagonist Interaction

This model also considers the interaction between agonist and antagonist MFUs that is important for myofascial force transmission and coordinated movement. In almost every MFU, a number of monoarticular fibers insert onto the intermuscular septum that separates two antagonist MFUs on the same plane. For example, in the MFU for elbow extension, the monoarticular fibers are situated in the lateral and medial heads of triceps and the anconeus muscle, and they collaborate with biarticular fibers from the long head of triceps to move the elbow joint into extension. The monoarticular components stabilize the joint during movement while the biarticular components synchronize movement between adjacent joints. In other words, the short vectors, created by the monoarticular fibers, and the long vectors from the biarticular fibers allow for precision and stability of each segment during movement. The MFU for elbow extension has its own antagonist myofascial unit that coordinates elbow flexion. When the elbow extends, the monoarticular fibers from the lateral and medial heads of triceps contract and the intermuscular septum where they insert will be stretched. The brachialis muscle inserts on the other side of this same septum. It is an elbow flexor and the monoarticular component of the MFU for elbow flexion. This connection means that during elbow extension brachialis is stretched a little too, causing its stretch receptors to fire. Thus, the deep fascia can be envisioned as a component in agonist and antagonist activity.

Myofascial Sequences

Biarticular muscle fibers (part of each MFU) link unidirectional MFUs positioned in a specific direction to form myofascial sequences.[11 (p 98)] This type of organization is said to guarantee the synchronization of single MFUs in order to develop forceful movements and to monitor upright posture in the 3 spatial planes.

A single myofascial sequence coordinates movement of several segments in one direction on one plane. Sequences on the same spatial plane (sagittal, frontal, or horizontal) can be considered as reciprocal antagonists. This means that areas of altered fascia can potentially produce recognizable patterns of extended tension that can develop along the same sequence, or be distributed on the same plane between antagonist sequences (Figure 3). This is thought to be possible because a part of the deep fascia slides freely over the muscle fibers, thereby transmitting tension along the length of the limb or trunk, yet another part is tensioned directly by muscle fibers that insert onto it and indirectly by its insertions onto bone. The combination of the biarticular muscle fibers found in each MFU and so-called myotendinous expansions (see Discussion section) forms the anatomical substratum of the myofascial sequences.

Myofascial Spirals and Centers of Fusion

Stecco also identifies small areas located principally over the retinacula that might monitor movements in intermediate directions between two planes, as well as movements of adjacent segments in different directions.[12 (p 208)] These small areas are called centers of fusion (CF) and combinations of these CF form myofascial spirals.

It is important to note here that studies have shown that retinacula are reinforced areas of the deep fascia itself, rather than separate bands as commonly illustrated in topographical anatomy texts.[28,29] Retinacula actually continue from one joint to the next via oblique collagen fibers within the deep fascia, creating macroscopically visible spiral formations. Stecco postulates[12 (p 213)] that during complex movements, such as walking or running, these spiral-form collagen fibers would progressively wind and unwind, and the ensuing tensioning of the retinacula could progressively activate, inactivate, and synchronize mechanore-

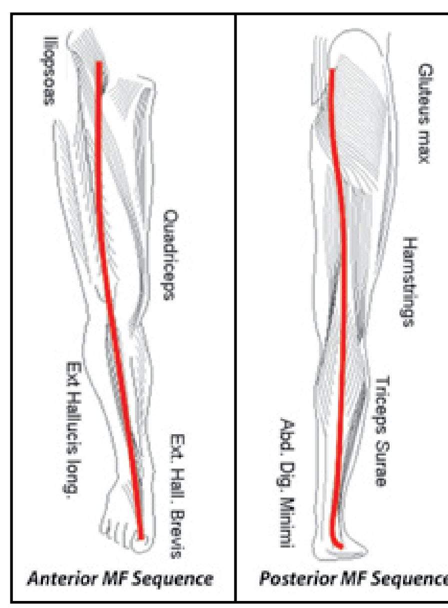

Figure 3. Myofascial sequences on the sagittal plane (anterior, posterior) in the lower limb. The fibers of the indicated biarticular muscles connect adjacent segments.

ceptors located within these periarticular structures.

MANUAL METHOD BASED ON THIS MODEL

A manual approach for treating the human fascial system, called the Fascial Manipulation© method, is based on the model described above. Once the initial obstacle of the new terminology is overcome, and the main principles are understood, clinicians apply this biomechanical model to interpret the spread of tensional compensations from one segment to another, and to trace back to initial disturbances. A fundamental concept for clinicians is the indication to go beyond treating the site of pain (CP) and to trace back to its fascial origin in corresponding key areas (CC and/or CF). As treatment is usually at a distance from the site of pain, or the inflamed area, this technique can be applied during the acute phase of a dysfunction.

A systematic evaluative process of movement using codified movement and palpatory tests guides therapists in selecting the combination of fascial alterations to be treated. Changes in range of movement, pain, and/or muscle recruitment are verified after treatment of each point.[30] In other words, therapists identify which CC and/or CF are involved in any given dysfunction of one or more MFUs. This method is applied in a wide variety of musculoskeletal dysfunctions, and treatment of segmental or multisegmental problems is approached through the analysis of chronological events involved in each individual case.

The manual technique itself is directed towards the deep muscular fascia. Therapists use their elbow, knuckle, or fingertips over the CC and/or CF, creating localized hyperemia through deep friction. Deep friction can apparently alter the ground substance of the deep fascia via mechanotransduction mechanisms[31] and this could restore gliding between collagen fibers. According to the Stecco model, it is important to apply friction precisely over the small areas where tension produced by muscle fiber contraction apparently converges.[32]

DISCUSSION

This biomechanical model shifts emphasis from muscles with origins and tendinous insertions moving bones, to motor units activating groups of muscle fibers united by fascia that bring about movement. Interpreting movement in terms of MFUs introduces a new paradigm to the current understanding of musculoskeletal function.

It does find some resonance in studies that examine motor unit activity, which are providing new understandings of movement[33] and muscle fatigue.[34] Motor unit activity determines movement and different movements require varying degrees of contractile force. This force depends on the number of motor units recruited, muscle fiber types, and motor neuron firing rates.[35 (p 20)] While humans appear to have an infinite number of combinations of motor-unit recruitment and discharge rates that can be used to vary muscle force, control strategies have reduced these options substantially. These strategies include definite patterns in the recruitment order of motor units and the use of discharge rate to grade muscle force, although motor-unit properties can apparently adapt within limited ranges when challenged. Motor unit recruitment is related to the mechanical function of the muscles, although many factors such as mechanics, sensory feedback, and central control can influence recruitment patterns.[36]

The possible relationship between alterations in fascia, pain, and motor unit recruitment clearly warrants further studies. Findings from studies of pain and motor unit recruitment do suggest that pain induces reorganization in motor unit recruitment. One study showed how injections of a saline solution into the infrapatellar pad caused anterior knee pain that reduced the coordination of motor units between the medial and lateral vasti muscles as compared to subjects without knee pain.[37] In another study, the authors indicate how pain induces a reorganization of motor unit recruitment strategy, involving changes in recruitment order and changes in the population of units recruited, favoring those with a slightly different force direction.[38] Furthermore, injections of inflammatory agents (Freund Adjuvans solution) into rat lumbar muscles have evidenced an increase in the proportion of dorsal horn neurons with input from the posterior lumbar fascia, demonstrating a correlation between deep muscles and areas of deep fascia at a distance.[39 (p 251)]

Stecco's hypothesis of deep fascia's role in proprioception and motor coordination[12 (p 15,16)] definitely pivots on demonstrating the afferent innervation of deep fascia. Different studies do suggest that fascia is richly innervated. The presence of abundant free and encapsulated nerve endings have been described in various regions such as the thoracolumbar fascia,[40] the brachial fascia,[41] fascia lata, crural fascia, and various retinacula.[42] While some of the nerve fibers found in fascia are probably involved in local blood flow control due to their adrenergic nature,[43] others do appear to be proprioceptors. Encapsulated mechanoreceptors and proprioceptors such as Pacini and Ruffini corpuscles and Golgi tendon organs are embedded in deep muscular fascia, with their connective tissue capsules in direct continuity with endomysium and perimysium.[44] This means that whenever a muscle fiber contracts, it inevitably stretches the fascia enclosing it and this may stimulate nearby embedded receptors.

Interestingly, as mentioned before, the histological studies have shown that collagen fiber distribution within deep fascia is well organized and not irregular, as generally reported, and it does correspond to precise motor directions. More specifically, in the limbs, two to 3 layers of parallel collagen fiber bundles form the deep fascia and adjacent layers are oriented in different directions.[45] The angle between the fibers of adjacent layers of the crural fascia has been measured and was found to be approximately 78°.[46] Loose connective tissue separates each layer permitting the collagen fiber layers to slide and to respond to tension (Figure 4). The deep fascia of the trunk has quite a different histological structure, as compared to limb fascia, as it is formed of a single layer of undulated collagen fibers adhering to the underlying muscles.[47] One study of the pectoral fascia indicates how tensioning of a particular area of this fascia

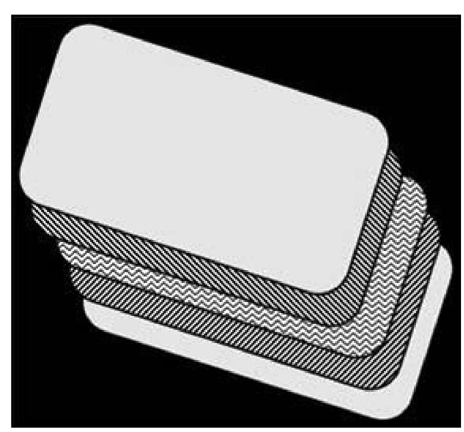

Figure 4. Layers of collagen fibers within deep fascia have different orientations. Note: Mechanoreceptors are embedded within these layers.

could activate specific patterns of proprioceptors, potentially providing directional and spatial afferent information.[48]

While the Stecco model focuses on the role that deep fascia could play in peripheral motor control, collaboration and integration with the central nervous system is duly recognized.[11 (p 164)] Nevertheless, the interrelationship that exists between muscle fiber contractions, mechanoreceptors embedded in deep muscular fascia and peripheral motor control is a rather controversial aspect of this model. Muscle spindles lie in parallel to muscle fibers and they do have a thin connective tissue capsule that is continuous with either the endomysium or the perimysium of the surrounding muscle fibers. Stecco proposes[12 (p 20)] that when gamma fiber stimulation causes intrafusal spindle fibers to contract a minimal stretch could be propagated throughout the entire fascial continuum, including tensioning the deep fascia at the CC. If this fascial continuum is elastic, then it could adapt to this stretch permitting muscle spindles to contract normally with subsequent correct activation of alpha motor fibers and muscular contraction. On the other hand, if there is excessive stiffness within the system, then particular small areas on the deep fascia (the CC/CF) will not be elastic and muscle spindle contraction could be less than perfect, distorting afferent information to the central nervous system and thereby interfering with correct motor unit activation (Figure 5). Incongruent motor unit activation could then result in uncoordinated movement, producing joint instability or pain.[49]

Studies addressing sensory processing do point to the muscle spindles as prime players in position and movement sense.[50] There is evidence that muscle spindles contribute to both the sense of limb position and limb movement, and that there is continuous interaction between the contraction of limb muscles and centrally generated motor command signals; however, the role of the fascia in this interplay does require further studies.

The Stecco model also suggests that if the fascia is in a physiologic state, sliding and tending appropriately, it could contribute to simultaneous adaptation between agonist and antagonist according to the inclination of the muscle fibers and the segment involved. Studies of myofascial force transmission mechanisms[51,52] do suggest some evidence for this hypothesis of deep fascia's role in agonist and antagonist interaction but this is another area requiring further investigation.

As part of the fascial anatomy studies carried out on unembalmed human cadavers, numerous myotendinous expansions linking adjacent body segments have been identified.[53] These myotendinous expansions are well documented in anatomical texts, yet no clear functional significance has ever been assigned to these structures. Some authors have suggested these expansions have a role in stabilizing tendons,[54] and the term tensegrity has been used to describe this type of connection existing between body segments.[55] These expansions extend well beyond any bony insertion of the muscle, forming a continuum with the deep fascia in adjacent segments. For example, in the upper limb, the lacertus fibrosus of biceps brachialis can be considered as a myotendinous expansion, yet pectoralis major, palmaris longus, latissimus dorsi, deltoid, triceps brachialis, and extensor carpi ulnaris all present myotendinous expansions of their deep fascia. A study of the functional relationship between shoulder stabilizers and hand-grip suggests that, in agreement with the Stecco model, this myofascial organization could be a means for transmission of tension along a myofascial sequence, permitting the coordination between stabilization of a proximal joint or joints while distal joints are involved in forceful movement.[56]

Fascial anatomy studies have also added to the growing consensus among anatomists that retinacula, in particular the ankle retinacula, may play an important role in proprioception and should not be considered merely as passive elements of stabilization, but a type of specialization of the fasciae for movement perception.[57] Ankle retinacula are thickenings of the deep fascia formed by 2 to 3 layers of parallel collagen fiber bundles, densely packed with a little loose connective tissue, and they present virtually no elastic fibers but many nerve fibers and corpuscles. In fact, the histological features of retinacula appear to be more suggestive of a perceptive function, whereas tendons and ligaments mainly play a mechanical role. Dissections have shown that the retinacula have specific muscular and bone connections that allow them to be sensitive to the tonus of the muscles. Given their continuity with deep fascia, and the fact that tendons typically pass beneath retinacula, any impediment in gliding of the retinacula would interfere with correct functioning of the tendons themselves. This could potentially lead to problems such as tenosynovitis, or dysfunction of the associated muscles, as well as altering the function of adjacent segments via disturbed proprioceptive afferents.

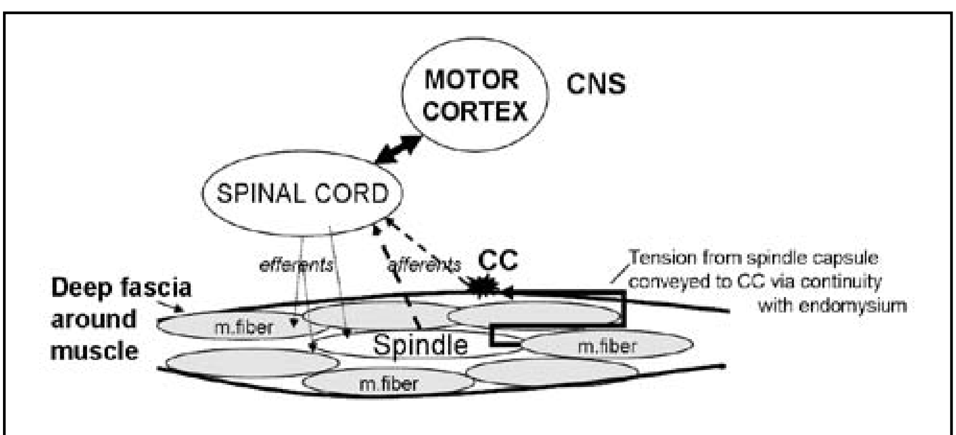

Figure 5. Schematic diagram illustrating possible mechanism of interaction between spindles, fascia, and CNS as suggested by Stecco.

CONCLUSION

The architecture of deep muscular fascia and its precise relationship to the muscles it surrounds forms the basis of an innovative biomechanical model for the human myofascial system. It suggests that deep muscular fascia could act as a coordinating component for motor units grouped together into functional units and that this connective tissue layer unites these functional units to form myofascial sequences. This holistic vision of the human fascial system is partially supported by ongoing evidence-based research into fascial anatomy. Clinically it is common to find patients with regional pain syndromes and some of the aspects presented in this biomechanical model could provide indications for comprehending the possible connection between different areas of pain. The Stecco model does employ an unusual terminology and numerous new abbreviations that can present an initial obstacle to comprehension. Nonetheless, this model introduces interesting perspectives for clinicians involved in the manual treatment of musculoskeletal dysfunctions but further well-conducted clinical studies to test its validity are necessary.

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REVIEW

The fascia of the limbs and back – a review

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Abstract

Although fasciae have long interested clinicians in a multitude of different clinical and paramedical disciplines, there have been few attempts to unite the ensuing diverse literature into a single review. The current article gives an anatomical perspective that extends from the gross to the molecular level. For expediency, it deals only with fascia in the limbs and back. Particular focus is directed towards deep fascia and thus consideration is given to structures such as the fascia lata, thoracolumbar fascia, plantar and palmar fascia, along with regional specializations of deep fascia such as retinacula and fibrous pulleys. However, equal emphasis is placed on general aspects of fascial structure and function, including its innervation and cellular composition. Among the many functions of fascia considered in detail are its ectoskeletal role (as a soft tissue skeleton for muscle attachments), its importance for creating osteofascial compartments for muscles, encouraging venous return in the lower limb, dissipating stress concentration at entheses and acting as a protective sheet for underlying structures. Emphasis is placed on recognizing the continuity of fascia between regions and appreciating its key role in coordinating muscular activity and acting as a body-wide proprioceptive organ. Such considerations far outweigh the significance of viewing fascia in a regional context alone.
Key words deep fascia; ectoskeleton; iliotibial tract; osteofascial compartments; palmar fascia; plantar fascia; superficial fascia; thoracolumbar fascia.

Introduction

'Fascia' is a vague term that is derived from the Latin for a band or bandage. It has long been used by gross anatomists to embrace a spectrum of undifferentiated mesenchymal tissues that wrap around what are sometimes regarded as being the more 'specialized' organs and tissues of the body, or form a packing material between them. The inherent implication of this traditional view is that fasciae are inconsequential residues that are less important than the tissues with which they are associated. Increasingly, the errors of this assumption are being exposed and undoubtedly fascia is of considerable importance to many surgeons, physiotherapists, orthotists, osteopaths, massage therapists and other professionals working in health-related disciplines. They are involved in the spread or containment of infections and/or pus, oedematous effusions and a multitude of other conditions, including compartment syndromes, fibromyalgia, Dupuytren's contracture and plantar fasciitis or fasciosis.

