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.

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.

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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

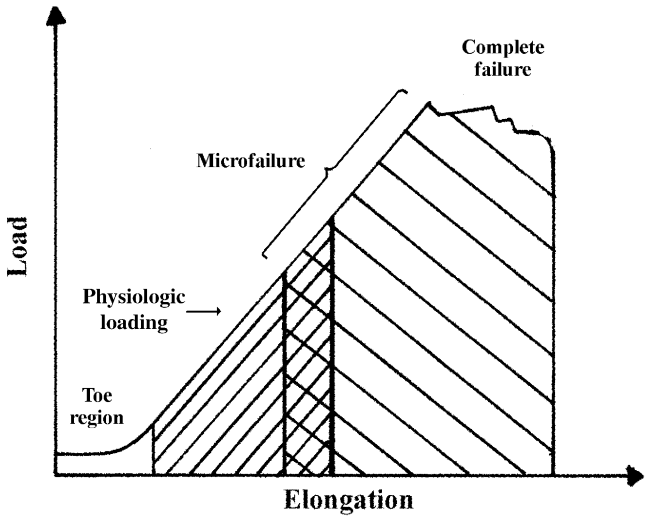

Fig. 1 Stress–strain curve of dense connective tissue. Most forces generated during daily life load the tissue in the linear region of the curve and produce non-permanent elongation. Microfailure with permanent elongation happens at extreme loads only and is accompanied by tearing and inflammation. The 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).

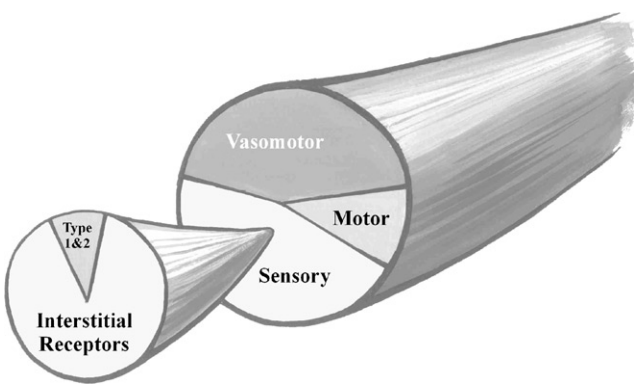

Fig. 2 Within a typical muscle nerve there are almost three times as many sensory neurons than motor neurons. Note that only a small portion of the sensory information comes from types I and II afferents which originate in muscle spindles, Golgi receptors, Pacinian and Ruffini endings. The majority of 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

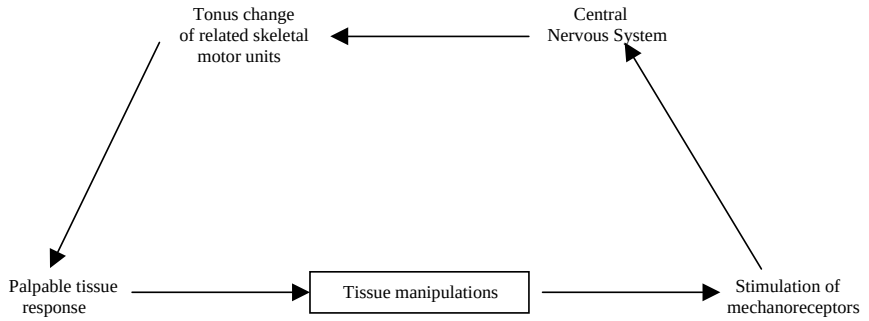

Fig. 3 The 'Central Nervous System Loop' (inspired by Cottingham). Stimulation of mechanoreceptors leads to a lowered tonus of skeletal motor units which are mechanically linked with the tissue under the practitioner's hand. The involved intrafascial mechanoreceptors are most likely Ruffini endings, 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

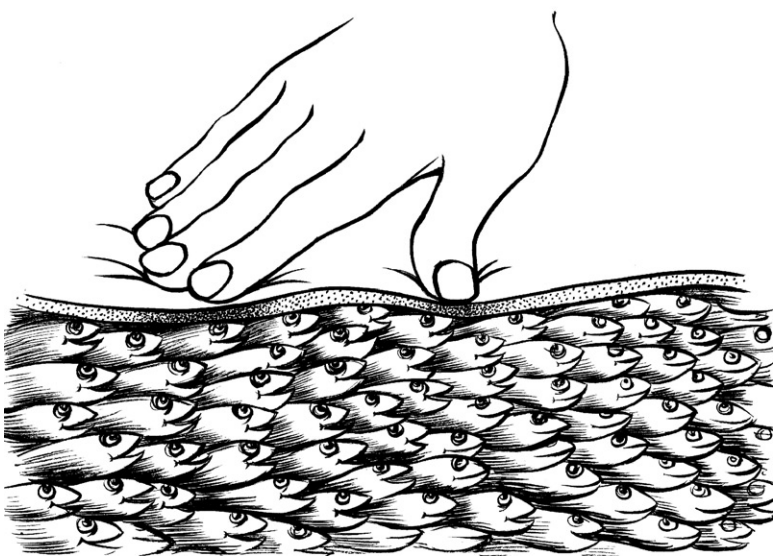

Fig. 4 Myofascial tissue as a school of fish. A practitioner working with myofascial tissue may feel several of the motor units responding to touch. If the practitioner then responds supportively to their new behavior, the working hand will soon feel other fish joining, and so forth. Figure by Twyla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