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## FASCIA SCIENCE AND CLINICAL APPLICATIONS: EDITORIAL

# A fascia and the fascial system



One of the main topics of the last Fascia Research Congress (Washington, 17–21 September 2015) was the terminology about fascia. Many researchers are convinced that the indiscriminate use of the term “fascia” in reference to various types of connective tissue often leads to confusion. Furthermore, inconsistent use of anatomical terms makes it difficult to compare results across research studies and to draw generalized conclusions (Langevin, 2014). This situation may be comparable to a time in anatomy history described by Adstrum (2014): “more than 50,000 terms were used to identify 5000 structures, so, anatomical terminology was in a state of chaos, incoherent, full of inequities, contradictions, and obscurities”. This striving for more clarity in terms of terminology resonates with a comparable attempt among researchers to describe manual therapies. In 2013, Paul Standley called for the establishment of a unified set of terms to enhance the construction of an evidence base to describe clinical efficacy of the various manual treatments. Indeed, he observed that most manual methods involve combinations of stretch, compression, shear and torque forces, and that “despite being called different names, many of these techniques used around the world really create the same (or nearly the same) effects on tissues and cells”.

One year ago a group was created by the Fascia Research Society to move towards a common definition of fascia and to clarify which anatomical structures could be included in this definition. Authors that had contributed to the ongoing terminology discussion in the Journal of Bodywork and Movement Therapies were invited to participate, as well as a representative of each category: osteopathic, chiropractic, medical doctor, basic science researchers, and movement practitioners. In order to converge towards a consistent terminology, we proposed adopting the Delphi Method in December 2014. This is a transparent and streamlined process for fostering a statement of consensus among a wide array of experts in a specific field and applying the methodology in other fields as well. During this process, it became evident that the choice of most suitable nomenclature depends on the type of investigation or perception of fascia. If the focus of the investigation is morphological, then a narrower fascia definition tends to

work best, such as the one proposed by FCAT (Federative Committee on Anatomical Terminology). If one intends to investigate functional aspects – such as force transmission or sensory capacities – then a wider definition of fascia tends to be more helpful. Both of these perspectives and related definitions are associated with major advantages which make the respective terminology superior when applying it within a specific context.

The Delphi process culminated in a personal committee meeting prior to the last Fascia Research Congress. Here the participants finally decided to distinguish the term “A FASCIA” from the term “THE FASCIAL SYSTEM”. The first term – ‘a fascia’ - indicates “*a sheath, a sheet or any number of other dissectible aggregations of connective tissue that forms beneath the skin to attach, enclose, separate muscles and other internal organs*”. This is a purely anatomical definition that has the aim of permitting everybody to know exactly what we’re talking about, to isolate these layers in cadavers and perform histological and morphological analysis, to sample the fasciae during surgery to evaluate pathological alterations and to study them in living subjects by means of imaging technology. Finally, it permits for the comparison of the results of anatomical studies performed by different researchers.

However, this distinct anatomical definition of ‘a fascia’ is not always helpful for use among all clinicians, because it may lead towards the exclusion of important tissues when trying to understand the function of the global fascial net during movement, including the interconnections of fascial tissues with joint capsules, aponeuroses, tendons, ligaments and intramuscular connective tissues (Schleip et al., 2012). Besides, as highlighted by Myers (2014), for those of us, however, who initiate treatment from the outside with our hands, the palpatory distinction between e.g. the transversalis fascia and parietal peritoneum can engage our interest as a research concern; but in practice the layers are inseparable; they move and respond together. For this reason, it is helpful to have the option of a second term – i.e. ‘the fascial system’ – which points the interest more into the functional aspects of the larger fascial net, including force transmission, sensory functions and wound regulation. With our proposed term “the fascial system” we

refer to a network of interacting, interrelated, interdependent tissues forming a complex whole, all collaborating to perform a movement. To better define what the fascial system is and what elements it includes, a new subcommittee was elected, consisting of Sue Adstrum, Robert Schleip, Carla Stecco, Paolo Tozzi and Can Yucesoy.

It is hoped that in the future both pathways of exploration may profit from the clarification of their respective terminologies. When communicating among clinicians interested in understanding the dynamics of the wider fascial system, then a more comprehensive terminology may be the most suitable. However, for the exploration of precise anatomical and histological relationships, and for the respective communication with medical scientists, the suggested definition of 'a fascia' with its much stricter inclusion parameters is highly recommended.

This will be an ongoing dialogue, in which much more work is necessary. This will include further terminological clarifications as well as further progress in trying to better understand the different backgrounds and values of the many diverse professions involved in the field of fascia research and fascia oriented therapies. A very big thank you needs to be expressed to all colleagues, who have contributed to this important clarification process in this journal.

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# Fascial plasticity – a new neurobiological explanation: Part 1

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**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. © 2003 Elsevier Science Ltd. All rights reserved.

## 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 1½ 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.

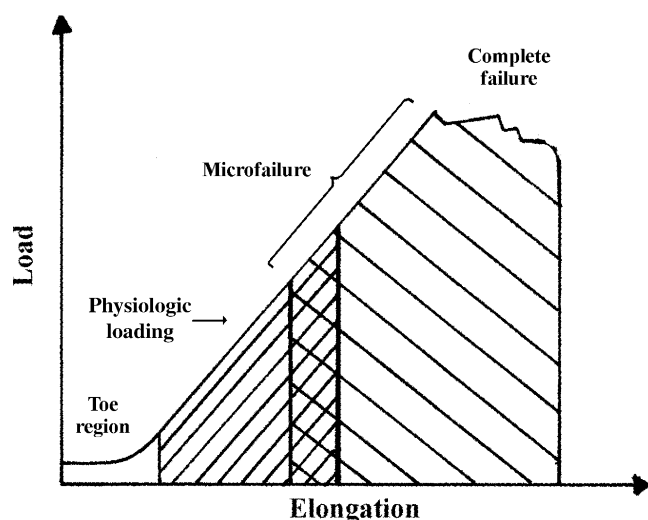
Nevertheless, the processes involved seem to require time as an

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
<b>Golgi</b> Type Ib	<ul style="list-style-type: none"> <li>● Myotendinous junctions</li> <li>● Attachment areas of aponeuroses</li> <li>● Ligaments of peripheral joints</li> <li>● Joint capsules</li> </ul>	<ul style="list-style-type: none"> <li>● <i>Golgi tendon organ</i>: to muscular contraction.</li> <li>● <i>Other Golgi receptors</i>: probably to <b>strong stretch</b> only</li> </ul>	<b>Tonus decrease</b> in related striated motor fibers
<b>Pacini and Paciniform</b> Type II	<ul style="list-style-type: none"> <li>● Myotendinous junctions</li> <li>● deep capsular layers</li> <li>● spinal ligaments</li> <li>● <b>investing muscular tissues</b></li> </ul>	<b>Rapid pressure changes</b> and vibrations	Used as proprioceptive feedback for movement control ( <b>sense of kinesthesia</b> )
<b>Ruffini</b> Type II	<ul style="list-style-type: none"> <li>● Ligaments of peripheral joints,</li> <li>● Dura mater</li> <li>● outer capsular layers</li> <li>● and other <b>tissues associated with regular stretching.</b></li> </ul>	<ul style="list-style-type: none"> <li>● Like Pacini, yet also to <b>sustained pressure.</b></li> <li>● Specially responsive to <b>tangential forces</b> (lateral stretch)</li> </ul>	<b>Inhibition of sympathetic activity</b>
<b>Interstitial</b> Type III and IV	<ul style="list-style-type: none"> <li>● <b>Most abundant receptor type.</b> Found almost everywhere, even inside bones</li> <li>● Highest density in periosteum.</li> </ul>	<ul style="list-style-type: none"> <li>● Rapid as well as sustained pressure changes.</li> <li>● 50% are <b>high-threshold</b> units, and 50% are <b>low-threshold</b> units</li> </ul>	<ul style="list-style-type: none"> <li>● Changes in <b>vasodilation</b></li> <li>● plus apparently in <b>plasma extra-vasation</b></li> </ul>

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 antibrachial, 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

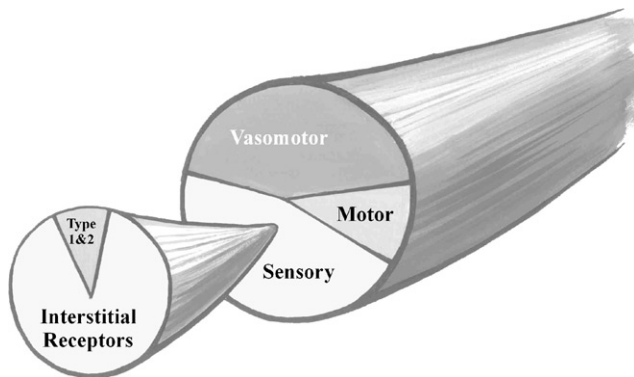
(HTP). A study of the Achilles tendon of cats revealed that about half of types III and IV endings encountered were LTP units and responded to light touch, even to touch as light as “with a painter’s brush” (Mitchell & Schmidt 1977). Based on this latter finding, does it not seem possible – indeed likely – 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).