Encouragingly, there has been a strong resurgence of interest into both basic and applied research in fasciae in recent times – as evidenced by the first international fascia research congress in Boston, USA (Findley & Schleip, 2007a) and the publication of a book by Lindsay (2008) that is aimed at health-care professionals and students. This has been an important motivation for tackling the current review, together with the observation that there has been no recent broad anatomical overview of fascia in any journal, which could help to stimulate further research. Relatively few scientists are interested primarily in fascia – many who publish on it have a different principal focus and fascia is often just part of a grander portfolio. However, the clinical significance of fascia could tempt others to enter the field if contemporary issues are addressed in a multidisciplinary fashion with the tools that modern science affords. Fasciae probably hold many of the keys for understanding muscle action and musculoskeletal pain, and maybe of pivotal importance in understanding the basis of acupuncture and a wide range of alternative therapies (Langevin et al. 2001, 2002, 2006a; Langevin & Yandow, 2002; Iatridis et al. 2003). Intriguingly, Langevin et al. (2007) have shown that subtle differences in the way that acupuncture needles are manipulated can change how the cells in fascia respond. The continuum of connective tissue throughout the body, the mechanical role of fascia and the ability of fibroblasts to communicate with each other via gap junctions, mean that fascia is likely to serve as a body-wide mechanosensitive signaling system with an integrating function analogous to that of the nervous system (Langevin et al. 2004; Langevin, 2006). It is indeed a key component of a tensegrity system that operates at various levels throughout the body and which has been

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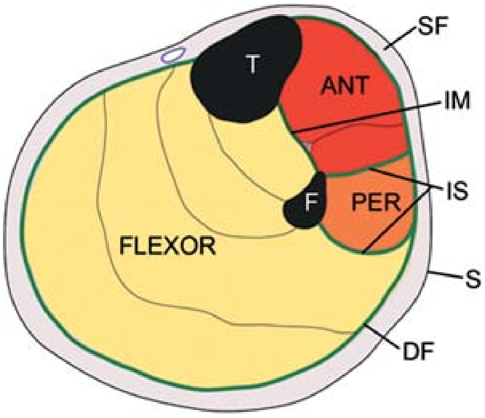

Fig. 1 A diagrammatic representation of a transverse section through the upper part of the leg showing the relative positions of the superficial (SF) and deep fascia (DF) in relation to the skin (S) and muscles. Note how the deep fascia, in association with the bones [tibia (T) and fibula (F)] and intermuscular septa (IS) forms a series of osteofascial compartments housing the extensor, peroneal (PER) and flexor muscles. If pressure builds up within a compartment because of an acute or overuse injury, then the vascular supply to the muscles within it can be compromised and ischaemia results. ANT, anterior compartment; IM, interosseous membrane.

considered in detail by Lindsay (2008) in the context of fascia.

Anatomists have long distinguished between superficial and deep fascia (Fig. 1), although to many surgeons, 'fascia' is simply 'deep fascia'. The superficial fascia is traditionally regarded as a layer of areolar connective or adipose tissue immediately beneath the skin, whereas deep fascia is a tougher, dense connective tissue continuous with it. Deep fascia is commonly arranged as sheets and typically forms a stocking around the muscles and tendons beneath it. However, regions of dense connective can also be found within the superficial fascia – e.g. the hollow canals of fibrous tissue enclosing the saphenous veins (Shah & Srivastava, 1966; Papadopoulos et al. 1981). According to Caggiati (1999), these saphenous canals reduce the risk of varicosities. They point out that varicosities more commonly affect the tributaries of the (great) saphenous veins, which lie outside the saphenous canal (Caggiati, 2000). There are also fascial canals in the fingers that are most familiar to hand surgeons and which are formed from Grayson's and Cleland's ligaments (Doyle, 2003). They form a protective tunnel for the digital vessels and nerves, preventing them from bowstringing when the finger is flexed.

All fasciae are forms of soft connective tissue, i.e. the 'connective tissue proper' of standard histology texts (Bloom & Fawcett, 1975). Fasciae are continuous with each other throughout the limbs – a principal long recognized by anatomists. Gerrish (1899), for example, implores the students of his day to note the continuity of fibrous membranes, i.e. how tendons, ligaments and fasciae blend with periosteum, how both tendons and fasciae can function as ligaments and how tendons can become fascial expansions. More recently, Gerlach & Lierse (1990) have emphasized the unifying role of connective tissue in the limbs and refer to an integrating 'bone–fascia–tendon system'. Thus, in one sense it is fitting that modern authors advocate a very broad definition of fascia that embraces all forms of soft connective tissue (Findley & Schleip, 2007b). On the other hand, it is confusing to regard more specialized structures such as tendons and ligaments as 'fascia' and thus similar to, for example, the areolar connective tissue beneath the skin. Furthermore, it is impractical in an article such as the current one to consider 'fascia' synonymous with 'connective tissue'. Thus, as tendons have been the subject of a recent review by the author (Benjamin et al. 2008), limited coverage will be given to this area in particular, or to the related topic of ligaments. In a further effort to keep the review within manageable bounds and because of the particular interests and expertise of the author, only fasciae in the limbs and back are considered and the primary focus is on deep fascia. It is recognized, however, that fasciae are also prominent and important elsewhere – particularly in the head, neck and pelvis. An attempt has been made to balance general principles of fascial biology with matters of specific regional interest that pertain largely to certain named fasciae alone.

General principles of fascial biology

The anatomical and histological forms of fascia

The presence of a significant layer of fat in the superficial fascia is a distinctive human trait (the *panniculus adiposus*), compensating for the paucity of body hair. It thus plays an important role in heat insulation. In hairy mammals, the same fascia is typically an areolar tissue that allows the skin to be readily stripped from the underlying tissues (Le Gros Clark, 1945). Where fat is prominent in the superficial fascia (as in man), it may be organized into distinctive layers, or laminae (Johnston & Whillis, 1950), although Gardner et al. (1960) caution that these may sometimes be a characteristic of embalmed cadavers and not evident in the living person. Furthermore, Le Gros Clark (1945) also argues that fascial planes can be artefactually created by dissection. Conversely, however, some layers of deep fascia are more easily defined in fresh than in fixed cadavers (Lytle, 1979).

The superficial fascia conveys blood vessels and nerves to and from the skin and often promotes movement between the integument and underlying structures. This is particularly evident over highly mobile joints and on the dorsum of the hand, where the skin has considerable freedom of movement so that it can slide easily over the extensor tendons during finger movements. Skin mobility protects both the integument and the structures deep to it from physical damage. Mobility is promoted by multiple

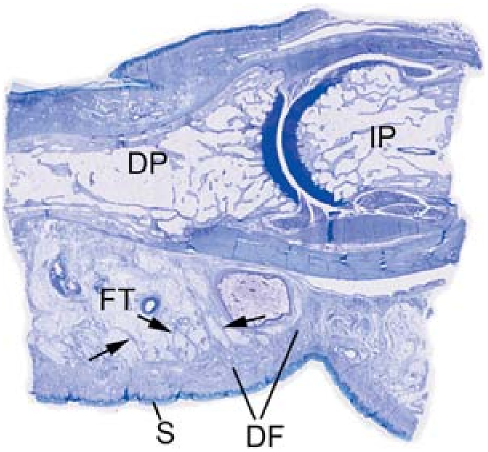

Fig. 2 A low power view of a sagittal section through a finger in the region of the distal interphalangeal joint. Note how the skin (S) on the palmar side is intimately associated with a thick region of dense fascia (DF) that anchors it in position and stops it sliding in the interests of a firm grip. At a deeper level, the bundles of fascial fibres (arrows) are mixed with fat, to form a pressure-tolerant, fibro-adipose tissue (FT). DP, distal phalanx; IP, intermediate phalanx.

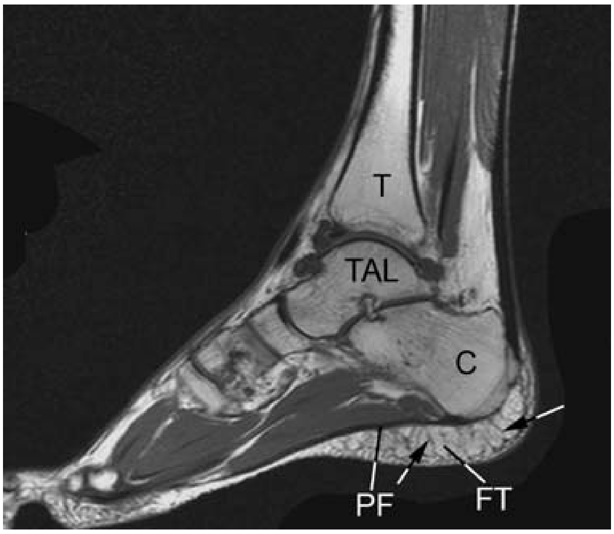

Fig. 3 A T2-weighted sagittal-plane MRI of the foot showing the extensive areas of fibro-adipose tissue (FT) that are characteristic of the heel, deep to the plantar fascia (PF). C, calcaneus; TAL, talus; T, tibia. The fibrous septa within the fibroadipose tissue are arrowed. Image kindly provided by D. McGonagle.

sheets of collagen fibres coupled with the presence of elastin (Kawamata et al. 2003). The relative independence of the collagen sheets from each other promotes skin sliding and further stretching is afforded by a re-alignment of collagen fibres within the lamellae. The skin is brought back to its original shape and position by elastic recoil when the deforming forces are removed. As Kawamata et al. (2003) point out, one of the consequences of the movement-promoting characteristics of the superficial fascia is that the blood vessels and nerves within it must run a tortuous route so that they can adapt to an altered position of the skin, relative to the deeper structures.

There are some sites where the skin is tightly bound to the underlying tissues to prevent or restrict movement – as in the palmar or plantar aspects of the hands and feet (Fig. 2). If movement were allowed to occur here within fascial planes, it would conflict with the requirements of facilitating a firm grip. Hence, loose connective tissue is sparse beneath the skin in the palms and soles. Indeed, it is completely absent at the finger creases on the palmar sides of the interphalangeal joints, so that the skin immediately covers fascial tendon sheaths. This explains why puncture wounds at the creases carry a risk of infection to these structures (Fifield, 1939). Although deep fascia elsewhere in the limbs is often not so tightly bound to the skin, nevertheless cutaneous ligaments extending from deep fascia to anchor the integument are much more widespread than generally recognized and serve to resist a wide variety of forces, including gravitational influences (Nash et al. 2004).

Fascia in the form of loose, areolar connective tissue surrounds skeletal muscle fibres (forming the endo- and epimysium) and creates thin films of tissue between adjacent muscles. Again, such fasciae are important in promoting movement – by allowing one muscle or fibre to move independently of its neighbour. By contrast, the deep fasciae in the limbs and back are typically dense connective tissue sheets that have large numbers of closely packed collagen fibres. The dominant cell is a fibroblast, although the accumulation of actin stress fibres within these cells in response to mechanical loading has led some authors to consider many of these cells to be myofibroblasts (Schleip et al. 2007) – see below. However, where dense fascia is subject to significant levels of compression [e.g. in certain retinacula (Benjamin & Ralphs, 1998, Kumai & Benjamin, 2002) or in parts of the plantar fascia], the cells have a chondrocytic phenotype and the tissue can be regarded as fibrocartilage. Yet other forms of fascia can be considered mixtures of fibrous and adipose tissues – 'fibro-adipose tissue'. This is particularly characteristic of the palms, the soles and the palmar/plantar pads of the fingers and toes (Figs 2 and 3). At all these locations, fibroadipose tissue functions as an important pressure-tolerant device (see below).

Of the different histological forms of fascia, it is areolar tissue that is characterized by the greatest diversity of cell types. Its resident fibroblasts are integral to mechanotransduction. They communicate with each other via gap junctions and respond to tissue stretch by shape changes mediated via the cytoskeleton (Langevin et al. 2005; Langevin, 2006). In stretched tissue, the cells are sheet-like and have large cell bodies, whereas in unstretched tissue, they are smaller and dendritic in shape, and have numerous, attenuated cell processes (Langevin et al. 2005). According to Bouffard

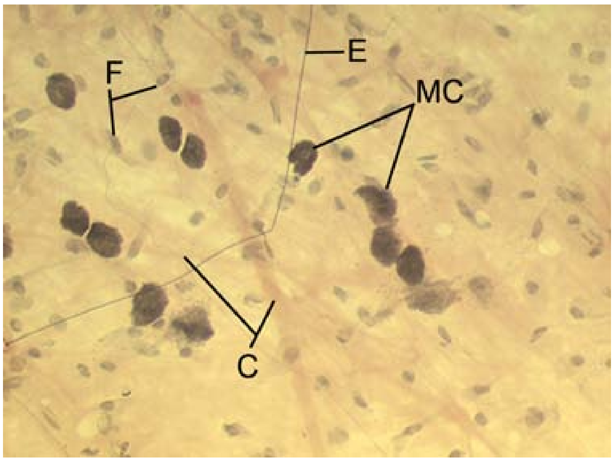

Fig. 4 A spread of rat superficial fascia showing the presence of pale bundles of collagen fibres (C) of different thicknesses and dark, uniformly-thin, elastic (E) fibres. There are also a number of large, heavily granulated mast cells (MC). The majority of the other cells are only recognizable by their nuclei and are likely to be fibroblasts (F).

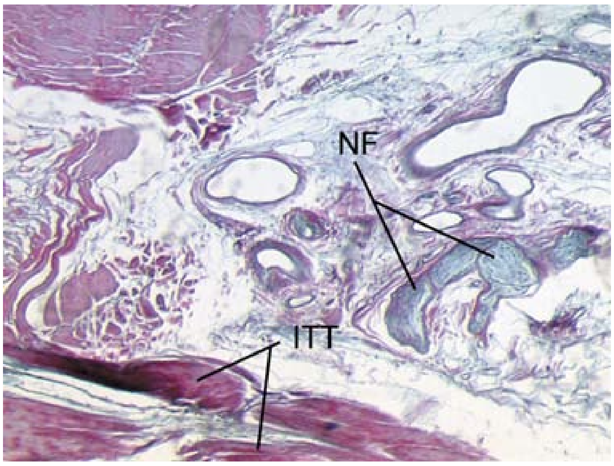

Fig. 5 The iliotibial tract (ITT) is a fascial structure that is composed of dense connective tissue. In the region of the lateral femoral epicondyle, it is juxtaposed to an area of adipose tissue that lies immediately deep to it, which contains prominent nerve fibres (NF).

et al. (2008), brief stretching decreases TGF-β1-mediated fibrillogenesis, which may be pertinent to the deployment of manual therapy techniques for reducing the risk of scarring/fibrosis after an injury. As Langevin et al. (2005) point out, such striking cell responses to mechanical load suggest changes in cell signaling, gene expression and cell-matrix adhesion. The cell shape changes may also influence tension with the connective tissue itself. The many important contributions of Langevin and her colleagues in recent years have significant implications for understanding the basis of the therapeutic responses to the physical manipulation of fascia that is central to numerous 'alternative therapies' (e.g. massage, yoga, Rolfing, etc.).

Mast cells are also found in areolar connective tissue (Fig. 4) together with a variable population of 'immigrant' cells that have entered the fascia by migrating across the walls of thin blood vessels coursing through them. These essentially represent white blood cells or their differentiation products and thus comprise lymphocytes, granulocytes and macrophages. All forms of fascia contain collagen and/or elastic fibres, although clearly deep fascia is more densely packed with fibres than areolar connective tissue (Fig. 5).

Standard histology texts give a broad treatment of connective tissue cell types and the subject has also been briefly addressed by Lindsay (2008) in his recent textbook on fascia. Although the fibroblast is obviously a key cell within fascia, extensive attention has already been paid to this cell by connective tissue biologists and thus interest is focused in the current article on the suggestion that many fascial 'fibroblasts' are really better viewed as myofibroblasts.

Myofibroblasts

The term 'myofibroblast' arose from wound-healing studies in the 1950s that were being conducted at the time the cytoskeletal concept was emerging (Gabbiani, 2003, 2007). Myofibroblasts were regarded as fibroblasts that acquire smooth muscle cell features when granulation tissue develops in a wound. The accumulation of actin stress fibres and the development of adherens and gap junction between cells, enable the cells to become contractile and help to close the edges of a wound (Gabbiani, 2007). After wound closure, myofibroblasts normally diminish in number by apoptosis (Desmouliere et al. 1997). However, in certain pathological circumstances, they can remain and their long-term presence is thought to be associated with a whole spectrum of fibrotic conditions (Gabbiani, 2003). Many affect fascia. A good example is Dupuytren's contracture – considered in further detail below. It is associated with the development of nodules of proliferative cells in the palmar aponeurosis which progress to form contractile, collagenous cords (Satish et al. 2008). Both the nodules and particularly the cords contain myofibroblasts. Typically, the myofibroblasts are surrounded by a region of extracellular matrix (ECM) that has certain similarities to the basal lamina around true muscle cells (Berndt et al. 1994). Myofibroblasts also appear in other fibrotic conditions. Fidzianska & Jablonska (2000) have drawn attention to a rare scleroderma-like condition known as congenital fascial dystrophy in which patients have difficulty in walking properly and adopt a tip-toe gait pattern, have limb contractures, limited joint mobility and very hard skin. The principle abnormalities visible in the superficial fascia and fascia lata of the lower limb are the formation of large-diameter collagen fibrils and the appearance of myofibroblasts.

It is worth noting that although there are changes to the actin cytoskeleton in loose connective tissue fibroblasts subjected to stretch, the cells do not form true stress fibres (or zonula occludens) and are thus not myofibroblasts

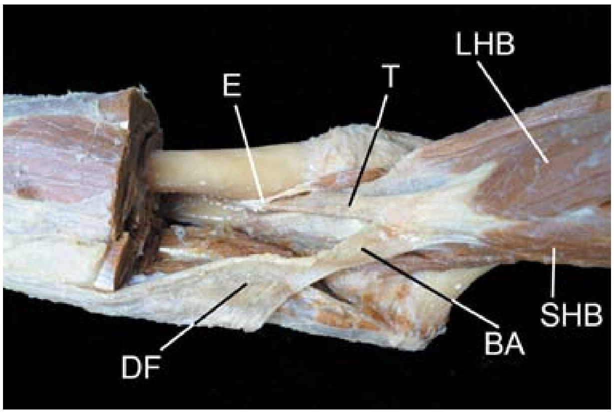

Fig. 6 The bicipital aponeurosis (BA) is a classic example of a fascial expansion which arises from a tendon (T) and dissipates some of the load away from its enthesis (E). It originates from that part of the tendon associated with the short head of biceps brachii (SHB) and blends with the deep fascia (DF) covering the muscles of the forearm. The presence of such an expansion at one end of the muscle only, means that the force transmitted through the proximal and distal tendons cannot be equal. LHB, long head of biceps brachii. Photograph courtesy of S. Milz and E. Kaiser.

