Fascial mechanoreceptors and their potential role in deep tissue manipulation

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Table 1:

Introduction

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 Golgi reflex arc as a breakthrough

Unfortunately, the precise details of the neural dynamics of fascia have rarely been explored. Cottingham presented a milestone proposal when he suggested a neurophysiological concept (Cottingham 1985) 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 & Gandeva 1990).

Studies of the fine antigravity regulation in bipedal stance have also revealed a new functional role of 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 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.

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 3 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 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

Fig 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 1993).

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 type 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 *type III & IV* sensory nerves which are hardly mentioned in most textbooks (Mitchell & Schmidt 1977).

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 type 1 &2 afferents which originate in muscle spindles, Golgi receptors, Pacinian and Ruffini endings. The majority of the sensory input comes from the group of type 3&4 afferents or Interstitial Receptors which are intimately linked with the Autonomic Nervous System.

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 type I & 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 the type III & 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, doesn't it seems possible – if not likely - that soft tissue manipulation might involve stimulation of these type III & 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 painreceptors. 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).

What are they doing?

This triggers of course 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. Yet already in 1974 a Japanese study revealed, that the type 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 type III & 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 JH & Pérez-Gonzáles 1970) whereas stimulation of type III receptors can both increase or 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 and 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 flexors 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 independently of the cortical brain. It is interesting to note that the very small connection between these two brains of a few thousands neurons consists of nine times as many neurons involved in processes in which the lower brains 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 of which 90% is created in the belly – as well as other neuropeptides, such as the substance *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. Which means that these ligamentous mechanoreceptors are probably used as proprioceptive feedback for preparatory regulation (preprogramming) of muscle tonus around this joint (Johansson 1991). For myofascial practitioners this is fascinating news, as it suggests 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 musculoemotional 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 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.

Fig4

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

Mechanoreceptors influence local fluid dynamics

Let's now look at some of the other effects of myofascial work. It is the large group of interstitial receptors that make up the majority of sensory input from myofascial tissue. Their activation triggers the autonomic nervous system to change the local pressure in fascial arterioles and capillaries. Additionally stimulation of Ruffini endings appears to have a similar effect in terms of a lowering of sympathetic activity (van den Berg & Capri 1999).

According to Kruger many of the interstitial fibers – if strongly stimulated - can apparently also influence *plasma extravasation*, i.e. the extrusion of plasma from blood vessels into the interstitial fluid matrix (Kruger 1987). Such a change of local fluid dynamics means a change in the viscosity of the extracellular matrix. This harks back to Ida Rolf's originally proposed gel-to-sol concept (Rolf 1977), yet this time with the inclusion of the client's nervous system. It also supports the assumption of Mark F Barnes, that myofascial manipulation might involve a change of the *system of ground regulation*, which according to Pischinger is defined as a functional unit of final vascular pathways, connective tissue cells and final vegetative neurons (Barnes 1997, Pischinger 1991). With an increased renewal speed in the ground substance it also appears more likely that the piezoelectric model which was explored in part 1 might play a role in immediate tissue plasticity.

Fig. 1 The 'Intrafascial Circulation Loop' (based on Mitchell & Schmid 1977). Fascia is densely innervated by interstitial tissue receptors. The autonomic nervous system uses their input (plus that of some Ruffini endings) to regulate local fluid dynamics in terms of an altered blood pressure in local arterioles and capillaries plus in plasma extravasation and local tissue viscosity. This change might then be felt by the hand of a sensitive practitioner.

If myofascial manipulation affects both the local blood supply as well as local tissue viscosity, it is quite conceivable that these tissue changes could be rapid and significant enough to be felt by the listening hand of a sensitive practitioners. This first autonomic feedback loop – here called *Intrafascial Circulation Loop'* - is based on the work of Mitchell & Schmidt (1977) and is illustrated in Fig. 1

Changes in hypothalamic tuning

And there is a second autonomic feedback loop. The interstitial mechanoreceptors can trigger an increase in *vagal tone* which leads towards more *trophotropic* tuning of the hypothalamus. Based on Gellhorn (Gellhorn 1967) this results in global neuromuscular, emotional, cortical and endocrinal changes that are associated with deep and healthy relaxation (see the paragraph '*Touch research with cats and humans'* in part 1). This *Hypothalamus- Loop-* is illustrated in Fig 2.

Fig.2…The 'Hypothalamus-Loop' based on Gellhorn.

Note that slow deep pressure usually leads to a more parasympathetic state. This activates the more trophotropic anterior lobe of the hypothalamus to lower the overall tonus of the body musculature.

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