### 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



**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.



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-González 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 stimulation of mechanoreceptors with resulting profound autonomic changes, and might therefore not need to rely on more *esoteric* assumptions (Arbuckle 1994).

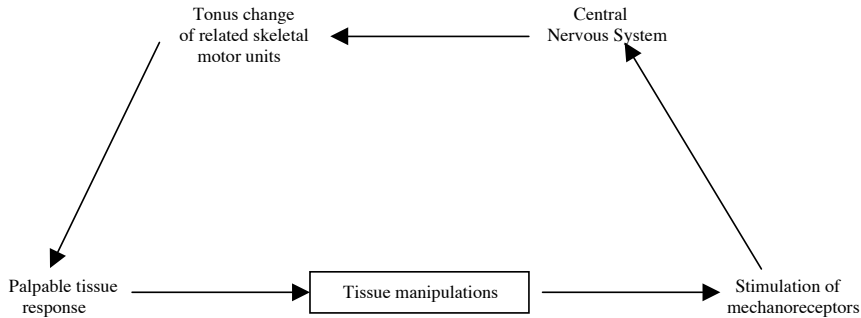
Recent discoveries concerning the richness of the *enteric nervous system* (Gershon 1999) have taught us that our 'belly brain' contains more than 100 million neurons and works largely independent of the cortical brain. It is interesting to note that the very small connection between

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 stimulation 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

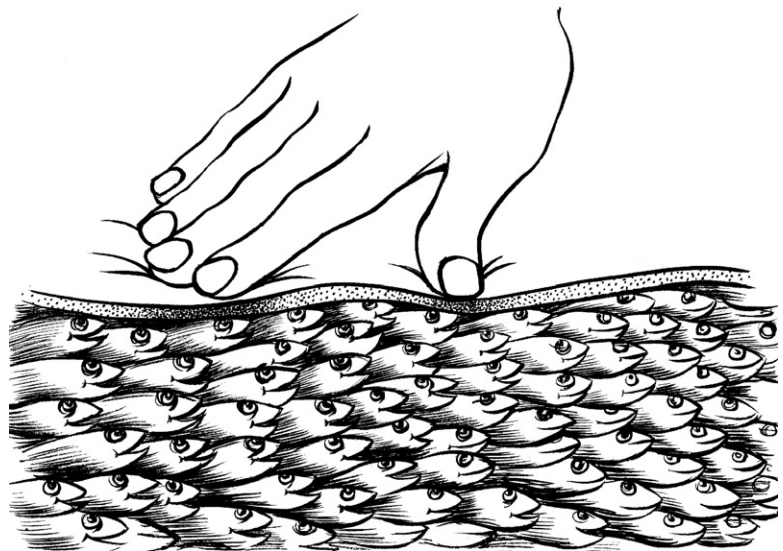
When discussing any changes in motor organization, it is important to realize that the central nervous system does not operate 'in muscles', i.e. a muscle is never activated as a whole. The functional units of the motor system are the so-called *motor units*, of which we have several million in our body, 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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# Fascial plasticity – a new neurobiological explanation

## Part 2

.....

**Robert Schleip**

**Abstract** Part 1 of this two part article showed that immediate fascial responsiveness to manipulation cannot be explained by its mechanical properties alone. Fascia is densely innervated by mechanoreceptors which are responsive to myofascial manipulation. They are intimately connected with the central nervous system and specially with the autonomic nervous system. Part 2 of the article shows how stimulation of these receptors can trigger viscosity changes in the ground substance. The discovery and implications of the existence of fascial smooth muscle cells are of special interest in relation to fibromyalgia, amongst other conditions. An attitudinal shift is suggested, from a mechanical body concept towards a cybernetic model, in which the practitioner's intervention are seen as stimulation for self-regulatory processes within the client's organism. Practical implications of this approach in myofascial manipulation will be explored.

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### Introduction

Part 1 of this article showed that fascial responsiveness cannot be explained by its mechanical properties alone. Fascia is populated by a dense network of mechanoreceptors. The majority of fascial sensory nerve endings which are stimulated by fascial manipulation are interstitial receptors (type III & IV) which have been shown to induce a change in local vasodilation. The additional group of Pacinian receptors seem to be involved in high-velocity manipulation, while Ruffini endings are mostly stimulated by slow deep pressure techniques, specially

if they involve tangential forces, i.e. lateral stretch (Kruger 1987). Stimulation of fascial mechanoreceptors leads to changes in muscle tonus which come primarily from a resetting of the gamma motor system, rather than the more volitional alpha motor coordination. Additionally, stimulation of Ruffini organs as well as of many of the interstitial receptors effects the autonomic nervous system, which can result in a lowering of sympathetic tone, or in changes in local vasodilation. Part 2 of this article will explore further implications and practical applications of this neurobiological orientation.

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## Mechanoreceptors influence local fluid dynamics

Let us 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 & Cabri 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 (Pischinger 1991, Barnes 1997). 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.

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 sensitive practitioners. This first autonomic feedback loop – here called '*Intrafascial Circulation Loop*' – is based on the work of Mitchell and Schmidt (1977) and is illustrated in Fig. 1.

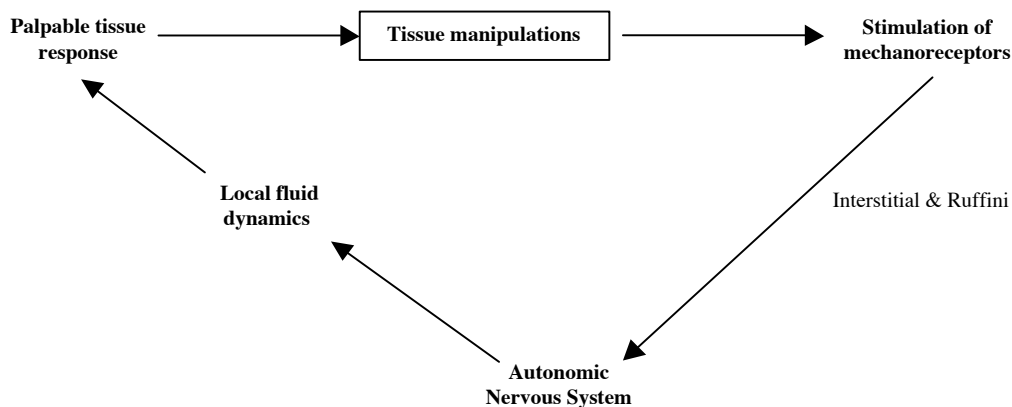
## Changes in hypothalamic tuning

And there is a second autonomic feedback loop. The interstitial mechanoreceptors can trigger an

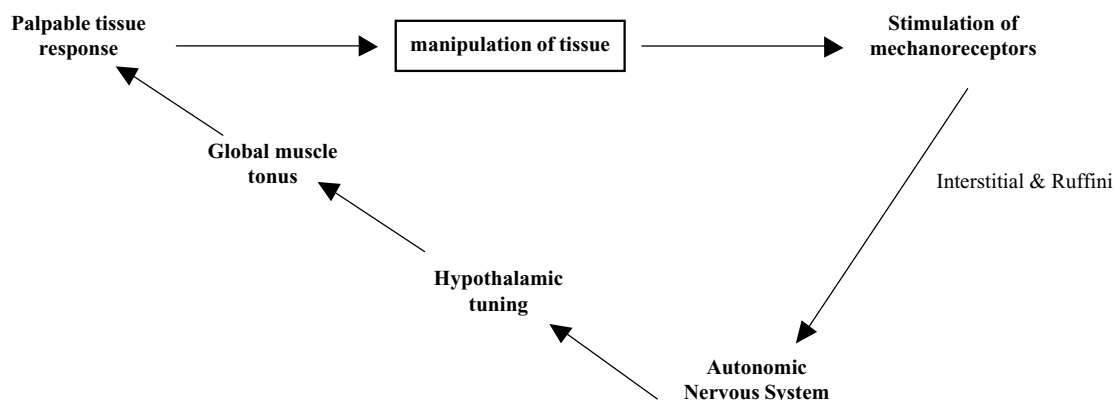
increase in *vagal tone* which leads towards more *trophotropic* tuning of the hypothalamus. Based on Gellhorn (1967) this results in global neuromuscular, emotional, cortical and endocrinal changes that are associated with deep and healthy relaxation (see the paragraph '*Touch research with cats and humans*' in Part 1). This *Hypothalamus-Loop* is illustrated in Fig. 2.