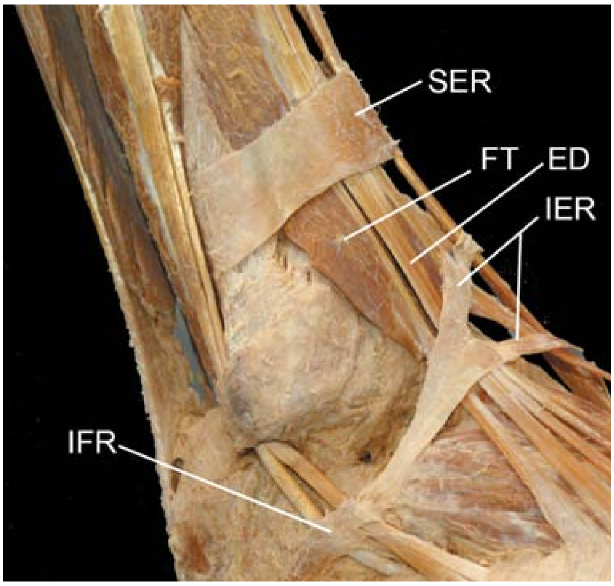

Fig. 7 The retinacula of the ankle region dissected artificially away from the rest of the deep fascia in traditional manner. Note how muscle fibres of fibularis (peroneus) tertius (FT) pass beneath the superior extensor retinaculum (SER), but how extensor digitorum (ED) is entirely tendinous at that level. IER, inferior extensor retinaculum; IFR, inferior fibularis (peroneal) retinaculum. Photograph courtesy of S. Milz and E. Kaiser.

(Langevin et al. 2006b). In contrast, Schleip et al. (2007) have reported myofibroblasts in the rat lumbar fascia (a dense connective tissue). The cells can contract *in vitro* and Schleip et al. (2007) speculate that similar contractions *in vivo* may be strong enough to influence lower back mechanics. Although this is an intriguing suggestion that is worthy of further exploration, it should be noted that tendon cells immunolabel just as strongly for actin stress fibres as do fascial cells and this may be associated with tendon recovery from passive stretch (Ralphs et al. 2002). Finally, the reader should also note that true muscle fibres (both smooth and skeletal) can sometimes be found in fascia. Smooth muscle fibres form the dartos muscle in the superficial fascia of the scrotum and skeletal muscle fibres form the muscles of fascial expression in the superficial fascia of the head and neck.

Fascial expansions of entheses

The region where a tendon, ligament or joint capsule attaches to a bone (its 'enthesis') is an area of great stress concentration, for it represents the meeting point between hard and soft tissues. Consequently, entheses are designed to reduce this stress concentration, and the anatomical adaptations for so doing are evident at the gross, histological and molecular levels. Thus many tendons and ligaments flare out at their attachment site to gain a wide grip on the bone and commonly have fascial expansions linking them with neighbouring structures. Perhaps the best known of these is the bicipital aponeurosis that extends from the tendon of the short head of biceps brachii to encircle the forearm flexor muscles and blend with the antebrachial deep fascia (Fig. 6). Eames et al. (2007) have suggested that this aponeurosis may stabilize the tendon of biceps brachii distally. In doing so, it reduces movement near the enthesis and thus stress concentration at that site.

A further example of a fascial expansion arising from a tendon is that which emanates from the quadriceps tendon as it attaches to the upper pole of the patella. Here, there is a fascial sheet that passes anterior to the patella to contribute superficial fibres to the patellar tendon (Toumi et al. 2006). In a similar manner, the Achilles tendon not only attaches to the posterior aspect of the calcaneus, but also has fascial continuity both with the plantar aponeurosis over the back of the heel (Wood Jones, 1944; Snow et al. 1995; Milz et al. 2002) and with the fibrous septa of the heel fat pad (M. Benjamin, unpublished observations). As teachers of gross anatomy well know, there are numerous, largely unrecognized fascial sheets interconnecting tendons and ligaments in the foot. Among the better known are the tendinous expansions of tibialis posterior that attach to every tarsal bone in the foot except the talus.

Retinacula

Retinacula are strap-like thickenings of dense connective tissue that bind down structures near joints (Fig. 7) and are sometimes used as autografts in the repair of torn ligaments (Saragaglia et al. 1997). Where the levels of compression between tendon and retinacula are significant, both may be fibrocartilaginous (Benjamin et al. 1995). Retinacula not only prevent tendons from bowstringing, but also provide a smooth contact surface for them to slide longitudinally

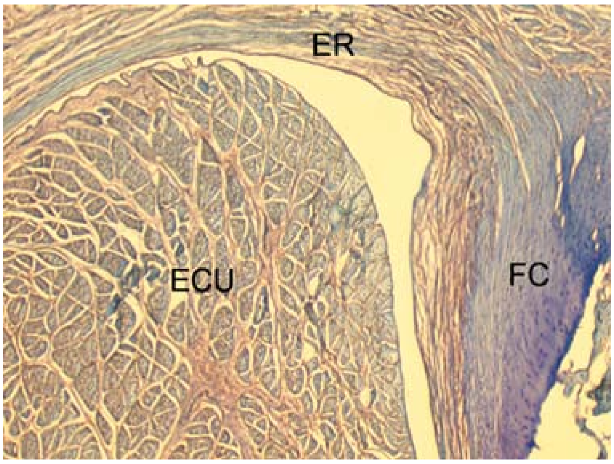

Fig. 8 A transverse section through the extensor retinaculum (ER) of the forearm in the region of the tendon of extensor carpi ulnaris (ECU). Note how the retinaculum itself is largely fibrous, but that its ulnar enthesis is fibrocartilaginous (FC).

when their associated muscle contracts. As they attach to bone to create tunnels for their associated tendons, a pathological mismatch can develop between the size of the tunnel and that of the tendons that is commonly referred to as stenosing tenosynovitis (Palmborg, 1952; Crimmins & Jones, 1995; Taki et al. 2007).

The various retinacula at the ankle have a criss-cross arrangement of collagen fibres that enhances their stability. Abu-Hijleh & Harris (2007) compare this arrangement to the straps that cross in a sandal or the laces in a shoe. The retinacula around the ankle average 1 mm in thickness, but variations are considerable (Numkarunarunrote et al. 2007). Retinacula can occasionally be congenitally absent and are commonly subject to traumatic ruptures (Numkarunarunrote et al. 2007). Either circumstance can result in a marked subluxation of the tendons beneath them.

Even where a retinaculum is dense fibrous connective tissue, its entheses may be fibrocartilaginous (Fig. 8). This is of particular significance in relation to the superior peroneal retinaculum that binds down the peroneal tendons behind the lateral malleolus. If the retinaculum is torn (e.g. as a skiing injury), the stiffening associated with its fibrocartilaginous entheses creates a sharp edge to the tissue that can 'razor' through the subluxed peroneal tendons (Kumai & Benjamin, 2003).

Retinacula are typically regional specializations of the deep fascia, though as Abu-Hijleh & Harris's (2007) excellent illustrations show, they are nowhere near as distinct as most elementary texts depict. There are also other regions of deep fascia that have a retinacular function, but are not formally referred to as 'retinacula' (Abu-Hijleh & Harris, 2007). Thus, there are thickenings of deep fascia along the medial and lateral borders of the foot that the authors compare to the stitches on the side of a shoe, helping to anchor the deep fascia on the dorsum of the foot and augment its stabilizing action on the extensor tendons. The deep fascia also acts as a retinaculum over the extensor tendons in the region of the metatarsophalangeal joints (Abu-Hijleh & Harris, 2007) and over the Achilles tendon in the lower part of the leg (Benjamin et al. 2007).

Typically, it is tendons rather than muscle bellies that pass beneath retinacula, because the compressive forces acting here are not conducive to the survival of highly vascularized tissues such as skeletal muscle. However, notable exceptions are flexor hallucis longus and the inconstant fibularis tertius, where muscle fibres also can lie deep to the superior extensor retinaculum (Haumont et al. 2007; Fig. 7). Significantly though, muscle fibres are retracted in a dorsiflexed foot, when pressure between the retinaculum and the structures deep to it is likely to be greatest. Nevertheless, the anatomical relationship between the muscle–tendon unit and the retinaculum is important clinically for it means that flexor hallucis longus in particular can be vulnerable to the effects of compression syndrome as it passes under the retinaculum. This may necessitate surgery on both the crural fascia and the retinaculum itself (Haumont et al. 2007).

Fascial innervation

Several reports suggest that fascia is richly innervated, and abundant free and encapsulated nerve endings (including Ruffini and Pacinian corpuscles) have been described at a number of sites, including the thoracolumbar fascia, the bicipital aponeurosis and various retinacula (Stilwell, 1957; Tanaka & Ito, 1977; Palmieri et al. 1986; Yahia et al. 1992; Sanchis-Alfonso & Rosello-Sastre, 2000; Stecco et al. 2007a). However, it is sometimes difficult to decide from the literature whether a particular piece of deep fascia is itself innervated or whether the nerve fibres lie on its surface or in areolar or adipose tissue associated with it. Changes in innervation can occur pathologically in fascia, and Sanchis-Alfonso & Rosello-Sastre (2000) report the ingrowth of nociceptive fibres, immunoreactive to substance P, into the lateral knee retinaculum of patients with patello-femoral malignment problems.

Stecco et al. (2008) argue that the innervation of deep fascia should be considered in relation to its association with muscle. They point out, as others have as well (see below in 'Functions of fascia') that many muscles transfer their pull to fascial expansions as well as to tendons. By such means, parts of a particular fascia may be tensioned selectively so that a specific pattern of proprioceptors is activated.

Despite the contribution of the above studies, our understanding of fascial innervation is still very incomplete and it is likely that there are regional differences of functional significance, as with ligaments. It is worth noting therefore that Hagert et al. (2007) distinguish between ligaments at the wrist that are mechanically important yet poorly innervated, and ligaments with a key role in sensory perception that are richly innervated. There is a corresponding histological difference, with the sensory ligaments having more conspicuous loose connective tissue in their outer regions (in which the nerves are located). Comparable studies are not available for deep fascia, although Stecco et al. (2007a) report that the bicipital aponeurosis and the tendinous expansion of pectoralis major are both less heavily innervated than the fascia with which they fuse. Where nerves are abundant in ligaments, blood vessels are also prominent (Hagert et al. 2005). One would anticipate similar findings in deep fascia.

From comparisons with tendons and ligaments, where nerve and blood vessels are generally a feature of the associated loose connective tissue sheaths (Hagert et al. 2007), it might be anticipated that fascial nerves would also be commonly surrounded by areolar connective tissue. The levels of mechanical loading to which dense connective tissue is adapted are not likely to be conducive to having nerves and densely packed collagen fibres too close together. Abnormal levels of mechanical loading have been suggested to cause nerve damage in knee retinacula (Sanchis-Alfonso & Rosello-Sastre, 2000). Nevertheless, Stecco et al. (2007a) do show evidence of fascial nerves that are closely related to densely packed collagen fibres and thus lie within the fascia itself. In the distal part of the iliotibial , however, it is the tissue adjacent to deep fascia that is more conspicuously innervated than the fascia itself (Fairclough et al. 2006; Fig. 5).

Some of the nerve fibres associated with fascia are adrenergic and likely to be involved in controlling local blood flow, but others may have a proprioceptive role. Curiously, however, Bednar et al. (1995) failed to find any nerve fibres in thoracolumbar fascia taken at surgery from patients with low back pain.

The functions of fascia

Some of the numerous functions that have been associated with fascia have inevitably been mentioned above in relation to its basic organization. Further important functions are dealt with specifically below.

The role of fascia in creating distinctive compartments for muscles and in acting as an ectoskeleton for their attachment

The unyielding character of the deep fascia enables it to serve as a means of containing and separating groups of muscles into relatively well-defined spaces called 'compartments'. The deep fascia integrates these compartments and transmits load between them. As the compartmentalizing role of deep fascia is a function it performs in conjunction with the associated bones and intermuscular septa (Fig. 1), the compartments are sometimes called osteofascial compartments. Each segment of the limbs (e.g. arm, forearm, thigh, leg or foot) has its own characteristic compartments separating functional groups of muscles with distinctive embryological origins, blood and nerve supplies. The compartments are generally named according to their position (anterior, posterior, medial, lateral, etc.) or the actions of their contained muscles (flexors, extensors, evertors, adductors, etc.).

The fascial intermuscular septa are often attached to periosteum, and as these two structures share the same developmental origin, it means that fascia rarely makes contact with bone without also attaching to it (Grant, 1948). It is not surprising therefore that fascial entheses have been implicated in overuse injuries – e.g. medial tibial stress syndrome (Bouche & Johnson, 2007). These authors have suggested that the tenting effect of tendons associated with eccentric muscle contraction in the posterior compartment of the leg increases the tensile load on the deep fascia. This is ultimately relayed to the fascial attachment site on the tibial crest. Bouche & Johnson (2007) reported a linear relationship between tension of any one (or all) of the tendons involved and strain levels in the tibial fascia. Simple inspection of cadaveric specimens in which muscle contraction has been simulated by pulling on the tendons with a pneumatic actuator, shows clear evidence of fascial tenting. The authors point out that (1) eccentric contraction of the implicated muscles is increased when exercising on hard surfaces to promote shock dissipation and that individuals with marked foot pronation also have increased eccentric contractions; (2) both running on hard ground and over-pronation are known risk factors for medial tibial stress syndrome ('shin splints'). Their fascial theory is supported by the common finding of inflammatory changes in the crural fascia of patients with shin splints.

Where deep fascia and intermuscular septa partition muscles, they may also serve for their attachment and this makes such fascia particularly thick (Grant, 1948). One of the most influential anatomists of the 20th century, Professor Frederic Wood Jones, coined the term 'ectoskeleton' to capture the idea that fascia could serve as a significant site of muscle attachment – a 'soft tissue skeleton' complementing that created by the bones themselves (Wood Jones, 1944). It is clearly related to the modern-day concept of 'myofascia' that is popular with manual therapists and to the idea of myofascial force transmission within skeletal muscle, i.e. the view that force generated by skeletal muscle fibres is transmitted not only directly to the tendon, but also to connective tissue elements inside and outside the skeletal muscle itself (Huijing et al. 1998; Huijing, 1999). Several authors have commented on the extent to which muscles attach to fascia as well as bone and on how this has been overlooked in the past. Thus Kalin & Hirsch (1987) found that only eight of the 69 interossei muscles they studied in the feet of dissecting room cadavers had attachments that were limited to bone. In the vast majority of cases the muscles had extensive attachments to ligaments and fascia that effectively link the muscles together to promote their contraction as a co-ordinated unit. A similar point is made by Chang & Blair (1985) in relation to the attachments of adductor pollicis. These authors described prominent attachments of its transverse head to fascia covering the palmar interossei that had not been recorded previously. As the palmar interossei and adductor pollicis are supplied by the same nerve and are both adductors of the thumb (Mardel & Underwood, 1991), it is likely that fascial interconnections promote their coordinated activity.

An interesting implication of recognizing the existence of fascial routes of force transmission is that in muscles with tendons at both ends, the forces within these tendons may be unequal (Huijing & Baan, 2001a). Furthermore, as Huijing et al. (2003) point out, what are generally taken to be morphologically discrete muscles from an anatomical perspective, cannot be considered isolated units controlling forces and moments. One can even extend this idea to embrace the concept that agonists and antagonists are mechanically coupled via fascia (Huijing, 2007). Thus Huijing (2007) argues that forces generated within a prime mover may be exerted at the tendon of an antagonistic muscle and indeed that myofascial force transmission can occur between *all* muscles of a particular limb segment.

It is intriguing to note that Huijing et al. (2003) follow Wood Jones's (1944) lead and liken compartment-forming fascia to a skeleton. Indeed, they suggest that one of the functions of myofascial force transmission is to stiffen this skeleton and hence augment its function. Whether surgery to this 'skeleton' (notably fasciotomies undertaken to reduce intracompartmental pressure – see below) changes the force-generating capacity of limb muscles is a valid question that does not seem to have been addressed, although Huijing & Baan (2001b) report significant changes in the forces generated by muscles in the anterior compartment of the rat leg following fasciotomy. Furthermore, as neighbouring muscles can be strongly attached to each other by extramuscular connective tissue, it may well be that the tenotomies favoured by some surgeons in the treatment of tennis elbow affect the force transmission of neighbouring muscles in way that has not been thoroughly explored.

Wood Jones (1944) was particularly intrigued by the ectoskeletal function of fascia in the lower limb. He related this to man's upright stance and thus to the importance of certain muscles gaining a generalized attachment to the lower limb when it is viewed as a whole weight-supporting column, rather than a series of levers promoting movement. He singled out gluteus maximus and tensor fascia latae as examples of muscles that attach predominantly to deep fascia rather than bone (Wood Jones, 1944). Viewing the deep fascia as an ectoskeleton emphasizes the importance of considering the responses of this tissue to distraction procedures designed to lengthen a limb segment. According to Wang et al. (2007), a lengthening rate of 1 mm day^{-1} in a rabbit model of distraction in which the tibia is ultimately increased in length by 20%, results in a corresponding re-modelling of the deep fascia. This ensures that the tensile forces operating in the fascia match the increasing length of the limb so that the fascia does not impede distraction.

Although gastrocnemius is strikingly lacking in any significant attachment to its overlying deep fascia (see above), it nevertheless gains extensive anchorage to a thick fascial sheet on its deep surface. As with any such well-developed aponeurosis on the surface of a muscle, this inevitably restricts the range of movement. Indeed, a surgical approach that has been advocated to improve the range of motion in cerebral palsy patients with an equinus deformity is to make a series of transverse incisions through this fascia (Saraph et al. 2000).