## Fascia is capable of spontaneous contraction



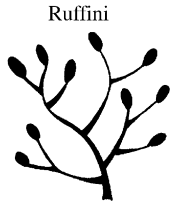

Yahia and her team in Montreal – after doing the study on the sensory innervation of fascia which was discussed in Part 1 – also conducted a fascinating study on the viscoelastic properties of the lumbodorsal fascia (Yahia et al. 1993). Performing various repeated tests with dynamic and static traction loading on fresh pieces of lumbodorsal fascia from cadavers, their findings supported the well-known force and time-dependent viscoelastic phenomena which have already been described by other researchers: creep, hysteresis, and stress relaxation (Chaitow & DeLany 2000). Yet they also



**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.



**Fig. 2** The ‘Hypothalamus-Loop’ based on Gellhorn (1967). 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.

Table 1 Fascial mechanoreceptors in myofascial manipulation		
	Responsiveness to manipulation	Results of stimulation
 <p>Golgi</p>	Probably only responsive to muscular contraction or to very <b>strong</b> manipulation.	<b>Tonus decrease</b> in related striated motor fibers.
 <p>Pacini</p>	Only responsive to <b>high velocity</b> or vibratory techniques	<ul style="list-style-type: none"> <li>● Increased local <b>proprioceptive attention</b></li> </ul>
 <p>Ruffini</p>	Specially responsive to <b>lateral stretch</b>	<ul style="list-style-type: none"> <li>● Increased local <b>proprioceptive attention</b></li> <li>● <b>Inhibition of sympathetic activity</b></li> </ul>
 <p>Interstitial</p>	<ul style="list-style-type: none"> <li>● 50% of these are <b>high threshold pressure units (HTP)</b></li> <li>● Other 50% are sensitive to <b>low pressure (LTP)</b></li> </ul>	<ul style="list-style-type: none"> <li>● Increased local <b>proprioceptive attention</b></li> <li>● Increase in <b>vasodilation</b> and respiration</li> <li>● Stimulation of HTP may produce <b>pain</b> and increase plasma <b>extravasation</b></li> </ul>

described for the first time a new phenomenon, which they termed *ligament contraction*. When stretched and held at a constant length repeatedly the tissues started to slowly increase their resistance (Table 1).

Since nobody had described such spontaneous contraction of connective tissue before, they performed repeated tests involving different temperatures, solutions, and humidity, all with similar results. After very carefully ruling

out the possibility of an experimental artifact, Yahia and associates finally concluded:

*A possible explanation for the contraction of fascia held under isometric conditions could be the intrusion of muscle fibers in the*

*lumbodorsal fascia. Indeed, many visceral muscles possess the ability to contract spontaneously. Price et al. (1981) demonstrated that strained and isometrically held intestinal muscles undergo relaxation followed by contraction. In order to test these specimens in a relaxed state (without spontaneous contraction), they used diverse techniques to suppress spontaneous activity, amongst them the use of epinephrine. An histological study of lumbodorsal fascia would therefore be desirable to evaluate whether muscles play a role in the contraction observed (Yahia et al. 1993).*

### The discovery of fascial smooth muscle cells

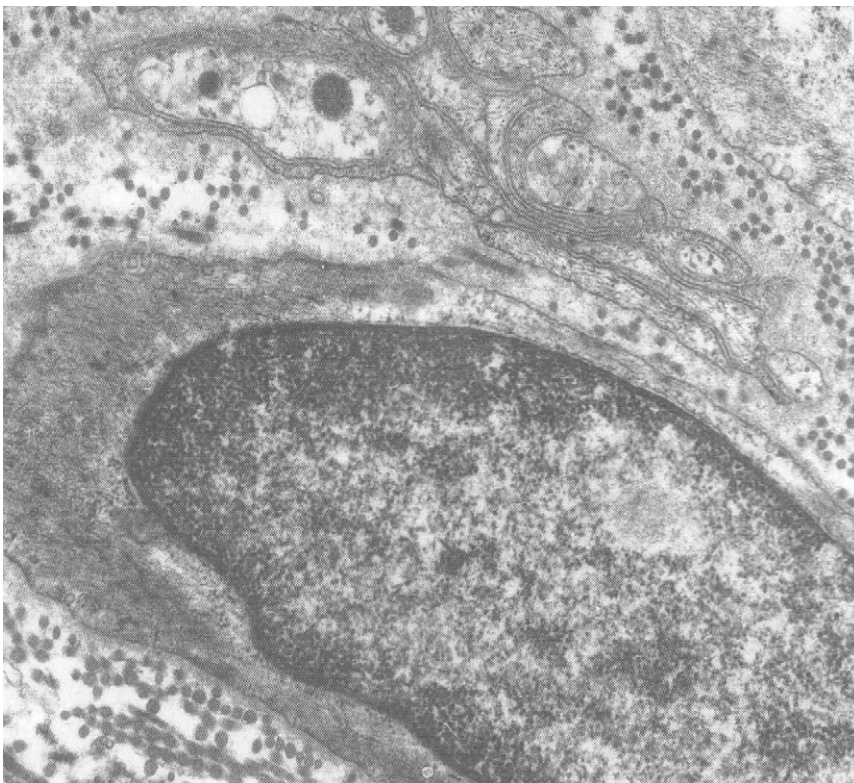
A few years later, in 1996, a German anatomy professor, Staubesand published an exciting new paper. He

and his Chinese co-worker Li studied the fascia cruris in humans with electron photomicroscopy for several years and found *smooth muscle cells* embedded within the collagen fibers (Staubesand & Li 1996) (Fig. 3). For a more detailed description of this discovery see Box 1 'Fascia is alive!'

Interestingly, this article also reported – similar to Yahia's innervation study – the widespread existence of intrafascial nerves. Staubesand describes a rich intrafascial supply of capillaries, autonomic nerves and sensory nerve endings. Based on his findings, he concluded that it is likely that these fascial smooth muscle cells enable the autonomic nervous system to regulate a fascial *pre-tension* independently of the muscular tonus (Staubesand & Li 1997, Staubesand et al. 1997). He therefore postulates

that this new understanding of fascia as an actively adapting organ gives fascia in general a much higher functional importance, and that the close links between fascia and autonomics may have far-reaching clinical implications.

Unfortunately, Staubesand was unaware that Yahia's research had already demonstrated that fascia has the ability to actively contract and to do so with measurable and significant effects. But Yahia could not isolate or identify the related muscle cells. Staubesand on the other hand had been able to identify and to photograph the related muscle cells, but he himself had no proof at that time that they are powerful enough to have any functional importance. Nevertheless it seems justified to say that both studies taken together show that there are smooth muscle cells embedded within fascia, and that it is highly probable that they are involved in the regulation of an intrafascial pre-tension.



**Fig. 3** Electron photomicroscopy of a typical smooth muscle cell within the Fascia cruris. Above it is the terminal portion of a type IV (unmyelinated) sensory neuron. (Photo reproduced with kind permission of Springer Verlag, first published in Staubesand 1996.)

### Myofibroblasts and tissue contractility

Compared with striated muscle cells, smooth muscle cells offer a more efficient transformation of chemical energy into mechanical strength. It has long been known that fibroblasts often transform into *myofibroblasts* which contain smooth muscle actin fibers and can therefore actively contract. This happens in pathological situations like Dupuytren's contracture, liver cirrhosis, in rheumatic arthritis and a few other inflammatory processes. Yet it is also a productive element of early wound healing, and myofibroblasts are found regularly in healthy skin, in the spleen, uterus, ovaries, circulatory vessels, periodontal ligaments and pulmonary septa (van den Berg & Cabri 1999).

**Box 1** Fascia is alive!

The following is an excerpt of an interview from the author with J. Staubesand, now emeritus professor of anatomy at the University of Freiburg, Germany

The complete version is available at [www.somatics.de](http://www.somatics.de)

Staubesand: We did some electron photomicrograph studies of the Fascia cruris, which is the connective tissue covering the lower leg in humans. Rather surprisingly we found isolated smooth muscle cells within the fascia. Additionally, we found some intrafascial nerve fibers and sensory nerve endings which have not been reported previously.

**Do you think these smooth muscle cells have any functional significance?**

That is indeed possible, although at this time one cannot say for sure. Because of the microscopically thin layers of tissue that we examined in our electron photomicrographic studies, we are not yet able to say anything about the relative three-dimensional density of the smooth muscle cells within fascia. Yet it appears likely that these smooth muscle cells are there for a functional reason. Based on our findings it seems quite possible that the body is able to regulate a fascial pre-tension via those smooth muscle cells, in order to adjust to different muscular tonus demands. This function would also explain the amazingly widespread presence of autonomic nerves and capillaries which we found in fascia.

It is true that such a function puts fascia into a very different picture than in the past, where it was believed that fascia would only adjust passively to short-term changes in tensional demands. This new picture of fascia as an actively adapting organ; and the widespread existence of various intrafascial neural receptors, gives fascia a much higher functional importance.

**What kind of nerve supply did you find in the fascial sheets that you studied? Were they sympathetic fibers?**

This we can't say with certainty. Further studies are necessary to clarify this question. Yet what we can say is that there are myelinated, as well as unmyelinated nerve fibers in fascia. The myelinated axons are generally regarded as sensory. The unmyelinated nerve fibers could have motor functions as the efferent nerves of the autonomic nervous system to the smooth muscle fibers, or they could also serve other autonomic nervous system functions. Based on studies from Heppelman and others (Heppelman 1995) on pain receptors in the joint capsule of the knee in cats and due to striking similarities with our observations we can assume that there are also pain receptors in the fascial tissues that we examined in humans.

In our studies we found that what had previously been described as perforations of the superficial fascial layer by the venae perforantes, are regularly created by a triade of vein, artery and nerve. And these perforations are quite numerous. For example, in humans there are about 150 such triad perforations in each leg.

**In your publications you also mentioned a possible relevance for the understanding and treatment of fibromyalgia**

With fibromyalgia the main understanding has been that the pain receptors are in the muscle tissue. Yet now we know that there are many sensory receptors, including pain receptors in fascia, which points our attention in fibromyalgia, as well as in many other kinds of soft-tissue pain syndromes to a much higher value of therapeutic interventions in the fascia itself.

**What is the most relevant aspect of your research for manual therapists?**

I believe that the most important aspect of our findings for your work relates to the innervation of fascia. The receptors that we found in the lower leg fascia in humans could be responsible for several types of myofascial pain sensations. If you could influence these fascial receptors with your manipulation this could be of significant importance.

Another aspect is the innervation and direct connection of fascia with the autonomic nervous system. It now appears that the fascial tonus might be influenced and regulated by the state of the autonomic nervous system. Plus – and this should have ramifications for your work – any intervention in the fascial system may have an effect on the autonomic nervous system in general and on all the organs which are directly affected by the autonomic nervous system. To put it more simply: any intervention on the fascia is also an intervention on the autonomic system.

From a teleological perspective it makes sense that an interspersing of smooth muscle cells into fascial sheets equips the organism with an accessory tension system to increase muscular tonus and offers an evolutionary survival advantage in fight/flight survival situations. Staubesand's study had demonstrated a *scissor-like* configuration of the collagen

fibers in the epimysium. This arrangement makes perfect sense as it allows a small quantity of intrafascial smooth muscle cells to effect a relatively large lattice network.

An interspersing of smooth muscle cells into fascial envelopes would also explain the following observation: The fascial lining of many organs consists mostly of

collagen fibers, whose small range of elasticity allows for minute length changes only. Yet the spleen can shrink to half size within a few minutes (which has been shown to happen in dogs when their blood supply in the spleen is needed due to strenuous activity). The most likely explanation for this are smooth muscle cells embedded within that organ capsule.



## Fascial tonus, breathing, and fibromyalgia

Tonus regulation of fascial smooth muscle cells is most likely achieved via the sympathetic nervous system as well as vasoconstrictor substances such as CO<sub>2</sub>. The discovery of fascial muscle cells therefore opens a doorway for exciting speculations about a direct link between fascial behavior and the pH of the body, which is directly linked to breathing function and CO<sub>2</sub> levels. As Chaitow, Bradley and Gilbert showed (Chaitow et al. 2002) there is already a clear link between smooth muscle contraction and depleted levels of CO<sub>2</sub> such as occurs in relative respiratory alkalinity. When there is a shift toward increased alkalinity due to – for example – hyperventilation – vasoconstriction is automatic and dramatic. Possibly, at this same time fascial smooth muscle cells contract and increase overall fascial tension. The implications for such changes in conditions such as fibromyalgia and chronic fatigue are enormous, since a common clinical finding is that most people with FMS and CFS are frank or borderline hyperventilators.

One can also speculate about the possible effect of increased serotonin levels on fascial smooth muscle cells. Serotonin has been known to be an enhancing agonist for smooth

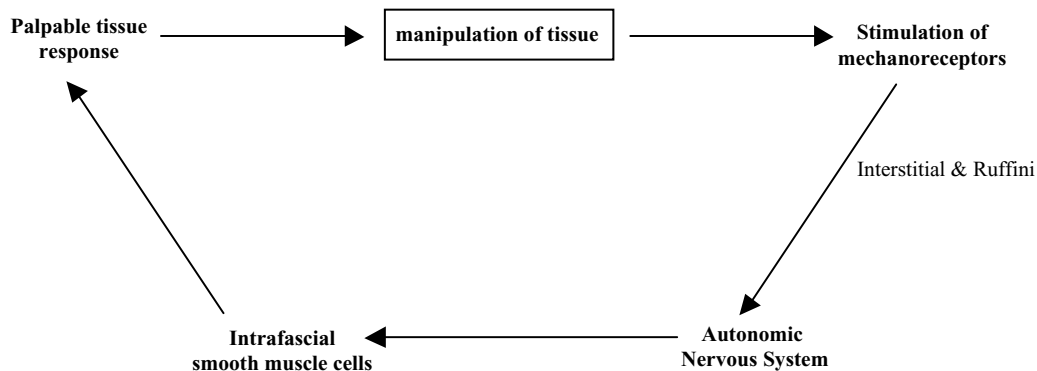
muscle contractions such as peristaltic activity or vasoconstriction in large pulmonary vessels. Unusually high levels of serotonin have recently been found in the cerebrospinal fluid of fibromyalgia patients (Pongratz & Späth 2001). A possible connection between fibromyalgia and serotonin-mediated hypertonicity of fascial smooth muscle cells might be a worthwhile investigation. On the other hand, serotonin has been shown to decrease the pain threshold of group IV receptors (Mitchell & Schmidt 1977), which could mean that the increased pain sensitivity of those receptors in fibromyalgia might be less of a motor dysfunction (fascial smooth muscle cell hypertonicity) but more of a sensory regulation dysfunction.

Based on Yahia and Staubesand, Fig. 4 illustrates a third autonomic feedback loop – which I call ‘*Fascial Contraction Loop*’ as a potential factor behind short-term fascial plasticity. Leaving aside the possible interactions of chemical vasoconstrictor substances for the moment, this ‘loop’ focuses on neural network dynamics alone. To put it simply: Stimulation of intrafascial mechanoreceptors (in this case mostly free nerve endings) triggers the autonomic nervous system to alter the tonus of intrafascial smooth muscle cells.

## How about visceral ligaments?

In visceral osteopathy it is often claimed that gentle manipulation of a visceral ligament can induce an immediate and palpable release within that ligament (Barral & Mercier 1988). Similar concepts have also been suggested for osteopathic work with skeletal ligaments (Barral & Croibier 2000, Crow et al. 2001). Since ligaments can be seen as special arrangements of fascia – often ligaments are nothing but local thickenings within larger fascial sheets – the question arises: how is this possible? As was shown in Part 1, that in order to create an immediate yet permanent lengthening of any substantial fascial structure with mechanical means, much larger amounts of force and/or time are required than are usually applied in gentle non-thrust manipulations.

Fascial smooth muscle cells and active fascial contractility have only been reported from large fascial sheets. This is also where the scissor-like fiber arrangement makes it possible for a relatively small amount of interspersed contractile cells to effect the whole fascial lattice network. It therefore seems unlikely that intra-ligamentous smooth muscle cells might be the basis of this reported osteopathic phenomenon.



**Fig. 4** The ‘*Fascial Contraction Loop*’ (based on Yahia and on Staubesand). Embedded between the collagen fibers of fascia are smooth muscle cells, which are regulated by the autonomic nervous system. Their activation can cause an active intrafascial tissue contraction.

It seems more likely that the osteopathic soft tissue manipulation stimulates mechanoreceptors within the treated ligament, which then induces a relaxation of related (smooth or striated) muscle fibers; and this is felt as a 'ligament relaxation' by the practitioner. Additionally, it is quite possible – particularly with stimulation of visceral ligaments – that specific metabolic ground substance changes and physiologic organ function changes might be triggered in the neighborhood, which could also be palpable. Yet the actual length of the ligament would not be altered. If true, this explanation would challenge – or modify – some of the current assumptions in osteopathy and would lead to several different practical consequences in that work.