This ectoskeletal role of fascia is particularly obvious in relation to the intrinsic muscles of the foot and tibialis anterior in the upper part of the leg. The firm fascial attachments of tibialis anterior account for the longitudinal orientation of the fascial fibres at this site (i.e. parallel to the long axis of the muscle) and for the difficulty in making a clean dissection. In marked contrast, the deep fascia on the back of the leg covering the muscle bellies of gastrocnemius, does not serve for muscle attachment at all (Grant, 1948). This indicates the importance of this particular muscle belly to be able to move independently of its fascia during the powerful contractions that it performs in its weight-bearing capacity. Advantage is taken of the absence of muscle–fascia attachment in this location in the design of surgical interventions to augment calf size, either for purely aesthetic reasons or to correct a deformity resulting from illness (Niechajev, 2005). Silicone implants can be placed between the investing deep fascia covering gastrocnemius and the muscle itself (Niechajev, 2005).

The general thinness of the deep fascia covering the large, flat pectoral muscles in the chest is in line with the need for the thorax to expand and contract during breathing (Grant, 1948). In contrast, the deep fascia is particularly thick in the leg in line with its compartmentalizing and ectoskeletal roles. Strong intermuscular septa pass inwards from the deep fascia to fuse with the periosteum of the tibia and fibula and create separate compartments for the dorsiflexor, peroneal and plantarflexor muscles (Fig. 1). The anterior compartment houses the dorsiflexor muscles (which include tibialis anterior, discussed above) and is of particular clinical significance in relation to compartment syndrome. This is a painful and potentially limb-threatening condition that occurs when pressure builds up within the space that is limited by the tough and unyielding deep fascia impairing blood flow. It can occur as a result of sudden trauma (e.g. a haematoma) or as a consequence of overuse. The resulting muscle ischaemia may require an emergency fasciotomy to reduce the pressure. If surgery is unduly delayed, serious systemic complications can arise, including renal failure (Mubarak & Owen, 1975). Anterior compartment syndrome is perhaps the best known, but compartment syndrome can occur elsewhere as well. From an anatomical perspective, it is worth remembering that the fascial compartments characterizing the hands and feet are small (matching the size of the muscles; Grant, 1948) and thus pressure can quickly build up from a haematoma and trigger a collapse of the local circulation.

The attachment of seemingly diverse muscles to a common fascia means that fascia is in a strategic position to co-ordinate muscle activity. This is not surprising bearing in mind the integrating role of the connective tissue sheaths within a given muscle, harnessing the activity of its muscle fibres into an integrated whole. In an influential paper, Vleeming et al. (1995) have highlighted the importance of the thoracolumbar fascia in integrating the activity of muscles traditionally regarded as belonging to the lower limb, upper limb, spine or pelvis and whose action is thus often considered in that territory alone. They have argued that a common attachment to the thoracolumbar fascia means that the latter has an important role in integrating load transfer between different regions. In particular, Vleeming et al. (1995) have proposed that gluteus maximus and latissimus dorsi (two of the largest muscles of the body) contribute to co-ordinating the contralateral pendulum like motions of the upper and lower limbs that characterize running or swimming. They suggest that the muscles do so because of a shared attachment to the posterior layer of the thoracolumbar fascia. Others, too, have been attracted by the concept of muscle-integrating properties of fascia. Thus Barker et al. (2007) have argued for a mechanical link between transversus abdominis and movement in the segmental neutral zone of the back, via the thoracolumbar fascia. They feel that the existence of such fascial links gives an anatomical/biomechanical foundation to the practice in manual therapy of recommending exercises that provoke a submaximal contraction of transversus abdominis in the treatment of certain forms of low back pain. Stecco et al. (2007a,b, 2008) have also given us further examples of how fascia in the upper limb links numerous different muscles together. They suggest that a basal level of tension that is loaded onto the fascia by flexor and extensor muscles alike, contributes to myofascial continuity and possibly activates specific patterns of proprioceptors associated with the fascia.

One of the most striking examples of how fascia links muscles together concerns the extensor muscles of the forearm in the region of the lateral epicondyle. Although standard anatomy texts often simplify the position greatly by saying that the extensor muscles attach to the common extensor origin, the reality is rather more complex. Clearly, the area of bone provided by the lateral epicondyle is insufficient to attach the numerous muscles on the back of the forearm that arise from the common extensor origin. What happens instead is that the muscles attach to each other in this region via fascia and by this means they can be crowded onto a limited surface of bone. This is well illustrated by the work of Briggs & Elliott (1985) who dissected 139 limbs, and found that extensor carpi radialis brevis (the muscle most commonly associated with tennis elbow) actually attached directly to the epicondylar region in only 29 cases. Much more frequently it was linked by fascia to extensor carpi radialis longus, extensor digitorum communis, supinator and the radial collateral ligament.

The circulatory-support function of deep fascia – the muscle pump phenomenon

An important function of deep fascia in the limbs is to act as a restraining envelope for muscles lying deep to them. When these muscles contract against a tough, thick and resistant fascia, the thin-walled veins and lymphatics within the muscles are squeezed and their unidirectional valves ensure that blood and lymph are directed towards the heart. Wood Jones (1944) contests that the importance of muscle pumping for venous and lymphatic return is one of the reasons why the deep fascia in the lower limb is generally more prominent than in the upper – because of the distance of the leg and foot below the heart. However, there is also a thick, inelastic fascia covering the scapular muscles *above* the level of the heart. This has a bearing on the high intramuscular pressure in supraspinatus that is implicated in shoulder pain and muscle isthemia (Jarvholm et al. 1988).

That the deep fascia is under tension from the contraction of its contained muscles is evident when it is punctured, for the muscle bulges through it (Le Gros Clark, 1945). The protrusions are known as fascial herniations and may merit a fasciotomy if muscle ischaemia is a risk (de Fijter et al. 2006). The anti-gravitational nature of the 'muscle pump' associated with the crural fascia is commonly mentioned in anatomy texts and has traditionally been reinforced to generations of medical students by the observation that long periods of standing still (e.g. as with soldiers on ceremonial duty) can cause blood to pool in the legs and feet, resulting in inadequate venous return and fainting. More recently, the emphasis has turned to deep vein thrombosis as a way of illustrating the importance of the muscle pump. The stagnancy of peripheral blood flow that comes from long periods of static posture (again linked to inadequate muscle activity) can lead to the formation of blood clots – which can be fatal. Understanding the role of fascia in the muscle-pumping actions of the lower limb depends on a sound understanding of the venous system. This is adequately covered in standard anatomy teaching texts and has also been addressed in a recent review article by Meissner et al. (2007). Briefly, a distinction is made between superficial and deep veins according to their relative position with respect to the deep fascia. The two sets of veins are linked by 'perforating veins' that penetrate the deep fascia, linking veins either side. All three sets of vessels have valves that prevent backflow and help to divide the hydrostatic column of blood into segments (Meissner et al. 2007). Valvular incompetence in the leg thus diminishes muscle pump function. Curiously, however, the perforating veins in the foot lack valves and thus do allow bidirectional flow. According to Meissner et al. (2007), the calf muscle pump is the most significant and has the largest capacitance, but is primed by muscle pumps in the foot. They view the influence of similar pumps in the thigh as being minimal.

On occasions, the deep fascia may have too severe a restraining influence on its contained muscles, so that they are in danger of inadequate perfusion because their vessels are occluded for prolonged periods. The outcome is known as a 'compartment syndrome' and can be acute or chronic. Acute compartment syndromes may be associated with trauma where there is bleeding within the compartment or may be elicited by a plaster-cast applied too tightly to a limb. Chronic compartment syndromes stem from an exercise-induced increase in intra-compartmental pressure that compromises normal neuromuscular function (Bourne & Rorabeck, 1989). The muscle ischaemia stemming from acute compartment syndrome can be limb- (and sometimes life-)threatening and represents a surgical emergency. The confining deep fascia must be cut to reduce the pressure. The urgency with which a fasciotomy needs to be performed in severe acute cases is indicated by the observation that significant necrosis can occur within 3 h (Vaillancourt et al. 2004). In mild cases of exercise-induced compartment syndrome, the pressure on the muscles may be reduced by applying ice.

In view of the importance of gravitational influences in accounting for the prominence of deep fascia in the leg, it might be surmised that fasciotomies would seriously impair the venous calf pumps. This is the conclusion of Bermudez et al. (1998) who caution that patients who have had fasciotomies are at risk of developing chronic venous insufficiency in the long term. This, however, contrasts with the earlier findings of Ris et al. (1993).

The protective role of fascia

In certain regions of the body, fascia has a protective function. Thus, the bicipital aponeurosis (lacertus fibrosus), a fascial expansion arising from the tendon of the short head of biceps brachii (Athwal et al. 2007), protects the underlying vessels. It also has mechanical influences on force transmission and stabilizes the tendon itself distally (Eames et al. 2007). There are also very tough sheets of connective tissue in the palms and soles. The palmar aponeurosis in the former, and the plantar aponeurosis in the latter (Fig. 9), protect the vessels and nerves that run deep to them between the proximal and distal parts of the hand and foot. They also tie the skin to the skeleton, controlling its displacement during locomotion (Bojsen-Moller & Flagstad, 1976). However, fascia in the form of dense connective tissue is not best suited to protect against the compressive forces that act during walking or the creation of a power grip. As fat is fluid and thus incompressible, it is far better for this task. This is well illustrated by the presence and regional distribution of tubular sleeves of adipose tissue around the digital nerves in the foot. The fat is strikingly present as the nerves pass through the weight-bearing ball of the foot *en route* to the toes (see Fig. 2 in Bojsen-Moller & Flagstad, 1976). Fat also protects the metatarsals themselves in the region – a point recognized by Bojsen-Moller & Flagstad, who referred to the fat in this region as creating sub-metatarsal cushions. Indeed, these cushions also protect the flexor tendons and their sheaths as they pass through the area.

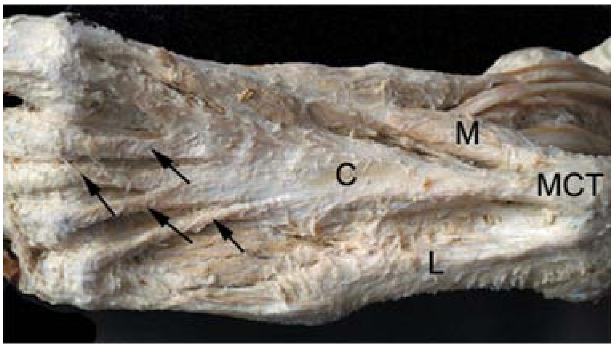

Fig. 9 The central (C), medial (M) and lateral (L) parts of the plantar aponeurosis in the sole of the foot. Note the extensive attachment of the aponeurosis to the medial calcaneal tubercle (MCT) and the distal expansions of the aponeurosis passing to the lesser toes (arrows). Photograph kindly provided by S. Milz and E. Kaiser.

Regional considerations of fascia

Even a cursory inspection of any large anatomy tome will inform the reader of a bewildering diversity of named fascia, reflecting the requirements of regional anatomy. In the limbs and back, these include the fascia lata and iliotibial tract, the clavipectoral, axillary, brachial, antebrachial, thoracolumbar, plantar, palmar, crural and gluteal fascia, and many retinacula and pulleys that are increasingly prominent towards the distal parts of the limbs. However, as Wood Jones (1944) points out, there is also an argument for considering such a daunting list of terms to be redundant and a hindrance to understanding general principles of fascial biology. It is with his cautionary note in mind that only a limited number of fascia are considered here individually by name. It is more important to see fascia as a connective tissue continuum throughout the body, uniting and integrating its different regions. Yes, the names are convenient labels – but little more than that. Just as Myers (1987) has argued persuasively that the traditional approach of anatomical dissection viewing muscles as independent units has obscured the 'trains of muscle continuity' that he eloquently describes, so too is the naming and study of a plethora of fasciae in isolation, a barrier to understanding the bigger picture of fascial function. It is thus critical to appreciate that loose connective tissue forms one of the great highways of the body – a favoured route by which blood vessels, nerves and lymphatics journey between regions (Wood Jones, 1944; Le Gros Clark, 1945). Evidence is also accumulating to suggest that fascia have an important integrating function as a proprioceptive organ and that they can coordinate the action of different muscles by acting as a common ectoskeleton that provides a large area of grip for muscle attachment (Wood Jones, 1944).

The palmar and plantar fasciae

Both the palm of the hand and the sole of the foot contain a tough sheet of dense connective tissue that protects the underlying vessels and nerves from the pressure associated with grip or body weight. Both are firmly attached to the thick skin overlying them, so as to limit movement between the integument and neighbouring structures and both send prolongations extending into the digits. The former sheet of connective tissue is called the palmar fascia and the latter the plantar fascia. Both are closely related to the tendon of a vestigial muscle, but Caughell et al. (1988) argue that the palmar fascia and the tendon of palmaris longus are best viewed as separate anatomical structures that develop independently. Nevertheless, palmaris longus does serve as a tensor of both the palmar and thenar fasciae (Stecco et al. 2007b). As Botte (2003) points out, this is an aid to grip, as by providing a firm anchorage for the palmar aponeurosis, the muscle helps to resist shearing forces in this region of the hand.

Palmar fascia

The palmar fascia (synonym *palmar aponeurosis*) is triangular-shaped and located in the hollow of the palm. Its apex lies proximally near the wrist and is continuous with the flexor retinaculum and the tendon of palmaris longus, and its base lies distally near the fingers and sends four prolongations (pretendinous bands) into the fingers to blend with the fibrous flexor sheaths surrounding the flexor tendons (Fifield, 1939; de-Ary-Pires et al. 2007). Branches of the bands give rise to cutaneous ligaments, eponymously referred to as Cleland's and Grayson's ligaments. These are thought to stabilize the neurovascular bundles within the digits during finger movements and to anchor the skin in a way that limits its displacement during finger flexion (de-Ary-Pires et al. 2007). Between the four digital processes are three fat-filled intervals containing neurovascular bundles and the lumbrical muscles. Their existence is of great importance to hand surgeons in connection with the spread of any infection from the fingers into the subaponeurotic spaces of the palm (Fifield, 1939). Similarly, there is a sheet of interosseous fascia jointly covering the interossei and the metacarpals that contributes to the formation of a potential deep fascial space of the palm between it and the deep flexor tendons and their lumbricals (Fifield, 1939). Again, this is significant in relation to the spread of pus. There is a vertical septum attached to the 3rd metacarpal bone that divides the deep fascial space into medial and lateral compartments and confines any pus to one side alone (Fifield, 1939). The existence of two other potential spaces also hinges on the presence of fascial sheets, i.e. the thenar and mid-palmar spaces (Fifield, 1939). Again, their importance relates to the tracking and confinement of pus.

The palmar fascia and its pretendinous bands are of particular interest in relation to Dupuytren's contracture, one of the most common conditions managed by hand surgeons, but also one frequently seen by many other doctors as well (Hart & Hooper, 2005). Patients present with various degrees of digital flexure, especially on the medial side of the hand, an observation that has occasionally led to the erroneous assumption that the ulnar nerve is implicated (Clay, 1944). The condition particularly involves the metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints and is regarded as irreversible. Non-surgical attempts at correction are usually unsuccessful, though local injections of collagenase in the region of the cords are reported to be beneficial (Badalamente et al. 2002; de-Ary-Pires et al. 2007). Nevertheless, surgery remains the preferred option for patients who have more than 30° of MCP joint contracture (Townley et al. 2006). A palmar fasciotomy, fascietomy or even finger amputation may be advocated to alleviate the condition. The disease classically commences with thickening and pitting of the skin, followed by the development of fibrous nodules. These are firmly linked to the skin and eventually form cords (thickenings of the pretendinous fascia) which contract to flex the MCP and PIP joints and produce the characteristic deformity (Townley et al. 2006). It is a proliferative fascial affliction that has features in common with wound healing, the formation of keloid scars and fibrosarcomas (Jemec et al. 1999; Fitzgerald et al. 1999; Townley et al. 2006). Murrell et al. (1987) have argued that an increased free radical production could lead to a degree of tissue damage that subsequently triggers the reparative fibrosis that is characteristic of the contracture.

Dupuytren's disease has an interesting and colourful history (Elliot, 1988; Flatt, 2001). It is thought to have originated with the Vikings, has sometimes been compared to the 'hand of Benediction' or referred to as the 'curse of the MacCrimmon clan'. The last name stems from its reputation for having interfered with their bagpipe-playing skills! It is commonly bilateral and characteristically affects men over the age of 50 years, impinging significantly on daily work and home life (Townley et al. 2006; de-Ary-Pires et al. 2007). Sufferers may complain about difficulties in performing manual tasks, wearing gloves, shaking hands with another person, washing or dressing (Townley et al. 2006). As it often affects older individuals, it may be a threat to independent living. In view of the predominance of Dupuytren's disease in males, it is interesting that androgen receptors are more highly expressed in the palmar fascia of patients with the condition than in normal hands (Pagnotta et al. 2002).

The considerable clinical significance of Dupuytren's contracture has led to a comprehensive evaluation of the gene-expression profiles of cells in the palmar fascia and the composition of the fascial ECM. Predictably, many of the molecules characteristic of tendons and ligaments are present in fascia as well. As in tendons and ligaments, the predominant collagen is type I (Brickley-Parsons et al. 1981). Type III collagen is virtually absent in normal palmar fascia, but accumulates in patients with Dupuytren's disease (Brickley-Parsons et al. 1981; Melling et al. 1999). Murrell et al. (1991) suggest that this may be related to the high density of fibroblasts in Dupuytren fascia, for they mimicked the altered type III/I collagen balance both in cultured Dupuytren's fibroblasts and in control cells simply by increasing cell density. Similarly, there is increased expression of type IV collagen (together with fibronectin, laminin and tenascin) in the myofibroblast-rich areas of the palmar fascia from Dupuytren's patients (Berndt et al. 1994). Decorin (a small dermatan sulphate-rich proteoglycan, DSPG) is the principal small proteoglycan (PG), and biglycan and large chondroitin sulphate/DSPG are present as minor components (Kozma et al. 2005). Decorin and biglycan have also been reported in the fascia lata and large chondroitin sulphate/DSPG appears in scarred fascia (Kozma et al. 2000).