### Acupuncture points and fascia

As we learned in Part 1 of this article, an electron photomicroscopy study of the Fascia cruris (Staubesand 1997, Staubesand & Li 1997) showed that there are numerous perforations of the superficial fascia layer that are all characterized by a perforating triad of vein, artery and nerve (Fig. 5). Staubesand could identify that most of the perforating nerves in these triads are unmyelated autonomic nerves.

A study by Heine around the same time also documented the existence of these triad perforation points in the superficial fascia. Heine, a German researcher who has been involved in the study of acupuncture and other complimentary health disciplines, found that the majority (82%) of these perforation points are topographically identical with the 361 classical acupuncture points in traditional Chinese acupuncture (Heine 1995).



**Fig. 5** The superficial fascia is perforated at specific points by a triad of nerve (left), vein (large body in middle) and artery. Based on Heine most of these perforation points are topographically identical with traditional Chinese acupuncture points. The perforating nerves usually innervate Pacinian and Meissner corpuscles under the skin.

This stimulated a German surgeon to conduct a clinical study together with Heine. They studied these fascial perforation points in patients suffering from chronic shoulder–neck or shoulder–arm pain. They found that the perforation points in these patients showed a peculiar anomaly. The perforating vessels were 'strangled' together by an unusually thick *ring of collagen fibers* around them, directly on top of the perforation hole. The surgeon then treated these points with microsurgery in order to loosen the strangulations and to achieve a *freer exit* of those vessels. This resulted in a significant improvement for the patients (Bauer & Heine 1998).

Many took this as clear evidence of a new mechanical explanation model for pain in relation to acupuncture points. Yet just a year later a back pain researcher from Spain published a study which seems to question some of Bauer and Heine's assumptions and which adds

an exciting new dimension (Kovacs et al. 1997). Using a well-orchestrated double-blind study design with patients suffering from chronic low back pain, *surgical staples* were implanted under their skin. An interesting point was that the location of the implants was defined by their innervation (as trigger points) and was carefully chosen not to coincide with Chinese acupuncture points. The result: Kovacs' treatment led to a clear pain reduction in the majority of his patients, with at least a similar statistical improvements to those that Bauer and Heine had with their patients.

Kovacs' suggested the following explanation: most likely a class of *neuropeptides*, called enkephalins, are released by both treatments, which then counteract the release of substance P and other neuropeptides which are associated with pain and which support the activation of nociceptive fibers. In other words: the stimulation of certain

noci- and/or mechanoreceptors under the skin stimulates the release of specific neuropeptides that help to deactivate pain receptors which are instrumental in the maintenance of chronic pain (Kovacs et al. 1997).

### A dynamic systems approach

The beauty of Kovac's approach lies in his view of the nervous system as 'a wet tropical jungle', i.e. in his inclusion of the liquid aspects of the nervous system. Compared to the more mechanically oriented treatment approach of Bauer and Heine, Kovac looks at the body as a *cybernetic system* in which an

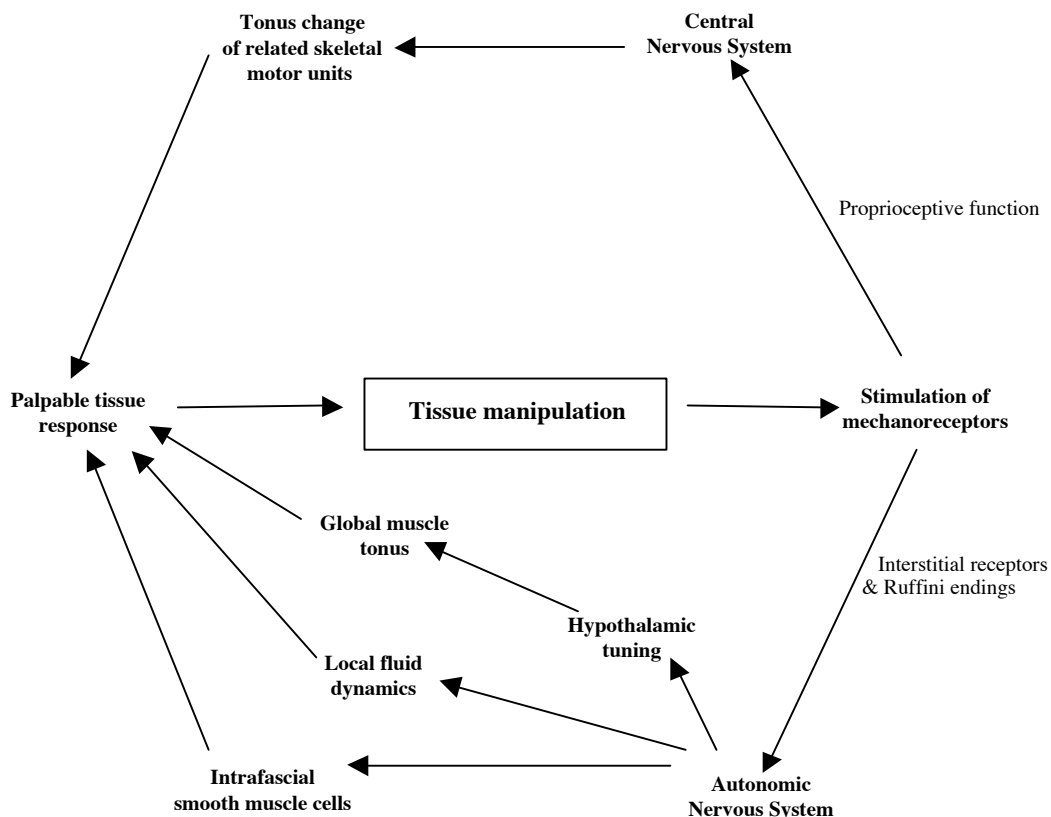
intervention is seen as stimulation for complex internal self-regulatory processes.

Cybernetic approaches often work with *flow charts* as useful simplifications for complex dynamic interdependencies. Figure 6 can be seen as a first attempt toward an analysis of some of the neural factors behind immediate fascial plasticity. It includes the four different feedback loops described earlier in this article. This flow chart does not include any neuroendocrine aspects, although it is very likely that they are significantly involved in myofascial manipulation. Following Kovac's example, it would be useful for future research to explore whether deep tissue work triggers a release of

specific neuropeptides, which might explain some of the profound short term as well as long-term effects of this work.

### From hero technician to a humble midwife

It seems clear that in order to better understand and to use fascial plasticity, we need to include the self-regulatory dynamics of the nervous system. This will include an attitudinal shift in the practitioner. If we are willing to move from a mechanical view of the body towards an inclusion of the neuroendocrine system, we are doing well to prepare our brain (and guts) to think in nonlinear system



**Fig. 6** Flow chart of several processes involved in the neural dynamics of immediate tissue plasticity in myofascial manipulation. This chart includes the four different feedback loops which were discussed in part one of this article series. The practitioner's manipulation stimulates intrafascial mechanoreceptors, which are then processed by the central nervous system and the autonomic nervous system. The response of the central nervous system changes the tonus of some related striated muscle fibers. The autonomic nervous system response includes an altered global muscle tonus, a change in local vasodilation and tissue viscosity, and a lowered tonus of intrafascial smooth muscle cells.

<b>Table 2</b>	
<b>Classical concept: The Body as a Mechanical Object</b>	<b>New neurobiological model: The Body as a Self-Regulatory Process</b>
The body is seen as a perfect or imperfect <b>machine</b> , governed mainly by classical Newtonian physics	The body is seen as a self-regulating (SR) <b>biological organism</b> , involving nonlinear system dynamics, complexity and autopoiesis
Typical ' <b>industrial age</b> ' viewpoint	Typical ' <b>information age</b> ' viewpoint
Clear distinction between <b>structure and function</b>	No clear distinction between structure and function
Less focus on nervous system	Strong inclusion of <b>nervous system</b>
Subject/object separation ('principles of <b>intervention</b> ')	Subject-object connection (' <b>interaction</b> ' instead of intervention)
<b>Problem-solving</b> attitude	Focus on <b>enhancing</b> already existing SR
A machine has a limited number of variables. An inner sense of absolute <b>certainty</b> in the practitioner is therefore seen as achievable and as desirable	The system has high degree of complexity with almost unlimited variables. Practitioner personality needs to be comfortable operating with <b>uncertainty</b> principles
Local ' <b>precision</b> ' is important and admired	Good <b>timing and gradation</b> (dosage) are getting at least as important
' <b>Master technician</b> ' as idol	' <b>Facilitator</b> ' or ' <b>midwife</b> ' as idols
Typical work example: Direct mobilization of a precise ' <b>spinal fixation</b> ' or sacral torsion by the practitioner	Typical work example: Inclusion of facilitated active client <b>micromovements</b> during the hands-on work

dynamics. The self-regulatory complexity of the nervous system could be compared with that of a rainforest or a metropolitan city. According to Senge and others, in dealing with such complex systems it usually does not work very well to assume the role of a *master* who interferes from the outside with heroic interventions and who believes to be able to predict his results with certainty. More often than not such linear interventions produce unforeseen long-term reactions which are counterproductive (Senge 1990).