ECM composition changes substantially with pathology, not only in the palmar fascia in Dupuytren's contracture (reviewed by Cordova et al. 2005) but also in the fascia lata subject to scarring (Kozma et al. 2000). In both cases, there is an increase in the expression of biglycan relative to that of decorin (Kozma et al. 2000, 2005). This is indicative of overall changes in dermatan sulphate expression that may influence collagen–glycosaminoglycan interaction and thus fibrillogenesis (Kozma et al. 2007). An increase in gene expression for a wide variety of ECM components has been reported in microarray studies of the palmar fascia of patients with Dupuytren's disease (Qian et al. 2004). Of particular interest are numerous changes in mRNA levels reported by these authors that can be interpreted as attempts to deal with the fibrosis that is characteristic of the disease-defining tissue nodules. Thus, Qian et al. (2004) report a marked upregulation of a variety of genes involved in apoptosis, inflammation and proteolysis including the Alzheimer protein, amyloid A4 protein precursor (consistent with inflammatory reactions in general), pro-apoptotic Rho proteins (involved not only in apoptosis, but also in cell contractility and myofibroblast differentiation), several matrix metalloproteinases (MMPs – enzymes important for matrix turnover) and contactin (a gene involved in actin cytoskeleton remodelling related to myofibroblast activity). More recently still, Satish et al. (2008) have highlighted a downregulation of genes coding for proteoglycan 4 (PRG4), fibulin-1 (FBLN-1) transcript variant D, and collagen type XV alpha 1 chain, and Forsman et al. (2008) have reported a dramatic downregulation of myoglobin and an upregulation of tyrosine kinase-like orphan receptor 2 (ROR2). Augoff et al. (2006) have found an increased activity of MMP2 activity, and Johnston et al. (2007) have highlighted a marked increase in the expression of MMPs 1, 13, and 14 and of ADAMTS14 in Dupuytren nodules. They also found that TIMP1 expression is much higher in nodules compared with control tissue. Lee et al. (2006) have encouraged us to note the significant upregulation of MafB in view of the role of Maf proteins in tissue development and regulation. In immunohistochemical preparations, MafB co-localizes with actin stress fibres in Dupuytren myofibroblasts. As a generality, Rehman et al. (2008) point out that many of the genes that are dysregulated in tissue taken from Dupuytren cords showed an increase in fold change compared with tissue taken from nodules. Interestingly, they draw attention to a particular involvement of genes associated with cytoskeletal formation – clearly a pertinent issue in view of the myofibroblastic phenotype of many cells from Dupuytren-associated fascia. Hopefully, further gene-profiling studies will help to guide the development of agents that could be used for therapeutic or diagnostic purposes. However, at the moment, the gap between our scientific knowledge of the changes that characterize Dupuytren tissue and the clinical application of this knowledge remains considerable (Cordova et al. 2005). It is pertinent to note the observation that 29 of the dysregulated genes identified by Satish et al. (2008) in fascial fibroblasts from Dupuytren's tissue were of unknown function, making it likely that novel pathways are operating in the disease about which we are currently ignorant.

Dupuytren's disease shares with fibrosarcomas an elevated expression of the *c-myc* oncogene – a gene known to be associated with the control of cell proliferation, apoptosis and differentiation (Evan et al. 1994). The parallels with wound healing are particularly intriguing, especially the prominence of myofibroblasts and the possible role of these cells in tissue contraction (Bisson et al. 2003, 2004). According to Bisson et al. (2003), myofibroblasts are most common in Dupuytren nodules (where approximately 10% of the cells express alpha smooth muscle actin), less common in the cords (roughly 3%) and least common in normal flexor retinaculum sampled as a control (just over 1%). Intriguingly, Bisson et al. (2003) reported a marked increase in the number of myofibroblasts from both Dupuytren cords and nodules in response to TGF-β1 treatment compared with control fascia. The percentage of myofibroblasts increased to approximately 25% in both cords and nodules, whereas the cells of control fascia showed no response. The authors suggest that this may relate to the high recurrence rate of the disease or its progression following injury. It is also of interest that high levels of nerve growth factor have been associated with the proliferative stage of the disease, when myofibroblast number increases markedly (Lubahn et al. 2007). The expression of platelet-derived growth factor is also upregulated in Dupuytren's disease (Terek et al. 1995) and Augoff et al. (2005) has reported changes in the expression of epidermal growth factor.

Plantar fascia

The plantar fascia (synonym *plantar aponeurosis*) is a superficially placed sheet of dense connective tissue that covers and protects the intrinsic muscles of the foot (Fig. 9). It helps to maintain the medial longitudinal arch and transmit forces from the hind to the forefoot (Erdemir et al. 2004). It can act both as a beam and a truss (Hicks, 1954; Salathe et al. 1986) – a beam during propulsion when the metatarsals are subject to significant bending forces and a truss when the foot absorbs impact forces during landing and during the stance phase of gait. Its function as a tie-beam for the foot is particularly well explained by Sarrafian (1987). He likens the foot to a twisted plate in which the hindfoot lies in the sagittal plane and the forefoot horizontally. It is the twist that occurs between hind and forefoot that creates the characteristic arches. When the foot is loaded by body weight acting through the ankle joint, the dorsum of the foot experiences compressive loading and the plantar side is subject to tensile loading. The plantar fascia thus acts as a tie-beam in the sole to relieve the tensile loading to which this area is subject.

The plantar fascia stretches from the calcaneus to the distal aspect of the metatarsophalangeal (MTP) joints, where it divides into five slips – one for each toe. The slips pass distal to the MTP joints, blending with the volar plates, via which they are attached to the skeleton (Bojsen-Moller & Flagstad, 1976). However, they are also attached to the skin of the forefoot in this region. As the fascia inserts *distal* to the MTP joints, it is 'cranked' around the heads of the metatarsals as the body is raised onto the toes (Fig. 10). The metatarsals act as pulleys around which the fascia is tightened so that the medial longitudinal arch of the foot can be raised without muscular effort. The mechanism is commonly referred to as a 'windlass' (Hicks, 1954). In everyday usage, this is a cylindrical piece of equipment used to move heavy weights. Thus a windlass is effectively a barrel, rotated by turning a crank. It is commonly used by mariners to help raise heavy anchors. The tightening of the plantar fascia that occurs by this windlass mechanism at toe-off, increases the height of the medial longitudinal arch and helps to convert the foot into a rigid lever during weight bearing. As the metatarsals are firmly planted on the ground to support body weight at toe-off, the moveable end of the foot now becomes the heel. Consequently, because of the predominantly medial attachment of the plantar fascia to the calcaneus, it automatically inverts and supinates the foot, as it is tightened (Barthold, 2001).

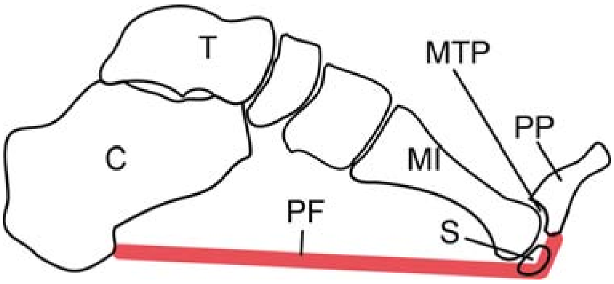

Fig. 10 A diagrammatic representation (modified from Fig. 4 of Hicks, 1954) to show the windlass mechanism by which the plantar fascia (PF) heightens the medial longitudinal arch of the foot. The fascia extends from the calcaneus (C) to beyond the level of the metatarsophalangeal joint (MTP), thus attaching to the proximal phalanx (PP) instead. Consequently as the foot is dorsiflexed, the fascia is tightened around the plantar surface of the MTP joint and the arch of the foot is heightened. MI, 1st metatarsal bone; S, sesamoid.

From a clinical perspective, the principal interest in this fascia lies in relation to plantar fasciitis (or fasciosis) – a common cause of heel pain that is well documented as an overuse injury and is particularly common in runners (Warren, 1990). Patients complain of tenderness and pain on the infero-medial aspect of the heel, i.e. at the site where the plantar fascia attaches to the medial tubercle of the calcaneus (Neufeld & Cerrato, 2008). It is commonly worse when arising from bed in the morning, or after periods of physical inactivity. It is frequently associated with biomechanical abnormalities of the foot, including over-pronation and a high medial longitudinal arch (Bolgla & Malone, 2004). Bony spurs may be present as well, though there is no clear cause–effect relationship between the presence of these spurs and heel pain. Furthermore, the spurs are rarely within the fascia itself, but lie instead on its deep surface (Kumai & Benjamin, 2002; Abreu et al. 2003). For this reason, Kumai & Benjamin (2002) have challenged the assumption that they are traction spurs and have suggested that they are more comparable to the peripheral osteophytes at the edge of articular cartilage in an osteoarthritic synovial joint.

Plantar fasciitis is normally treated conservatively, but surgery ('fasciotomy') is sometimes advocated for intransient cases. Among the possible consequences that are recognized by an invasive approach are a reduction in the stability of the medial longitudinal arch and an increased strain that can be placed on other foot ligaments in compensation (Cheung et al. 2004). Plantar fascial thickness is commonly determined sonographically and is known to be greater in patients with plantar fasciitis (Kane et al. 2001; Pascual Huerta & Alarcon Garcia, 2007) and in diabetics (Duffin et al. 2002). However, Pascual Huerta & Alarcon Garcia (2007) caution that the thickness of the plantar fascia varies regionally within a given individual and that the site where measurements are taken is not always clearly defined. Such methodological shortcomings mean that it is difficult to decide how thick a plantar fascia should be before it is viewed as pathologically thickened (Pascual Huerta & Alarcon Garcia, 2007). In diabetics, the thickening is probably a general sign of the hyperglycaemia that occurs in this condition, promoting glycosylation in tissues dense in collagen – hence in fascia (Barbagallo et al. 1993). Significantly, from a clinical perspective, the extent of plantar fascial thickening is thought to be a good predictor of complications known to arise from type I diabetes (Craig et al. 2008). In diabetics, thickening of the plantar fascia (and Achilles tendon) may increase the likelihood of foot ulcers because of the altered foot biomechanics that accompany such changes – particularly the formation of a cavus foot, i.e. a foot characterized by a high arch (Giacomozzi et al. 2005).

Thoracolumbar fascia

The thoracolumbar fascia (lumbar fascia or thoracodorsal fascia) is the deep fascia of the back. It lies in both the thoracic and lumbar regions of the trunk and covers the erector spinae complex. Its basic structure is dealt with in most general anatomy books (e.g. Standring, 2004) and more detailed accounts are available in the more specialized text of Vleeming et al. (2007). In the thoracic region, it forms a thin covering for the extensor muscles of the vertebral column. Medially, it is attached to the spines of the thoracic vertebrae, and laterally it is attached to the ribs, near their angles. In the lumbar region, it is also attached to the vertebral spines, but in addition it forms a strong aponeurosis that is connected laterally to the flat muscles of the abdominal wall. Medially, it splits into anterior, middle and posterior layers. The first two layers surround quadratus lumborum and the last two form a sheath for the erector spinae and multifidus muscles. Below, it is attached to the iliolumbar ligament, the iliac crest and the sacroiliac joint. Via its extensive attachment to vertebral spines, the thoracolumbar fascia is attached to the supraspinous and interspinous ligaments and to the capsule of the facet joints. Willard (2007) argues that this 'interspinous–supraspinous–thoracolumbar ligament complex' provides central support to the lumbar spine. It also transfers stress from a multitude of muscles to numerous facet joint capsules.

Particular interest is directed towards the posterior layer of the fascia, for this is important in transferring forces between spine, pelvis and lower limbs (Vleeming & Stoeckart, 2007). It is a superficially placed membrane that is a strong, glistening sheet of connective tissue, linking two of the largest muscles of the body – latissimus dorsi and gluteus maximus. According to Vleeming et al. (1995), it connects these muscles functionally in a way that promotes the co-ordinated activities of the upper and lower limb, notably the pendulum-like actions of the contralateral arms and legs during walking and running. As Vleeming & Stoeckart (2007) comment, the coupling between gluteus maximus and latissimus dorsi means that we need to be more cautious about categorizing a particular muscle as being an 'upper limb' or 'lower limb' muscle.

Although it has long been recognized that transversus abdominis and the internal oblique muscles are attached via the middle layer of the thoracolumbar fascia to the tips of the vertebral transverse processes, Barker et al. (2007) argue that the strength and significance of this attachment has been underestimated. Their dissections lead them to conclude that the middle layer of the thoracolumbar fascia has a sufficiently thick and strong attachment to the vertebral transverse processes that these processes could be avulsed by strong transversus abdominis contractions. Furthermore, they also suggest that the strength of the connections between the thoracolumbar fascia and the transversus abdominis muscles is such that the latter could be important in lumbar segmental control (Barker et al. 2004, 2007).

The fascia lata and the iliotibial tract

The fascia lata is the deep fascia of the thigh – a veritable stocking forming an ectoskeleton for its muscles. It is thicker where it is more exposed laterally than it is medially, and the thickening is called the iliotibial (IT) tract or band. The IT tract also serves as a tendon for tensor fascia latae and gluteus maximus. It has extensive attachments to the lateral intermuscular septum in the thigh (another fascia), is attached to the lower part of the femur and is finally anchored to Gerdy's tubercle at the upper end of the tibia (Fairclough et al. 2006). The IT tract is a common site of an overuse injury, well documented in runners and cyclists, located where the tract passes over the lateral femoral epicondyle (Fredericson & Wolf, 2005). Both the fascia lata in general and the IT tract in particular are used as sources of dense connective tissue for a wide variety of surgical procedures including sealing lung tissue (Molnar et al. 2003), correcting eyelid deformities (Flanagan & Campbell, 1981) and providing a suitable substitute for a torn anterior cruciate ligament (Joseph, 1988). The attraction of using the fascia as an autograft stems from the sparsity of its cell populations and thus its low nutritional requirements (Flanagan & Campbell, 1981).

IT tract syndrome is commonly attributed to excessive friction between the tract and the lateral femoral epicondyle just above the knee (Fredericson & Wolf, 2005), although Fairclough et al. (2006) argue that compressive forces between bone and fascia at this site are more significant. They point out that as the IT tract is part of a whole fascial stocking completely encircling the thigh, is extensively connected to the lateral intermuscular septum and constantly anchored to the lower part of the femur, it cannot move forward and backwards, as commonly surmised, during flexion and extension of the knee. They propose instead that the tract gives the illusion of a forward-backward translation, because of a shifting pattern of tension within its anterior and posterior fibres as the knee moves from full extension to 30° of flexion (at which angle, the symptoms are generally most acute). This is at odds with the views of Gerlach & Lierse (1990) that the IT tract is not part of the fascia lata and is a structure that glides in its own fascial bag. However, in a recent article, Vieira et al. (2007) also emphasize the numerous sites of bony connections of the IT tract, spanning from the hip to the tibia. The main thrust of Vieira et al.'s (2007) argument is that the IT tract has a structure in line with its importance as a lateral stabilizer or brace for the knee.

Concluding remarks

Fascia has long been of interest to surgeons and viewed with considerable importance by paramedical practitioners including manual therapists, osteopaths, chiropractors and physical therapists. 'Fascial release techniques' are routine working practices in such professions. However, despite the wealth and diversity of studies that have been reviewed in the current work, there is a paradoxical feeling that fascial research is still in its infancy, largely because it is not 'mainstream'. Yet it is likely to become so in the foreseeable future if it does indeed hold the key to understanding aspects of musculoskeletal problems such as low back pain and fibromyalgia. As a substantial number of visits by patients to primary care centres relate to musculoskeletal disorders, the importance of attracting further interest in fascia from the research community is obvious.

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PILOT STUDY

Treating patellar tendinopathy with Fascial Manipulation

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Summary According to Fascial Manipulation theory, patellar tendon pain is often due to uncoordinated quadriceps contraction caused by anomalous fascial tension in the thigh. Therefore, the focus of treatment is not the patellar tendon itself, but involves localizing the cause of this incoordination, considered to be within the muscular fascia of the thigh region.

Eighteen patients suffering from patellar tendon pain were treated with the Fascial Manipulation technique. Pain was assessed (in VAS) before (VAS 67.8/100) and after (VAS 26.5/100) treatment, plus a follow-up evaluation at 1 month (VAS 17.2/100).

Results showed a substantial decrease in pain immediately after treatment ($p<0.0001$) and remained unchanged or improved in the short term.

The results show that the patellar tendon may be only the zone of perceived pain and that interesting results can be obtained by treating the muscular fascia of the quadriceps muscle, whose alteration may cause motor incoordination and subsequent pathology.