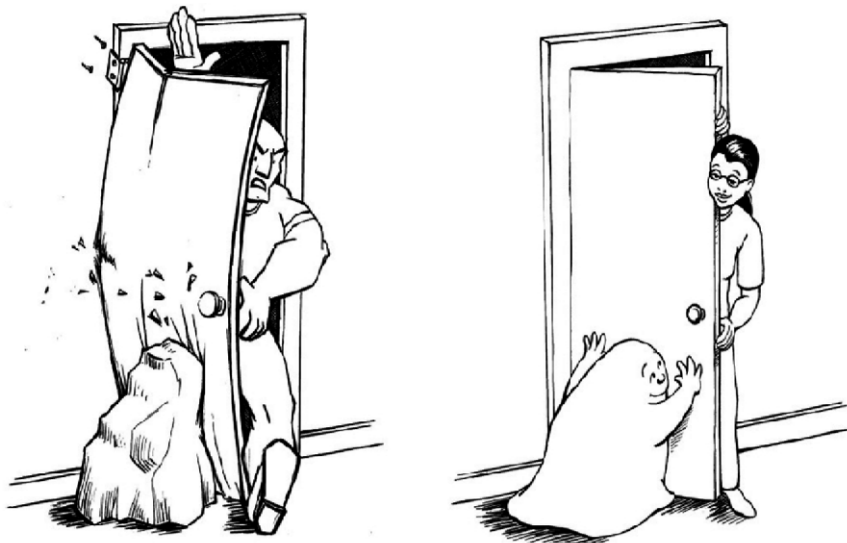
Usually, it works better to assume the more humble role of a *facilitator*, who is curiously interested in learning and whose personality is more comfortable to deal with uncertainty principles. In the context of a bodywork session, practitioner and client then work together as '*a learning team*' (Petersen 2000).

Table 2 shows some of the consequences of this shift. Rather than seeing practitioner and client as clearly separable entities (subject and object) and discussing different

'principles of intervention' in manual therapy in which the practitioner performs a number of active techniques on a mostly passive client, it is suggested that there is benefit to be gained by involving the client as an active

partner in an 'interaction' process, for example with specific micromovements during the fascial manipulations.

Note that the common distinction between structure (e.g. bones and connective tissue) and function



**Fig. 7** A door blocked by a rock requires a different approach than if one deals with an animated obstacle. Similarly, a blocked joint or immobile tissue can be understood in purely mechanical terms or as an active self-regulatory system. The choice of approach depends largely on whether the practitioner sees any neural dynamics involved in the specific situation on the client's side. This article makes a point to see fascia as enervated and as alive, and therefore suggests to treat fascia more with the second approach.

(neuromuscular organization) is no longer useful within such a picture. The Nobel laureate Ludwig von Bertalanffy puts it this way:

*The antithesis of structure and function, morphology and physiology, is based upon a static conception of the organism. In a machine there is a fixed arrangement that can be set in motion but can also be at rest. In a similar way the pre-established structure of, say, the heart is distinguished from its function, namely, rhythmical contraction. Actually, this separation between a pre-established structure and processes occurring in that structure does not apply to the living organism. For the organism is the expression of an everlasting, orderly process, though, on the other hand, this process is sustained by underlying structures and organized forms. What is described in morphology as organic forms and structures, is in reality a momentary cross section through a spatio-temporal pattern. What are called structures are slow processes of long duration, functions are quick processes of short duration. If we say that a function such as the contraction of a muscle is performed by a structure, it means that a quick and short process is superimposed on a long-lasting and slowly running wave. (von Bertalanffy 1952).*

## A different role model

The role of a ‘master technician’ in Table 2 can best be described by the following story: The heating system of a big steam boat was broken and for several days nobody could fix it. Finally, a master technician was called in. He just walked around and looked at everything and finally took out a little hammer from his pocket and hit a little valve, which immediately fixed the problem and the machine started working again. When his bill of \$1000 arrived, the captain didn’t want to believe such a high sum for such little work, so he

asked for a more specified bill. The next day the new bill arrived, it said:

*“For adjusting a little valve: \$ 0.01.  
For knowing where: \$ 999.99”.*

Many bodywork practitioners still worship this story as an ideal of *mastery* in their work, although it

clearly belongs in the realm of dealing with a mechanical universe. If one is willing to deal with fascia in a dynamic systems perspective, it is more appropriate to assume the role of a *midwife* or *facilitator* that is skillfully assisting a self-regulatory process of the

**Table 3** Practical applications

### WHERE TO WORK:

1. *Short and tight tissues*  
Bring attention to the primary (inappropriately) shortened and hypertoned myofascial tissues.
2. *Include antagonists*  
Include bringing attention to the antagonistic muscle fibers of the related joint.
3. *Respect receptor density*  
Give extra time and attention to those tissues that have an usually high density with mechanoreceptors (suboccipital muscles, periosteum, palmar and plantar fascia, myotendinous junctions, ligaments).
4. *Face and Hands*  
Give high attention to those myofascial fibers that move the face or hands
5. *Abdomen and Pelvis*  
Deep pressure on visceral nerves as well as sustained pressure on the pelvis have been proven to increase vagal tonus

### HOW TO WORK:

6. *Timing*  
**For tonus decrease:** slow and melting to induce parasympathetic state and to avoid myotatic stretch reflex  
**For focusing attention:** stimulating, calling attention, more rapid changes, but never boring.
7. *Ruffini-angle*  
Tangential pressure (lateral stretch) is ideal to stimulate Ruffini organs, which tend to lower sympathetic tone.
8. *Attention to ANS*  
Pay great attention to the state of the autonomic nervous system (which influences the body’s overall tonus regulation).
9. *Unusual sensations*  
Create unusual body sensations that are most likely to be interpreted as ‘significant’ by the filtering action of the reticular formation of the central nervous system; i.e.:  
(a) unusually **strong** stretch of those fibers  
(b) unusually **subtle** stimulation (‘whispering effect’)  
(c) unusually **specific** stimulation  
(d) sensations that are always slightly **changing/moving** in a not precisely predictable manner
10. *Immediate feedback inclusion*  
As soon as you sense the beginning of a tonus change, mirror this back with your touch in some way to the tissue. The more precise, immediate and refined your feedback inclusion is, the more effective your interaction will be.
11. *Animistic Thinking*  
A motherly caring attitude towards lots of little gnomish entities inhabiting the tissue triggers usually the highest ‘sensory acuity’ in the practitioner’s (mammalian) nervous system.

### CLIENT PARTICIPATION

12. *AMPs*  
Engage the client in active micromovement participation (AMP). The slower and more refined they are and the more attention they demand, the better.
13. Ask and allow for a *deepening of proprioception*.
14. *Relate body perceptions* and movements to functional activities and include the external space orientation as well as the social meaning aspects of altered body expressions.

organism. This ideal is expressed in the Chinese saying:

*“Give a man a fish, and you feed him for a day.  
Teach him how to fish, and you feed him for a life time”.*