Introduction

Patellar tendinopathy is a very frequent pathology among sportspeople, who perform multiple jumps in sports such as basketball, volleyball, and beach-volley, such that it is also called "jumper's knee". Patellar tendinopathy is also reported in subjects who often climb stairs, hike, and squat (Eifert-Mangine et al., 1992; Molnar and Fox, 1993; Morelli and Rowe, 2004). Indeed, the continuous repetition of certain movements, above all excessive strength training of the extensor compartment (quadriceps),

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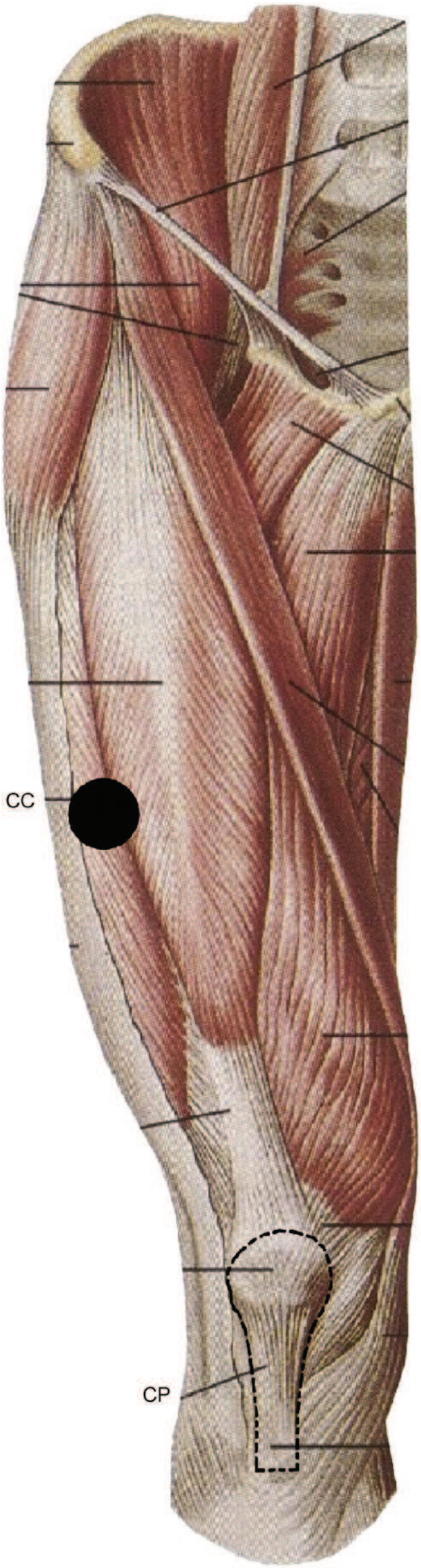
might cause various minor traumas and strains that are considered to be at the origin of this overuse syndrome. Symptoms, at first characterized by pain during strain, can evolve into inflammation (tendinitis) (Duri et al., 1999; Warden and Brukner, 2003) with pain at rest, and eventually lead to tissue alteration and, in some cases, even tendon rupture. The origin of these symptoms is commonly attributed to disease of the patellar tendon, and so the majority of known treatments focus directly on the tendon itself (Cyriax, 1982; Kountouris and Cook, 2007; Vulpiani et al., 2007; Willberg et al., 2007). In recent years, some authors (Simons et al., 1999; Stecco and Stecco, 2007) have begun to consider alterations of the patellar tendon as the consequence of chronic, uncoordinated movement of the knee joint due to incorrect activation of the knee extensor muscles. For this reason, many therapies, including quadriceps stretching, muscle energy techniques and tensional release through massage therapy (Chaitow, 2003), focus on the muscles of the extensor compartment. Similar principles can also be found in another manual technique, known as Fascial Manipulation (Stecco 1996, 2004), however, according to its theoretical model, continuous repetition of the same movement could cause "densification" of the muscular fascia, thereby altering the efficiency of muscle contraction. Other authors (Pellecchia et al., 1994; Rolf, 1997; Schleip, 2003) have also indicated that fascia is a plastic and malleable tissue, able to adjust to the mechanical, thermal and metabolic stresses, and can possibly be restored to its physiological condition through external manipulation treatment. Hence, the aim of this pilot study is to explore the effectiveness of Fascial Manipulation in alleviating the pain component in patellar tendinopathy and possible implications are also discussed.

In Fascial Manipulation, a map of over one hundred fascial points exists, that, when treated appropriately, are believed to restore tensional balance. In order to select the points to be treated the fascial system is first divided into basic elements, or myofascial units (MFUs). Each MFU includes all of the motor units responsible for moving a joint in a specific direction and the overlying muscular fascia. Hence, movements of single body segments are considered to be governed by six MFUs, responsible for movements in the three spatial planes (sagittal, frontal, horizontal). All the forces generated by a MFU are considered to converge in one point, called the centre of coordination (CC); each CC has a precise anatomical location within the muscular fascia. If the fascia in this specific area is altered, or "densified", then the entire MFU contracts in an anomalous manner resulting in non-physiological movement of the corresponding joint, which can be a cause of joint pain. According to the Fascial Manipulation model, the area where the patient perceives pain is called the centre of perception (CP), thus, for each MFU one CP is described. In patellar tendinopathy, the MFU of extension of the knee, called MFU of antemotion genu (AN-GE), is the more frequently implicated. It is formed by the knee joint, the monoarticular muscular fibres of vastus medialis, intermedius and lateralis, the biarticular muscular fibres of rectus femoris and the relative muscular fascia. The patella and the anterior region of the knee are considered as the CP of this MFU, while the CC is situated over the vastus intermedius muscle, halfway on the thigh (Figures 1 and 2). The location of this CC overlaps with the acupuncture point ST32 (Bossy et al., 1980), and with one of the trigger points of the quadriceps group, as described by Simons et al. (1999).

Figure 1 Deep fascia (fascia lata) of the thigh.



Materials and methods

Eighteen patients (13 males, 5 females; mean age 29.2), with unilateral sub-acute (from 1 to 3 months) or chronic (more than 3 months) patellar tendon pain (mean duration of symptoms 8.6 months) were treated according to the methodology of Fascial Manipulation (Table 1). Subjects with clinical signs of acute joint inflammation (oedema, heat, and rubor) were excluded from this study, as were subjects with meniscopathy and advanced degenerative osteoarthritis, as evidenced by MRI and X-rays. A complete physical examination of the knee was carried out, including inspection of the joint, assessment of the range of motion, muscle strength, and palpation.

Prior to commencing treatment, patients were asked to evaluate the severity of their pain, as experienced during two specific movement tests, on a VAS scale from 1 to 10 [10 = worst possible pain, 0 = no pain]. This subjective evaluation was repeated after one treatment session and the sessions were then suspended. At a follow-up, 1 month after treatment, a third measurement was recorded. The mean value of the VAS scale measurements was then calculated (Table 2) and the analysis of the differences in pain was accomplished by comparing the results obtained with appropriate statistical tests (Kurskal–Wallis test and Dunn's multiple comparison test as a control).

Treatment procedure: Movement tests, as indicated by Fascial Manipulation protocol, were performed before treatment to evaluate each MFU involved in movement of the knee joint (Figure 3). Results from these movement tests were scored according to Fascial Manipulation protocol, on a scale from 1 to 3 asterisks: pain = *, weakness = * and limited movement = * (Table 3). The CCs of the most dysfunctional MFUs (those with two or three asterisks) were then subjected to a comparative palpation assessment prior to selection of the points requiring treatment.

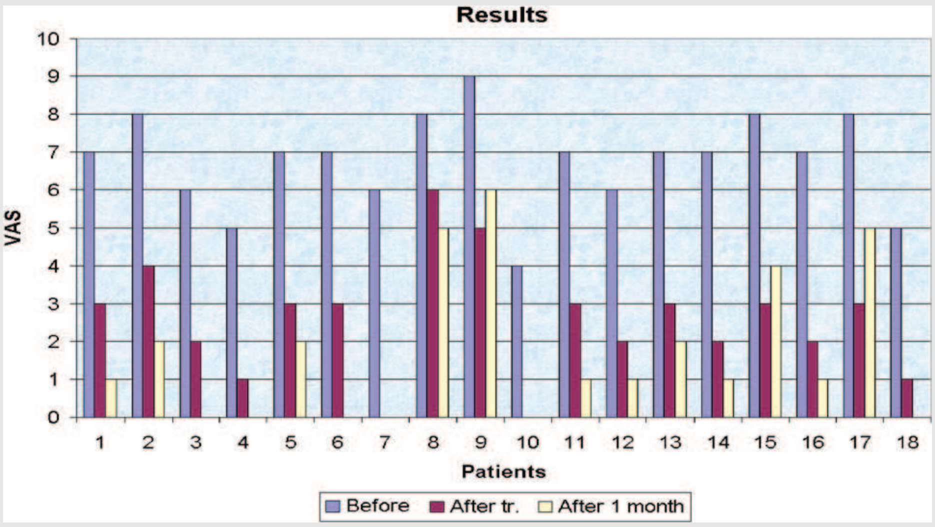
Two specific movement tests were also evaluated: going down a 30 cm high step, weight bearing on the suffering limb, and a flat-feet jump, starting from a position of total knee bending (squatting) (Figure 4). Subjective pain experienced during these two tests was assessed using the Vas scale measurement procedure.

Figure 2 Schematic representation of the centres of coordination (CC) and perception (CP). The CC is over the vastus intermedius muscle, and the CP is located over the anterior part of the knee.

Table 1 General characteristics of subjects.

Patient	Gender	Age	Duration of symptoms (months)	Sport
1	M	32	6	Basket
2	M	34	11	None
3	M	30	5	Soccer
4	F	17	1	Volley
5	M	37	10	None
6	F	35	2	Soccer
7	M	40	1	Bike
8	M	18	6	Basket
9	M	36	15	Running
10	M	19	4	Basket
11	M	33	24	None
12	F	31	5	Running
13	F	17	12	Basket
14	M	31	6	Beach-volley
15	M	42	18	None
16	F	17	3	Volley
17	M	38	24	Body build
18	M	19	3	Volley
Total	13 M, 5 F	Mean value 29.2	Mean value 8.67 months	/

Table 2 Results: VAS scale measurement of pain experienced during two specific movement tests (going down a 30 cm high step and flat-feet jump test) before treatment, immediately after treatment and 1 month after treatment.



An operator, other than the therapist who treated the patients, performed the movement tests and supervised the pain assessments.

A single therapist performed all treatments and the CC of AN-GE was treated in all cases. The treatment of this CC is performed with the patient supine, and the therapist standing to the side of the suffering limb. The therapist uses their elbow over the muscular fascia in the area between vastus lateralis and rectus femoris muscles, halfway on the thigh (Figure 5), applying pressure towards the vastus intermedius. Once the most altered area has

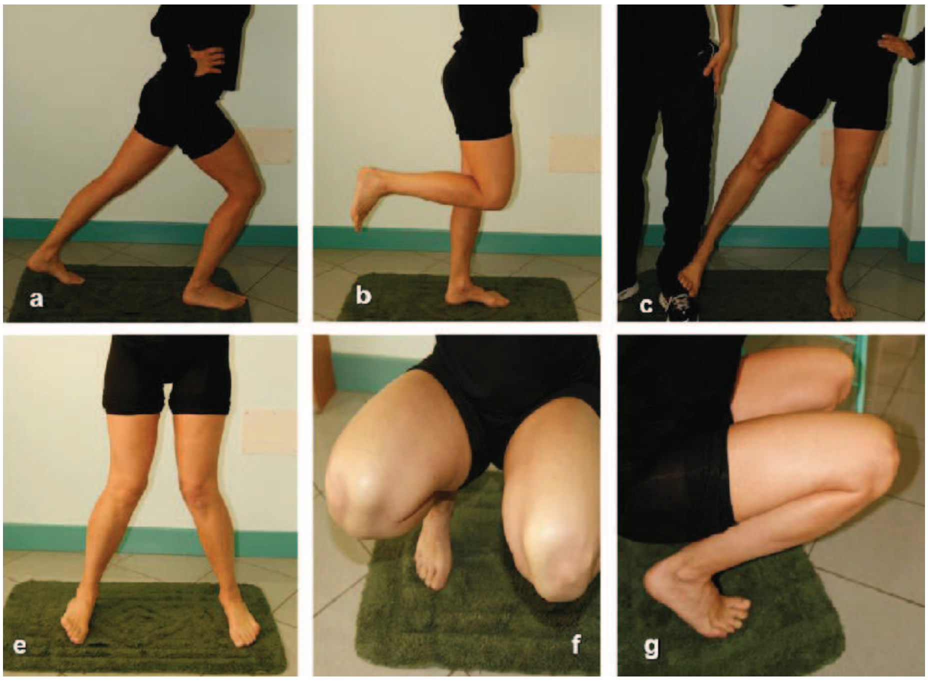


Figure 3 The motor tests for the knee segment according to Fascial Manipulation technique: (a) AN-GE test, (b) RE-GE test, (c) LA-GE test, (e) ME-GE test, (f) IR-GE test, and (g) ER-GE test.

Table 3 Evaluation of movements before treatment, according to fascial manipulation.

Patient	AN	RE	LA	ME	IR	ER
1	**	*	*			
2	***	*			*	
3	**					
4	*					
5	**	*				
6	**			*		
7	**					
8	***	*				*
9	***	**	*		*	
10	*				*	
11	**	*				
12	**					
13	**	*				
14	**					
15	***	**				
16	**			*		
17	***	*				
18	*					

been located, static pressure is initially applied and, subsequently, a deep friction or mobilization of the fascial tissues is employed. Verbal feedback from the patient aids in accurate localization of the exact point that provokes local and referred pain. Intermittent friction is maintained for a total of about 5 min, until the fascia glides freely and the patient refers that pain has decreased significantly.

All patients were asked to suspend sporting activities for at least 4 days after treatment, to avoid further stress on the treated structures.

At a 1-month follow-up, the two specific movement tests were re-assessed and VAS scale measurements were recorded.

Results

According to the results of the movement tests, all patients demonstrated a deficit (pain and/or weakness and/or limited movement) in the MFUs on the sagittal plane, with a specific involvement of the MFU of AN-GE. Pain assessments of the entire study group during the two specific movement tests before treatment (mean VAS 67.8/100) and after treatment (mean VAS 25.6/100) indicated a significant decrease of pain immediately after treatment ($p < 0.0001$) in all patients.

In particular, two cases (nos. 7 and 10) had a complete regression of pain immediately after treatment (mean VAS from 50/100 to 0/100) and this result was maintained at the 1-month follow-up. In another four patients (nos. 3, 4, 6, and 18), a good immediate post treatment result was

Figure 4 On the left: going down a 30 cm high step, weight bearing on the suffering limb; on the right: flat-feet jump test, starting from an angle of total knee bending.

Figure 5 Treatment position of the centre of coordination AN-GE according to the Fascial Manipulation technique.

recorded (mean VAS from 57.5/100 to 17.5/100) and, furthermore, at the follow-up, tendon pain had disappeared completely (VAS 0/100). At the follow-up, nine cases (nos. 1, 2, 5, 8, 11, 12, 13, 14, and 16) demonstrated a further reduction in pain as compared to immediately after treatment (mean VAS from 31.1/100 to 17.8/100). Only three patients (nos. 9, 15, and 17) referred that while pain had decreased immediately after treatment (mean VAS from 83.3/100 to 36.7/100) it had then increased again (to mean VAS 50/100), although not to the pre-session levels.

Discussion

According to this pilot study, it is evident that after one session of Fascial Manipulation a certain reduction of pain was recorded in every patient and that these results can be maintained or may partially regress. The aim of the Fascial Manipulation therapy is to restore gliding between the intrafascial fibres. Raising the temperature of selected areas of the fascia (corresponding to the CC points), via manual pressure, could allow for transformation of the ground substance, transforming it from a pathological status of GEL (dense fascia) to a physiological status of SOL (fluid fascia). This variation in density probably allows for two events. Firstly, during the application of manual pressure, the connective tissue adapts and the intrafascial free nerve endings may slide within the fascia more freely, which could explain the sudden decrease in pain during massage in the treated area. The second event could evolve over the following days: with enhanced fluidity of the ground substance, physiological tensioning of the fibres within the fascia during muscular contraction could allow for correct deposition of new collagen and elastic fibres according to the lines of applied force. Subsequent restoration of gliding between connective tissue layers of the fascia would enable tensional adjustments during muscular contraction, resulting in appropriate tensioning of periarticular structures such as tendons and capsules. This restitution of elasticity to the fascia could also explain the satisfactory results maintained over time.

In the Fascial Manipulation model, the CC is considered a point of vectorial convergence for muscular forces or the point of the muscular fascia where altered myofascial traction concentrates. Thus, for each segment, we can identify six CCs, one for each direction on the three planes of movement. A pathological CC can be pinpointed by a specific clinical exam (movement tests), and not only by palpation, which differs somewhat from the procedure for trigger point identification. Hence, a CC could be considered as a type of "key trigger point".

The myofascial connections within each MFU, and between different MFUs, can provide an alternative explanation for referred pain distribution (Stecco et al., 2007, 2008), which often does not follow either nerve pathways or the morphology of a single muscle (Hwang et al., 2005). When muscular fascia alters, it is feasible that the various motor units of the implicated muscles cannot coordinate their activity appropriately. Subsequent unaligned joint movement could cause non-physiological stretch of the receptors within the fascia, resulting in a nociceptive signal (Baldissera, 1996). In this way, according to Fascial Manipulation theory, when the CC is in an altered state it can be considered as the origin of pain (cause), and the joint (CP) as the area where pain is referred (consequence).

In those cases with partial resolution of symptoms, even though there had been some reduction in pain, indicating a correct interpretation of the problem, we hypothesize that more treatment sessions would have been necessary. In fact, the muscular fascia guarantees the anatomical and functional continuity of the anterior compartment muscles. In particular, the deep fascia of the ileopsoas continues with the fascia of the rectus femoris, and the fascia lata is continuous with the crural fascia that envelops the tibialis anterior muscle. Hence, if, for hypothesis, the fascia lata is chronically densified, then it is possible that alterations will occur in contiguous muscular fasciae in an attempt to compensate for this anomalous tension, with consequent alterations in the CCs of adjacent segments.

In those cases where we have recorded a re-intensification of pain after treatment, it should be pointed out that, as compared with other patients in the study group, they were far more complicated clinical cases. In fact, they did not only present tendinopathy, but case 9 also reported low back pain, case 15 Achilles tendinopathy and subcalcaneal pain and case 17 groin pain. Clinical cases such as these lead us to hypothesize a global disorder, or a postural imbalance involving numerous body segments. To make our study as consistent as possible, we decided to treat the same CC in all patients. In everyday practice, this CC has proven to be the most frequently involved point in this disorder. However, by treating one single CC, responsible for the imbalance of one single MFU, it cannot be enough to restore balance in similar global disorders.

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Fascial plasticity – a new neurobiological explanation: Part 1

Robert Schleip

In myofascial manipulation an immediate tissue release is often felt under the working hand. This amazing feature has traditionally been attributed to mechanical properties of the connective tissue. Yet studies have shown that either much stronger forces or longer durations would be required for a permanent viscoelastic deformation of fascia. Fascia nevertheless is densely innervated by mechanoreceptors which are responsive to manual pressure. Stimulation of these sensory receptors has been shown to lead to a lowering of sympathetic tonus as well as a change in local tissue viscosity. Additionally smooth muscle cells have been discovered in fascia, which seem to be involved in active fascial contractility. Fascia and the autonomic nervous system appear to be intimately connected. A change in attitude in myofascial practitioners from a mechanical perspective toward an inclusion of the self-regulatory dynamics of the nervous system is suggested.

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Introduction

Fascia – what a fascinating tissue! Also known as dense irregular connective tissue, this tissue surrounds and connects every muscle, even the tiniest myofibril, and every single organ of the body. It forms a true *continuity* throughout our whole body. Fascia has been shown to be an important element in our posture and movement organization. It is often referred to as our *organ of form* (Varela & Frenk 1987, Garfin et al. 1981).

Many approaches to manual therapy focus their treatment on the fascia. They claim to alter either the density, tonus, viscosity or arrangement of fascia through the application of manual pressure (Barnes 1990, Cantu & Grodin 1992, Chaitow 1980, Paoletti 1998, Rolf 1977, Ward 1993). Their theoretical explanations usually refer to the ability of fascia to adapt to physical stress. How the practitioner understands the nature of this particular responsiveness of fascia will of course influence the treatment. Unfortunately, fascia is often referred to in terms of its *mechanical* properties alone. This series of articles will not only explore the neural dynamics behind fascial plasticity, but will also offer new perspectives for myofascial treatment methods.

The classical gel-to-sol model

Many of the current training schools which focus on myofascial treatment have been profoundly influenced by Rolf (1977). In her own work Rolf applied considerable manual or elbow pressure to fascial sheets in order to change their density and arrangement. Rolf's own explanation was that connective tissue is a *colloidal substance* in which the ground substance can be influenced by the application of energy (heat or mechanical pressure) to change its aggregate form from a more dense '*gel*' state to a more fluid '*sol*' state. Typical examples of this are common gelatin or butter, which get softer by heating or mechanical pressure. This gel-to-sol transformation, also called *thixotropy* (Juhan 1987), has been positively confirmed to occur as a result of long-term mechanical stress applications to connective tissue (Twomey and Taylor 1982).

But the question arises: is this model also useful to explain the immediate *short-term* plasticity of fascia? In other words, what actually happens when a myofascial practitioner claims to feel a '*tissue release*' under the working hand? In most systems of myofascial manipulation, the duration of an individual 'stroke' or technique on a particular spot of tissue is between a few seconds and $1\frac{1}{2}$ minute. Rarely is a practitioner seen – or is it taught – to apply uninterrupted manual pressure for more than 2 minutes. Yet often the practitioners report feeling a palpable tissue release within a particular 'stroke'. Such rapid – i.e. below 2 minutes – tissue transformation appears to be more difficult to explain with the thixotropy model. As will be shown later, studies on the subject of '*time and force dependency*' of connective tissue plasticity (in terms of creep and stress relaxation) have shown that either much longer amounts of time or significantly more force are required for permanent deformation of dense connective tissues (Currier & Nelson 1992).

Additionally the problem of reversibility arises: in colloidal substances the thixotropic effect lasts only as long as the pressure or heat is applied. Within minutes the substance returns to its original gel state – just think of the butter in the kitchen. This is definitely not an attractive implication of this model for the practitioner.

Piezoelectricity – or the body as a liquid crystal

Oshman and others have added *piezoelectricity* as an intriguing explanation for fascial plasticity (Oshman 2000, Athenstaedt 1974). Piezo (i.e. pressure) electricity exists in crystals in which the electric centers of neutrality on the inside of the crystal lattice are temporarily separated via mechanical pressure from the outside and a small electric charge can be detected on the surface. Since connective tissue can be seen to behave like a 'liquid crystal' (Juhan 1987), these authors propose that the cells which produce and digest collagen fibers (called fibroblasts and fibroclasts) might be responsive to such electric charges. To put it simply: pressure from the outside creates a higher electric charge, which then stimulates the fibroblasts to increase their production rate of collagen fibers in that area. Additionally the fibroclasts might have a selective behavior not to 'eat' fibers which are electrically charged. In a nutshell: more stress, more charge, more fibers. Similar processes have already been shown to exist in bone formation after fractures as well as in wound healing.

Nevertheless, the processes involved seem to require time as an important factor. The half-life span of non-traumatized collagen has been shown to be 300–500 days, and the half-life of ground substance 1.7–7 days (Cantu & Grodin 1992). While it is definitely conceivable that the production of both materials could be influenced by piezoelectricity, both life cycles appear too slow to account for immediate tissue changes that are significant enough to be palpated by the working practitioner.

The traditional explanations are insufficient

Both models, thixotropy and piezoelectricity, are appealing concepts to explain long-term tissue changes. Yet it seems, additional models are needed when it comes to short-term plasticity. Laboratory studies on the subject of time and force dependency of connective tissue plasticity (in vitro as well as in vivo) have shown the following results: in order to achieve a permanent elongation of collagen fibers one needs to apply either an extremely forceful stretch of 3–8 percent fiber elongation, which will result in tissue tearing along with inflammation and other side effects which are usually seen as undesirable in a myofascial session. E.g. for an 18 mm distal iliotibial band such permanent elongation happens at 60 kg and more (Threlkeld 1992). Or it takes more than an hour (which can be taken at several intervals) with softer 1–1.5 percent fiber elongation, if one wants to achieve permanent deformation without tearing and inflammation (Currier & Nelson 1992, Threlkeld 1992).

For short-term application of stress the typical relationships are shown in Fig. 1. Microfailure is seen as the breaking of some individual collagen fibers and of some fiber bundles which results in a

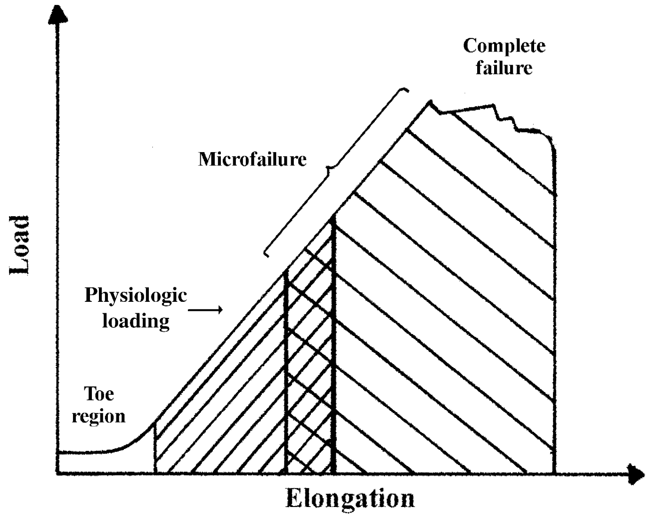

Fig. 1 Stress–strain curve of dense connective tissue. Most forces generated during daily life load the tissue in the linear region of the curve and produce non-permanent elongation. Microfailure with permanent elongation happens at extreme loads only and is accompanied by tearing and inflammation. The region of overlap of the microfailure zone with the physiologic loading zone varies with the density and composition of the tissue, yet for most fascial tissues it would be well above a 20 kg loading (drawing based on Threlkeld 1992). Figure by Twyla Weixl, Munich, Germany.

permanent (plastic) elongation of the tissue structure. This is followed by a cycle of tissue inflammation and repair. Based on measurements with different kinds of paraspinal tissues, Threlkeld calculates that microfailure occurs at around 224–1.136 N which equals 24–115 kg (Threlkeld 1992). While high-velocity thrust techniques might create forces within that range, it seems clear that the slower soft tissue manipulation techniques are hardly strong enough to create the described tissue response.

This research leads to a simple thought experiment. In everyday life the body is often exposed to pressure similar to the application of manual pressure in a myofascial treatment session. While the body naturally adapts structurally to long-term furniture use, it is impossible to conceive that adaptations could occur so rapidly that any uneven load distribution in sitting (e.g. while reading this article) would permanently alter the shape of your pelvis within a minute. It seems essential therefore that we find additional models – besides the thixotropic and piezoelectric concepts – to account for the palpable tissue changes that occur in a treatment session.

The need for a more rapid self-regulatory system

From an evolutionary perspective it makes sense that animals have a slowly adapting plasticity system in order to adjust to patterns of long-term use. In addition to this capacity they have also developed a more rapid system of adapting their form and local tissue density to temporary demands. This regulation system is open for adaptation to how the animal *perceives* its interaction with the environment. It seems logical that this ability of being more rapidly adaptable is mediated by – or at least connected to – a body system which is involved in the perception of our needs as well as of the environment. Traditionally, this body system has been called the nervous system.

It is therefore suggested that the self-regulatory qualities of the client's nervous system must be included in an explanatory model of the dynamics of fascial plasticity in myofascial manipulations. The author's own experiments in treating anesthetized people (with very similar results to that noted when manually treating very fresh pieces of animal meat) have shown that without a proper neural connection, the tissue does not respond as it does under normal circumstances (Schleip 1989).

Although it has not been considered very much in recent times, the inclusion of the nervous system in attempting to understand fascial responsiveness is not a new concept altogether, since the founder of osteopathy Andrew Taylor Still wrote more than a century ago.

> *The soul of man with all the streams of pure living water seems to dwell in the fascia of his body. When you deal with the fascia, you deal and do business with the branch offices of the brain, and under the general corporation law, the same as the brain itself, and why not treat it with the same degree of respect? (Still 1899).*

The nervous system as a wet tropical jungle

Many people think of the nervous system as an old-fashioned telephone switchboard system of the industrial age and therefore incapable of representing finer and more complex processes such as 'life energy', etc. The reader is cordially invited to consider this to be an outdated model. Current concepts in neurobiology see the brain more as a primarily *liquid system* in which fluid dynamics of a multitude of liquid and even gaseous neurotransmitters have come to the forefront. Transmission of impulses in our nervous system often happens via messenger substances that travel along neural pathways as well as through the blood, lymph, cerebrospinal fluid or ground substance (Kandel 1995). This global system for rapid body regulations is inseparably connected with the endocrinal and immune system. Rather than picturing the nervous system as a hard-wired electric cable system (which in the view of many bodyworkers is then of course incapable of being involved in more subtle energetic phenomena) picture it in your mind's eye as a *wet tropical jungle* (Schleip 2000). This jungle is a self-regulatory field with an amazing amount of complexity, continual reorganization and plasticity, even in adults.

The Golgi reflex arc as a breakthrough

Unfortunately, the precise details of the neural dynamics of fascia have rarely been explored. Cottingham (1985) presented a milestone proposal when he suggested a neurophysiological concept which was readily adopted by other authors (Ward 1993, Schleip 1989) and which will be briefly described here: Golgi receptors are said to be found all over in dense proper connective tissues. They exist in ligaments (here called *Golgi end organs*), in joint capsules, as well as around myotendinous junctions (here called *Golgi tendon organs*). These sensory receptors are arranged in series with fascial fibers and respond to slow stretch by influencing the alpha motor neurons via the spinal cord to lower their firing rate, i.e. to soften related muscle fibers. Cottingham suggested that during soft tissue manipulation – as well as in Hatha yoga postures and slow active stretching – these Golgi receptors are stimulated, which results in a lower firing rate of specific Alpha motor neurons, which then translates into a tonus decrease of the related tissues.

Too bad – it is not a simple reflex!

Unfortunately, later research has shown that passive stretching of a myofascial tissue does *not* stimulate the Golgi tendon organs (Jami 1992). Such a stimulation happens only when the muscle fibers are actively contracting. The reason for this lies in the arrangement of the Golgi tendon receptors. They are arranged in series with the muscle fibers. When the muscle with its related myofascia is passively elongated, most of the stretch will be taken up or 'swallowed' by a resulting elastic elongation of the muscle fibers. This is of course different in active client contractions, in which the Golgi tendon organs function to provide feedback information about dynamic force changes during the contraction (Lederman 1997).

But there are other Golgi receptors

Does this mean that deep tissue work (in which the client often is passive) will not involve the Golgi reflex loop? Perhaps, but not necessarily. These measurements have been done with passive joint extension movements, and not yet with the application of direct tissue pressure as in a myofascial manipulation.

Furthermore, it is important to note that only *less than 10%* of the Golgi receptors are found wholly within tendon. The remaining 90% are located in the muscular portions of myotendinous junctions, in the attachment transitions of aponeuroses, in capsules, as well as in ligaments of peripheral joints (Burke and Gandeva 1990).

Studies of the fine antigravity regulation in bipedal stance have also revealed a new functional role for Golgi receptors. In order to handle the extreme antigravity balancing challenges as a biped, our central nervous system can reset the Golgi tendon receptors and related reflex arcs so that they function as very delicate antigravity receptors (Dietz et al. 1992). This explains that some of the leg's balancing reactions in standing occur much quicker than it would take for a nerve impulse from the brain to the leg. In other words, the previously discussed and well-documented role of the Golgi organs (as a feedback mechanism about dynamic force changes during active contractions) covers only a minor functional role of these organs. For example, little is known about the sensitivity and related reflex function of those Golgi receptors that are located in ligaments (Chaitow 1980) or in joint capsules. It seems possible – yet also quite speculative – to assume that these less-explored Golgi receptors could indeed be stimulated with some stronger deep tissue techniques (Table 1).

And there are Ruffini and Pacini corpuscles

A detailed histochemical study of the thoracolumbar fascia at the Biomedical Engineering Institute of the Ecole Polytechnique in Montreal revealed that it is richly populated by *mechanoreceptors* (Yahia et al. 1992). The intrafascial receptors which they described consist of three groups. The first group are the large *Pacini* corpuscles plus the slightly smaller Paciniform corpuscles. The egg-shaped Pacini bodies respond to rapid changes in pressure (yet not to constant unchanging pressure) and to vibrations. A bit smaller are the *Paciniform* corpuscles, which have a similar function and sensitivity. A second group are the smaller and more longitudinal *Ruffini* organs which do not adapt as quickly and therefore respond also to long-term pressure. It seems likely that the Pacinian receptors are being

Table 1 Mechanoreceptors in fascia

Receptor type	Preferred location	Responsive to	Known results of stimulation
Golgi Type Ib	● Myotendinous junctions ● Attachment areas of aponeuroses ● Ligaments of peripheral joints ● Joint capsules	● *Golgi tendon organ*: to muscular contraction. ● *Other Golgi receptors*: probably to **strong stretch** only	**Tonus decrease** in related striated motor fibers
Pacini and Paciniform Type II	● Myotendinous junctions ● deep capsular layers ● spinal ligaments ● **investing muscular tissues**	**Rapid pressure changes** and vibrations	Used as proprioceptive feedback for movement control **(sense of kinesthesia)**
Ruffini Type II	● Ligaments of peripheral joints, ● Dura mater ● outer capsular layers ● and other **tissues associated with regular stretching**.	● Like Pacini, yet also to **sustained pressure**. ● Specially responsive to **tangential forces** (lateral stretch)	**Inhibition of sympathetic activity**
Interstitial Type III and IV	● **Most abundant receptor type**. Found almost everywhere, even inside bones ● Highest density in periosteum.	● Rapid as well as sustained pressure changes. ● 50% are **high-threshold** units, and 50% are **low-threshold** units	● Changes in **vasodilation** ● plus apparently in **plasma extra-vasation**

stimulated only by high-velocity thrust manipulations as well as in vibratory techniques, whereas the Ruffini endings will also be activated by slow and deep 'melting quality' soft tissue techniques.

Both types of intrafascial mechanoreceptors, the Pacinian/Paciniform and the Ruffini bodies, are found in all types of dense proper connective tissue, i.e. in muscle fascia, tendons, ligaments, aponeuroses, and joint capsules. In myotendinous junctions the Pacinian corpuscles are more frequent on the tendinous site (as opposed to the Golgi tendon organs which are more frequent on the muscular site). They have also been shown to be more frequent in the deeper portions of joint capsules, in deeper spinal ligaments, and *in investing* (or enveloping) muscular fasciae like the antebrachial, crural, abdominal fascia or the fascia of the masseter, the lateral thigh, in plantar as well as palmar tissues, and in the peritoneum (Stilwell 1957). The Ruffini endings are specially dense in tissues associated with regular *stretching* like the outer layer of joint capsules, the Dura mater, the ligaments of peripheral joints, and the deep dorsal fascia of the hand. At the knee joint the Ruffini endings are more frequent at anterior and posterior ligamentous and capsular structures, whereas Pacinian bodies are more accumulated medially and laterally of the joint (van den Berg & Capri 1999).

It is of interest to note that Ruffini endings are specially responsive to *tangential forces* and lateral stretch (Kruger 1987) and that stimulation of Ruffini corpuscles is assumed to result in a lowering of sympathetic nervous system activity (van den Berg & Capri 1999). This seems to fit to the common clinical finding that slow deep tissue techniques tend to have a relaxing effect on local tissues as well as on the whole organism.

Our reference scene

Figure 3 illustrates the neural tissue plasticity dynamics at this level. It is suggested that the following scene should be used as a reference point for this article. Imagine a practitioner working slowly with the connective tissue around the *lateral ankle*, in an area with no striated muscle fibers. (Choosing this reference scene allows us to focus on intrafascial dynamics only, and – for the purpose of this article – to ignore the stimulation of intramuscular mechanoreceptors and other effects which would be involved in the analysis of many other myofascial working situations.) If that practitioner reports a 'tissue release', what has happened? Possibly the manual touch stimulated some Ruffini endings which then triggered the central nervous system to change the tonus of some motor units in muscle tissue which is mechanically connected to the tissue under the practitioner's hand.

An unknown universe within us

In order to discuss the third group of intrafascial mechanoreceptors described by Yahia and her colleagues in Montreal, it is necessary to go on a short excursion. It commonly comes as a big surprise to many people to learn that our richest and *largest sensory organ* is not the eyes, ears, skin, or vestibular system but is in fact our muscles with their related fascia. Our central nervous system receives its greatest amount of sensory nerves from our myofascial tissues. Yet the majority of these sensory neurons are so small that until recently little has been known about them (Engeln 1994).

If one studies a typical muscle nerve (e.g. the tibial nerve), it consists of almost three times more sensory fibers than motor fibers. This points to a fascinating principle that sensory refinement seems to be much more important than the motor organization. However let us not get distracted by this. While many of the nerve fibers in a typical motor nerve have a vasomotor function, which regulate blood flow, the largest group of fibers are *sensory nerves*. Now comes the really interesting point: of these sensory nerves only a small fraction, or 20%, belong to the well-known types I and II nerves which originate in muscle spindles, Golgi organs, Pacini corpuscles and Ruffini endings (see Fig. 2). The majority, or four times as many, belong to an interesting group of *types III and IV* sensory nerves which are hardly mentioned in most textbooks (Mitchell & Schmidt 1977).