## Where to work

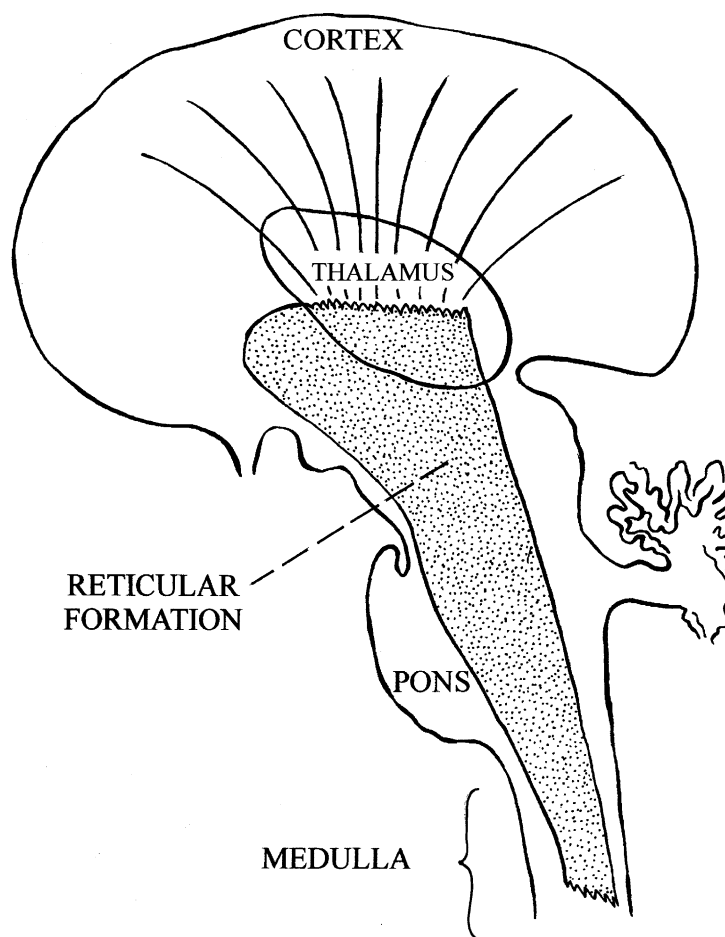
Table 3 gives some recommendations for the practical work. Since myofascial work seems to be more focused on softening or *release* of tight tissues (Rolf 1977, Barnes 1990) rather than on an increase of tonification, it usually includes work on those myofascial tissues which appear as unnecessarily short and tight (see rule 1 in Table 3). Yet if one includes the self-regulatory system dynamics of the client's motor coordination, it is also useful to include work on the antagonists of those hypertonic tissues (rule 2). For example if the client shows a chronic anterior pelvic tilt (not only in standing and walking but also in supine and prone position on the table) and the Modified Thomas Test (Tunnel 1998) has revealed that one or several hip flexor muscles are short, it is often helpful to work with the upper hamstrings and gluteals (rule 2) in addition to direct work with the shortened hip flexors (rule 1).

The basis for rule 2 rests primarily on the clinical experience of the author. Nevertheless, the following theoretical explanation might be applicable: Agonists and antagonists of a specific skeletal joint are neurologically closely connected via a complex network of spinal and supraspinal reflexes and feedback loops (Kandel 1995). Any tonus change in the agonists will tend to also trigger changes in the related antagonists, and vice versa. Bringing attention to the antagonistic fibers of the primarily shortened myofascial tissue might therefore provide an additional input for the

nervous system regulation around this joint. Giving this additional invitation for the nervous system to *‘please re-evaluate your tonus regulation around this joint’* might therefore be more efficient than only repeating the same access road (via the shortened agonistic tissues) again and again. Nevertheless, usually more work should be done on the shortened agonistic tissues than on their opposing antagonists.

An understanding of the ‘inner anatomy’ of the client, i.e. of the client's body scheme organization within the cortex, supports rule 4, i.e. to give extra attention to the myofascial tissues which are involved in movements of the face

and hands. Together both representational areas make up about two-thirds of the ‘inner body organization’ in the brain. In the cortex, there is a general tendency for local spreading: the excitement of a local cortical area will tend to influence surrounding areas in its neighborhood. E.g. if the practitioner achieves a healthy tonus change in previously tightened hand – and face muscles within 15 minutes of myofascial work, it is more likely that this change – involving two-thirds of the clients internal body image organization – will spread to the rest of the body, compared to if the practitioner works for an hour on the trunk only (which makes up



**Fig. 8** The manual practitioner needs to understand the filtering action of *Reticular formation* in the spinal cord and brain stem. Only if this system interprets the practitioner's touch as significant or interesting, will it allow this input to reach higher areas of the clients body organization.

only a minor portion of the somatomotor cortex).

Rule 5 rests on the research which was discussed in Part 1 of this article (Folkow 1962, Koizumi & Brooks 1972).

## How to work

The basis for rule 7 has been explored in Part 1, same for rule 8 which relates to Gellhorn's research on trophotropic and ergotropic tuning states (Gellhorn 1967).

Rule 9 acknowledges the fact that the reticular formation (see Fig. 8) usually filters out all manual input which is interpreted as nonsignificant by the clients central nervous system. An example: while probably sitting and reading this article, the reader's underwear and other pieces of clothes are touching the body, sometimes with an amount of pressure comparable to very fine craniosacral bodywork. Also the ischial tuberosities may be exposed to pressure comparable to strong myofascial work. Yet both inputs are readily ignored in everyday life and do not lead to significant short-term changes.

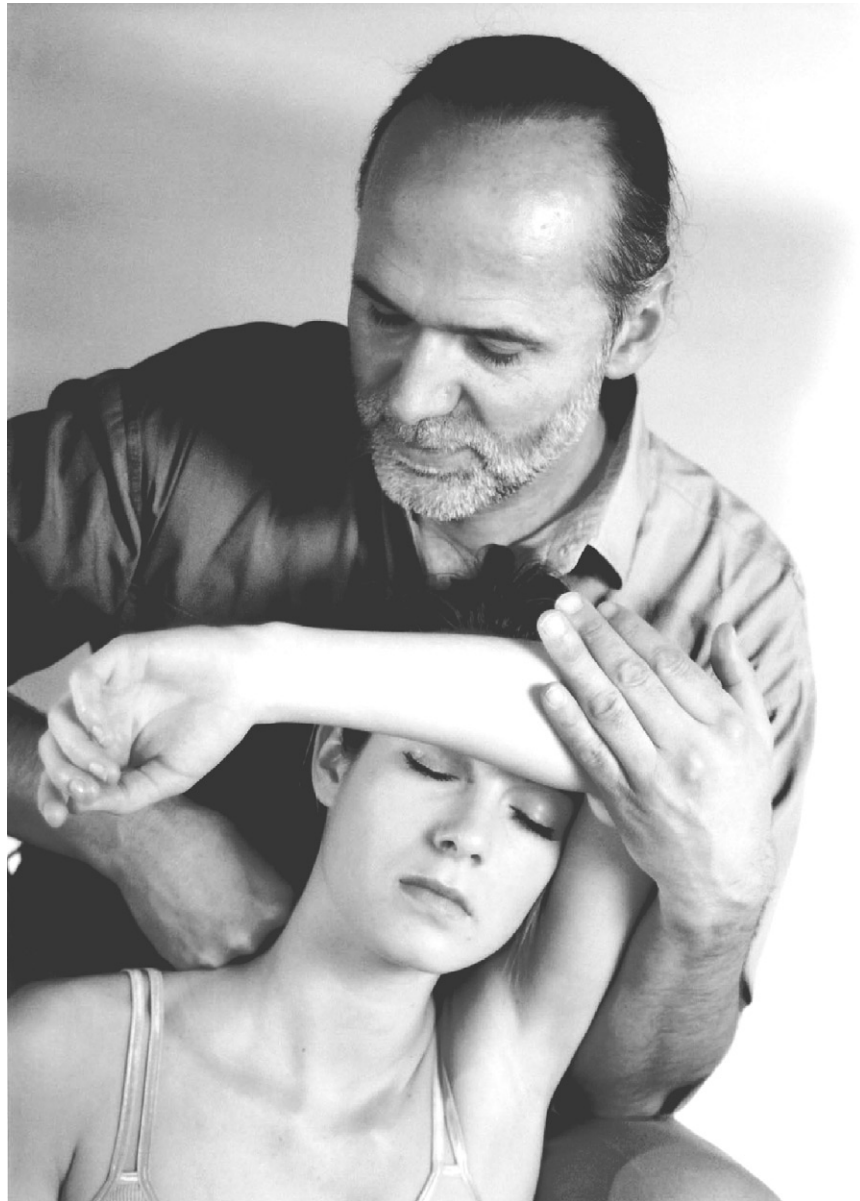
Rules 10 and 11 underline the importance of palpatory sensitivity. Imagine the school of fish which we used as an analogy in Part 1 for the hundreds of motor units under the practitioner's hand or elbow. If lets say one or two of those fish (motor units) start changing their tonus and if the practitioner's hand is able to perceive this, it can mirror this change back to the tissue and might influence other fish to flow in the same direction. Whereas if the practitioner's hand or elbow is not sensitive enough to perceive this change, this chance might be lost. The question then comes up: How can we increase the sensory acuity? The author's own teaching experience supports the observation,

that our own (mammalian) nervous system tends to work at it's highest sensory acuity if we are engaged in a motherly bonding relationship context. Imagining dozens of baby like gnomish entities inhabiting the fascial tissue therefore usually works better than imagining nerves,

collagen fibers or other images from dissections or anatomy books.