What do we know about this hidden network?

These hidden neurons are much smaller in diameter and are now commonly called *interstitial muscle receptors.* A better name would be *interstitial myofascial tissue receptors* since they also exist abundantly in fascia. A minority of these nerves are covered by a very thin myelin sheath (type III), but 90% of these nerves are unmyelinated (type IV). These interstitial receptors are slower than the types I and II nerves and most of them originate in *free nerve endings*.

In the past it was assumed that these nerve endings are mostly pain receptors. Some have also been shown to be involved in thermo- or chemoception. While many of these receptors are multimodal, research has shown that the majority of these interstitial receptors do in fact function as *mechanoreceptors*, which means they respond to mechanical tension and/or pressure (Mitchell & Schmitt 1977).

This large group of interstitial mechanoreceptors can be further divided into two subgroups of equal size: low-threshold pressure units (LTP units) and high-threshold units (HTP). A study of the Achilles tendon of cats revealed that about half of types III and IV endings encountered were LTP units and responded to light touch, even to touch as light as "*with a painter's brush*" (Mitchell & Schmidt 1977). Based on this latter finding, does it not seem possible – indeed likely – that soft tissue manipulation might involve stimulation of types III and IV receptors?

Recent insights into the physiology of pain have shown that several interstitial tissue receptors function both as mechanoreceptors (usually as HPT units) and as pain receptors. In the presence of pain – and the support of various neuropeptides – their sensitivity changes such that normal physiological pressure changes often lead to strong and chronic firing of these receptors. This explains why current research has revealed that pain often exists without any mechanical irritation of nervous structures as was frequently assumed by the root-compression model (Chaitow & DeLany 2000).

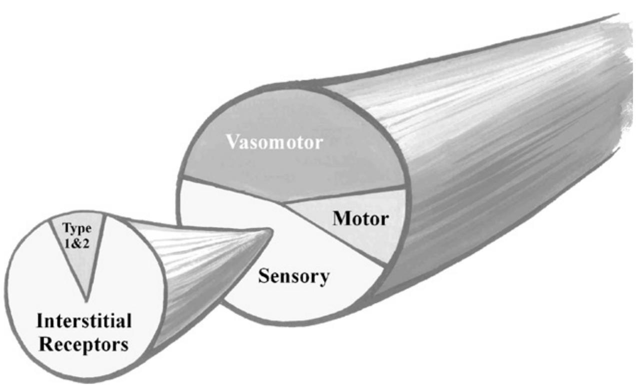

Fig. 2 Within a typical muscle nerve there are almost three times as many sensory neurons than motor neurons. Note that only a small portion of the sensory information comes from types I and II afferents which originate in muscle spindles, Golgi receptors, Pacinian and Ruffini endings. The majority of the sensory input comes from the group of types III and IV afferents or interstitial receptors which are intimately linked with the autonomic nervous system. Figure by Twyla Weixl, Munich, Germany.

What are they doing?

This of course triggers the question about the natural functional role of interstitial mechanoreceptors in the body. What regular consequences or reactions have been associated with an excitation of this hidden and rich sensory network? Of course some of them function as pain receptors. By 1974 a Japanese study had already revealed that types III and IV receptors in the fascia of temporalis, masseter and infrahyoid muscles show '*responses to the mandibular movement and the stretching of the fascia and the skin*', and it was therefore suggested that these nerve endings are concerned '*with the sensation of position and movement of the mandible*' (Sakada 1974).

Furthermore the majority of these types III and IV mechanoreceptors have been shown to have *autonomic functions*, i.e. stimulation of their sensory endings leads to a change in heart rate, blood pressure, respiration, etc. Stimulation of type IV receptors tends to increase arterial blood pressure (Coote & Pérez-Gonzáles 1970) whereas stimulation of type III receptors can both increase and decrease blood pressure. Several studies have shown that an increase of static pressure on muscles tends to lower arterial blood pressure (Mitchell & Schmitt 1977). It seems that a major function of this intricate network of interstitial tissue receptors is to fine tune the nervous system's regulation of blood flow according to local demands, and that this is done via very close connections with the autonomic nervous system.

Touch research with cats and humans

Based on this research it should not come as a surprise that slow deep pressure on the soft tissue of cats has been shown to lead to a reduction in muscle tonus measured by EMG activity (Johansson 1962) and that slow stroking of the back in cats produces a reduction in skin temperature as well as signs of inhibition of the gamma motor system (von Euler & Soderberg 1958).

Furthermore, it has been proven that deep mechanical pressure to the human *abdominal region* (Folkow 1962), as well as sustained pressure to the *pelvis* (Koizumi & Brooks 1972), produces parasympathetic reflex responses, including synchronous cortical EEG patterns, increased activity in vagal fibers, and a decreased EMG activity.

According to the model of hypothalamic tuning states by Ernst Gellhorn, an increase in vagal tone does not only trigger changes in the autonomic nervous system and related inner organs, but also tends to activate the anterior lobe of the hypothalamus. Such a '*trophotropic tuning*' of the hypothalamus then induces a lower overall muscle tonus, more quiet emotional activity, and an increase in synchronous cortical activity, both in cats as well as in humans (Gellhorn 1967). It therefore appears that deep manual pressure – specifically if it is slow or steady – stimulates interstitial and Ruffini mechanoreceptors, which results in an increase of vagal activity, which then changes not only local fluid dynamics and tissue metabolism, but also results in global muscle relaxation, as well as a more peaceful mind and less emotional arousal.

On the other hand, sudden deep tactile pressure or pinching or other types of strong and rapid manipulations have been shown to induce a general contraction of skeletal muscles (Eble 1960), particularly of 'genetic flexor muscles' (Schleip 1993) which are innervated via a ventral primary ramus from the spinal cord.

Talking to the belly brain

Mechanoreceptors have been found abundantly in visceral ligaments as well as in the Dura mater of the spinal cord and cranium. It seems quite plausible that most of the effects of visceral or craniosacral osteopathy could be sufficiently explained by a simulation of mechanoreceptors with resulting profound autonomic changes, and might therefore not need to rely on more *esoteric* assumptions (Arbuckle 1994).

Recent discoveries concerning the richness of the *enteric nervous system* (Gershon 1999) have taught us that our 'belly brain' contains more than 100 million neurons and works largely independent of the cortical brain. It is interesting to note that the very small connection between these two brains of a few thousand neurons consists of nine times as many neurons involved in processes in which the lower brain tells the upper one what to do, compared with the number of neurons involved in the top-down direction. Many of the sensory neurons of the enteric brain are mechanoreceptors, which – if activated – trigger among other responses, important *neuroendocrine* changes. These include a change in the production of *serotonin* – an important cortical neurotransmitter 90% of which is created in the belly – as well as other neuropeptides, such as *histamine* (which increases inflammatory processes).

What are we doing?

Myofascial manipulation involves a stimulation of intrafascial mechanoreceptors. Their stimulation leads to an altered proprioceptive input to the central nervous system, which then results in a changed tonus regulation of motor units associated with this tissue (Fig. 3). In the case of a slow deep pressure, the related mechanoreceptors are most likely the slowly adapting Ruffini endings and some of the interstitial receptors; yet other receptors might be involved too (e.g. spindle receptors in affected muscle fibers nearby and possibly some intrafascial Golgi receptors).

Measurements on the mechanoreceptors of the knee joint ligaments have shown that their stimulation leads to weak effects in alpha motor neurons, yet to powerful changes in gamma motor neurons. This means that these ligamentous mechanoreceptors are probably used as proprioceptive feedback for preparatory regulation (preprogramming) of muscle tonus around this joint (Johansson et al. 1991). For myofascial practitioners this is fascinating news, as it suggests

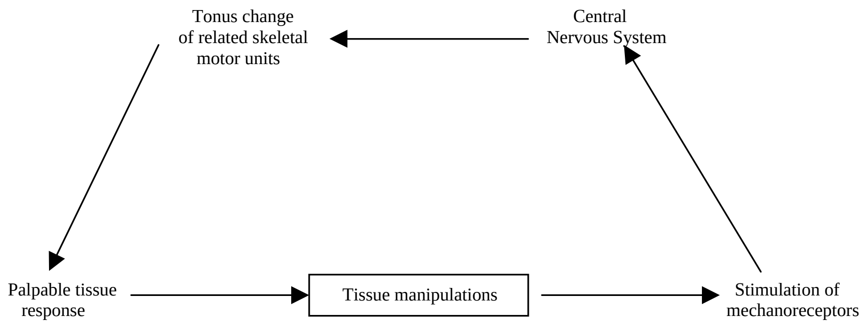

Fig. 3 The 'Central Nervous System Loop' (inspired by Cottingham). Stimulation of mechanoreceptors leads to a lowered tonus of skeletal motor units which are mechanically linked with the tissue under the practitioner's hand. The involved intrafascial mechanoreceptors are most likely Ruffini endings, Pacinian corpuscles (with more rapid manipulations), some of the interstitial receptors, plus possibly some intrafascial Golgi receptors.

that simulation of fascial mechanoreceptors may primarily lead to changes in gamma motor tone regulation. While the alpha and gamma motor system are usually coactivated, there are some important differences between them. The alpha system originates primarily in the cortex, and it is particularly involved in volitional and precise movements of the extremities, whereas the gamma system originates in older brain stem structures and plays a strong role in the more global and unconscious postural organization of antigravity-extensor muscles and chronic musculo-emotional attitudes (Glaser 1980, Henatsch 1976, Juhan 1987).

No muscle is a functional unit

When discussing any changes in motor organization, it is important to realize that the central nervous system does not operate 'in muscles', i.e. a muscle is never activated as a whole. The functional units of the motor system are the so-called *motor units*, of which we have several million in our body, much like a school of fish that have learned to swim together. Depending on the quality of sensory feedback, these millions of motor units can be individually regulated (Basmajian & De Luca 1985). We can now apply this understanding to our reference scene, in which a practitioner is working on the connective tissue around the lateral ankle. When the practitioner reports a tissue release, it may be that it is caused by a lowered firing rate of only a few fish (motor units) in the vicinity, and that this movement is transmitted to the tissue under the practitioner's hand. If the practitioner then feels the change and responds in a supportive way toward these particular fish, other fish may soon follow the new direction, which of course leads to additional 'release sensations' for the practitioner, and so on (Fig. 4).

Conclusion

Immediate fascial plasticity cannot be understood by mechanical properties alone. Fascia is densely innervated by mechanoreceptors. Manual stimulation of these sensory endings probably leads to tonus changes in motor units which are mechanically linked to the tissue under the practitioner's hand. At least some of these responses are primarily regulated by a change in gamma motor tone, rather than in the more volitional alpha motor system. Of particular interest are the Ruffini organs (with their high responsiveness to tangential pressure) and the very rich network of interstitial receptors, since stimulation of both of these receptors can trigger profound changes in the autonomic nervous system. Part 2 of this article series

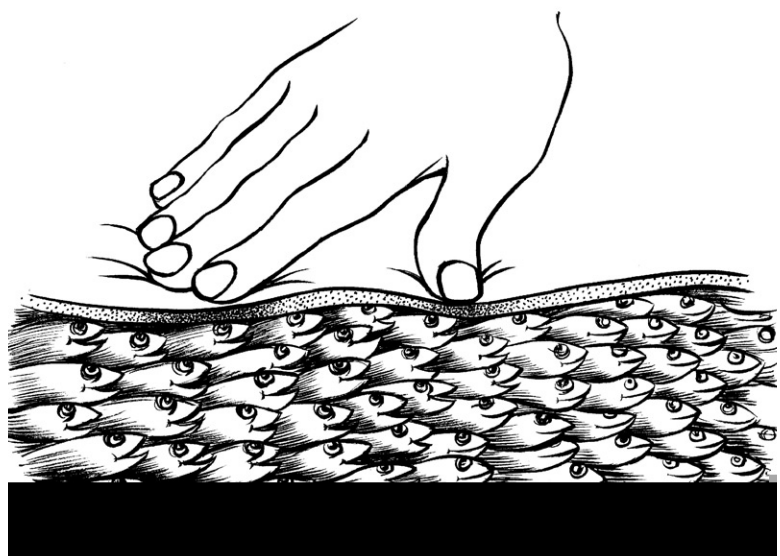

Fig. 4 Myofascial tissue as a school of fish. A practitioner working with myofascial tissue may feel several of the motor units responding to touch. If the practitioner then responds supportively to their new behavior, the working hand will soon feel other fish joining, and so forth. Figure by Twyla Weixl, Munich, Germany.

will include the discovery and function of intrafascial smooth muscle cells. It will show how fascial mechanoreceptors can trigger immediate viscosity changes of the ground substance, and how fibromyalgia might be related to all that. Several practical applications for the practitioner will be given.

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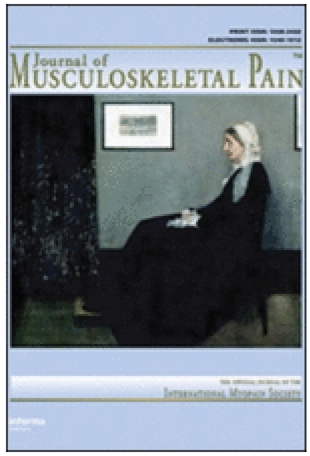

Biomechanical Properties of Fascial Tissues and Their Role as Pain Generators

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Manuscript Type:	Review
Keywords:	myofibroblasts, fascial tonicity, delayed onset muscle soreness (DOMS), fascial innervation, micro tearing

Biomechanical Properties of Fascial Tissues and Their Role as Pain Generators.

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Key words:

Myofibroblasts, fascial tonicity, delayed onset muscle soreness (DOMS), fascial innervation, micro tearing

Objectives

While fasciae have virtually been treated as the 'cinderella tissue of orthopedic research' during recent decades, new methodological findings and hypotheses suggest that the bodywide fascial network may play a more important role in musculoskeletal medicine than commonly assumed. However, there is great diversity in the literature, as to which tissues are included under the term 'fascia' – be it the superficial fascia, the endomysium, perineurium, visceral membranes, aponeuroses, retinaculae or joint/organ capsules. Following the proposed comprehensive terminology of the 1st Fascia Research Congress this brief review considers all collagenous connective tissues as 'fascial tissues' whose morphology is dominantly shaped by tensional loading and which can be seen to be part of an interconnected tensional network throughout the whole body (1). While morphological differences between aponeuroses and lattice-like or irregular fasciae can still be properly described with this terminology, it allows to see tissue specifications -

such as septae, capsules, or ligaments - as local adaptations of this ubiquitous network based on specific loading histories.

What are the biomechanical functions of this fascial network, and what role do they play in musculoskeletal dysfunctions? This brief review will highlight the load bearing function of different fascial tissues and also their proneness to micro tearing during physiological or excessive loading. It will review histological studies indicating a proprioceptive as well as nociceptive innervation of fascia. Finally the potential role of injury, inflammation and/or neural sensitization of the posterior layer of the human lumbar fascia in non specific low back pain will be explored.

Findings

While a tensional load bearing function of tendons and ligaments has never been disputed, recent publications from Huijing et al. revealed that muscles also transmit a significant portion of their force via their epimysia to laterally positioned tissues such a to synergistic or antagonistic muscles (2). The reported contribution of M. transversus abdominis to dynamic lumbar spinal stability has been associated with the load bearing function of the middle layer of the lumbar fasciae in humans (3). Similarly, EMG based measurements of the 'flexion-relaxation phenomenon' suggest a strong tensional load bearing function of dorsal fascial tissues during healthy forward bending of the human trunk [with a reported absence of such load shifting in low back pain patients] (4).

Recent ultrasound based measurements indicate that fascial tissues are commonly used as elastic springs [catapult action] during oscillatory movements such as walking, hopping or running, in which the supporting skeletal muscles contract rather isometrically (5).

Fascial tissues are prone to viscoelastic deformations such as creep, hysteresis and relaxation. Such temporary deformations alter fascial stiffness and may take up to several hours for complete recovery. Load bearing tests also reveal the existence of a gradual transition zone between reversible viscoelastic deformation and complete tissue tearing. Various degrees of micro tearing of collagenous fibers and their interconnections have been documented to occur within this zone (6).

Fascia is densely innervated by myelinated nerve endings which are assumed to serve a proprioceptive function. These include Pacini (and paciniform) corpuscles, Golgi tendon organs, and Ruffini endings (7). In addition they are innervated by free endings. Newer histological examinations have shown that at least some of those free nerve endings are substance P containing receptors which are commonly assumed to be nociceptive (8). Delayed onset muscle soreness (DOMS) can be induced by repetitive eccentric contraction. A recent experimental study suggests that the epimysial fascia plays a major role in the generation of related pain symptoms (9).

Panjabi's new explanatory model of low back pain injuries suggests that single trauma or cumulative microtrauma causes subfailure injuries of dorsal fascial tissues and their embedded mechanoreceptors, thereby leading to corrupted mechanoreceptor feedback and resulting in further connective tissue alterations and neural adaptations. (10).

Langevin reports that the posterior layer of the lumbar fascia tends to be thicker in chronic low back pain patients and also to express less shear motion during passive trunk flexion (11). In addition our group has shown a high density of myofibroblasts, whose existence is usually associated with excessive loading or injury repair – in the same fascial layer (12). Surgical examinations by Bednar and Dittrich report the finding of frequent signs of injury and inflammation in the lumbar fascia of low back pain patients (13,14). And finally, injection of an inflammatory agent into the rat lumbar back muscles resulted in a dramatic increase of the proportion of dorsal horn neurons with input from the superficial lumbar fascia (15).

Conclusions

Fascial tissues serve important load bearing functions. Severe tensional loading can induce temporary viscoelastic deformation and even micro tearing. The innervation of fascia indicates a potential nociceptive function. Micro tearing and/or inflammation of fascia can be a direct source of musculoskeletal pain. In addition, fascia may be an indirect source of e.g. back pain, due to a sensitization of fascial nerve endings associated with inflammatory processes in other tissues within the same segment.

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