## Active client participation

If it is true that myofascial manipulation includes the



**Fig. 9** Example of the use of AMPs (active movement participation) of the client in a Roling® structural integration session. While deeply melting with one hand into the tissue and specific joints of the upper thorax, the author guides the client to support his myofascial work with subtle and nonhabitual slow motion participations. Here the client performs a lateral bending movement of the thorax combined with a cranially directed extension (following the elbow) in order to increase an opening of the thoracic vertebral joints. (Photo reproduced with kind permission of European Roling Association.)

self-regulation dynamics of the client's nervous system, then it makes sense to involve the client more actively in the session. Figure 9 shows a typical example of using active micromovement participation in a sitting client. Refined verbal and tactile guidance from the practitioner serve to facilitate subtle slow motion participations of the client such that the nervous system is more deeply involved in the coordination around a specific joint or area.

Recent insights into the organization of the motor cortex have shown that it is less organized around topographical body parts but rather around complex elementary movements towards specific spacial end-point directions (Graziano et al. 2002). Rule 14 takes this insight even further, by preferring movement participations with a clear functional intention (e.g. reaching for something, or pushing something away) which involve the client's nervous system more fully than mere mechanically/geometrically described movements (Reed 1996).

## Conclusion

Fascia is alive. Practitioners working with this truly fascinating tissue should understand that it is innervated by four different kinds of mechanoreceptors. Without an inclusion of their responsiveness to various kinds of touch, the immediate tissue release effects in myofascial manipulation cannot be adequately explained. Fascia has been shown to contain smooth muscle cells which seem to be responsible for its ability of active 'ligament contraction'. There are strong links between fascia and the autonomic nervous system which effect fascial tonus, local tissue viscosity, and possible fibromyalgia. A shift from a mechanically oriented

'technician' point of view towards an inclusion of the self-regulation dynamics of the clients nervous system is therefore advocated. Rather than seeing the practitioner as the expert technician, client and practitioner work together as a learning team in order to open new options for movement and posture organization.

## ACKNOWLEDGEMENTS

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# Fascia Research 2012: Third International Fascia Research Congress

**KEYWORDS:** Fascia, musculoskeletal manipulation, research, congresses

In October 2007, I opened the First International Fascia Research Congress by noting that the congress was the realization of a dream 30 years in the making. During the 1970s, I completed my residency training in physical medicine and rehabilitation, and the coursework and research training for my doctorate. Those studies taught me to fix muscles, bones, and nerves, and to train new movements and restore function in people with arthritis, stroke, spinal cord injury, amputations, and cerebral palsy (to list just a few). They also exposed me to the underlying principles of physiology, biochemistry, and psychology that guided the clinical treatments. But when it came to connective tissue, all I and my colleagues knew was that, when you heated a rat tail, you could stretch it. There was no other relevant research that I could find. At that moment, three decades ago, I started dreaming of a fascia research congress that would bring together widely separate research disciplines in the service of the clinician.

Every year, publications on fascia in Medline have risen: by 2005, they had reached 600 annually from 100 annually in the 1970s. In reviewing this literature with colleagues in 2005 (that is, reading more papers in a month than you want to see in your entire life), we were able to identify some key leaders in the field, and we extended invitations to those leaders to present at the first congress. To our amazement, almost all agreed immediately to come, although some did not at first realize the connection that their research had to fascia, despite their personal use of manual therapies!

Those of you who attended the 2007 congress (or who own the DVD set) have heard Dr. Frederick Grinnell explain that, despite many years of receiving Rolwing® Structural Integration, he had no idea that there was any connection between manual therapy and his research until we invited him to be a keynote speaker. The importance of his studies into the shape of the fibroblast cell, which makes up fascia and which can change shape from dendritic to lamellar and back again, is demonstrated in other research presented at the 2007 and 2009 congresses. These related studies show that manual therapies<sup>(1)</sup>, acupuncture<sup>(2)</sup>, and stretching akin to yoga<sup>(3)</sup> all have an effect on fibroblast morphology, demonstrable at the cellular level.

Interactions between clinicians and researchers form the basis for the fascia congresses. Achieving this interaction was difficult at first, because the National Institutes of Health (NIH) conference grant that supported the first congress required that *all* clinical presentations be excluded from the NIH-sponsored portion of the conference. The research presentations were not “watered down,” and even many scientists struggled to keep up in talks outside their own specific area. Yet both groups hung in, expanding their perspectives until heads were about to explode. This clinician–scientist interaction was reported in *Science* magazine (“Cell Biology Meets Rolwing”<sup>(4)</sup>). I myself have watched the DVD recordings 4 or 5 times to develop a more complete understanding of the science and its application to my clinical practice.

When Dr. Peter Huijing agreed to host the second congress in 2009 at his university in Amsterdam, post-congress clinical workshops were added. Again, the congress DVD has allowed me to view the presentations again and again, each time learning something more.

I am pleased to inform the scientists and clinicians alike that this artificial separation will not be necessary at the third fascia congress in Vancouver, March 28–30, 2012. Its theme, Fascia Research—What Do We Notice? What Do We Know?, will incorporate dialogue between clinicians and scientists into the conference itself, with each presentation (whether by clinician or scientist) followed by both clinical and science summaries and questions. Post-congress workshops will provide even more opportunity to develop clinical implications or, for those so inclined, research proposals.

As in 2007 and 2009, keynote speakers will present entirely new material. Areas covered will range from basic science to clinical explorations, including mechanical loading and connective tissue change (Al Banes, PhD, University of North Carolina, NC, USA); fluid dynamics in connective tissue remodeling (Rolf Reed, PhD, University of Bergen, Norway); fascial anatomy (Carla Stecco, MD, University of Padua, Italy); repetitive motion disorders (Mary Barbe, PhD, Temple University, PA, USA); myofascial pain (Cesar Fernandez de las Penas, DO, Universidad Rey Juan Carlos, Spain); and clinical trials on manual therapies (Karen Sherman, PhD, University of Washington, WA, USA).

In a sneak preview of some of the information to be presented, I draw from a review of Dr. Reed’s studies

on loose connective tissue and his extrapolation of his findings to remodeling of the connective tissue structure of the heart<sup>(5)</sup>. He was initially interested in the involvement of the extracellular matrix (ECM) in fluid movement between peripheral blood vessels, interstitial fluid, and lymph. He found that this movement is mediated by the extracellular fiber network, with active involvement of connective tissue cells. In acute inflammation, the physical properties of loose connective tissue can change within minutes, resulting in an increase in fluid flow by a factor of more than 100. Reed proposes that “connective tissue cells apply tensile forces on ECM fibers that in turn restrain the under-hydrated ground substance from taking up fluid and swelling,” and he has identified a mechanism for collagen gel contraction mediated by fibroblasts and specific integrins and prostaglandins. Thus, loose connective tissue is neither static, nor passive.

Questions to be addressed in 2012:

- Dr. Reed has extrapolated these principles to the heart. Do other tissues respond in a similar way?
- Are there differences in other locations in the body according to the definitions of fascial tissues by Langevin and Huijing<sup>(6)</sup> presented at the last congress?
- How can these changes be measured, both at the cellular and the gross level?
- How is the fluid flow in the ECM affected by various therapies?

For those of you who have difficulty with the scientific terminology (and I admit to being one of those), let me illustrate the importance of fluid dynamics in another way. A few years ago, I was speaking with Emily Conrad D’Aoud, creator of Continuum Therapy. She related some success in working with a woman with paraplegia, and she shared with me a video she had prepared on the progress of this person, who was initially unable to move her legs, but through application of the undulating motion of Continuum, became able to crawl. To my eyes, the client was able to move her arms in such a way as to transmit forces through the fascia of the trunk to the lower extremities. However, Emily’s narration spoke of fluid flow, of aligning the client’s fluids with the universe. I invited her to present her findings to the spinal cord injury unit at my Veterans Administration Hospital, but insisted that she keep the sound turned off, because I could see no relevance in her fluid theories, and I wanted the team to focus instead on the movement she was able to create. I thought that the fluid theory would make the audience less receptive to the results. The team was duly impressed with her presentation. At the time, I thought that the many sessions of undulating motion were just repetitive practice, allowing the patient to learn some subtle ways of moving. However, in light of Dr. Reed’s

work, it seems that not only does fascial structure affect fluid flow, it also remodels in response to fluid flow. So perhaps what we were seeing is a restructuring of the fascial tissues in response to the undulatory movements of Continuum. Today, I think that Emily and her generation of clinicians who have developed fascia-oriented therapies are more correct than even they knew. Science is just beginning to catch up with their clinical observations.

In her brilliant paper in the 2009 fascia congress program book, Dr. Denise Hocking used intravital microscopy to show that ECM fibers stretch from muscle cells to nearby arterioles and that, at the beginning of a muscle contraction, these fibers open up a nitric oxide receptor that causes vasodilation and an immediate increase in blood flow to the exercising muscle<sup>(7)</sup>. Although this increase in blood flow with exercise was known by exercise physiologists even when I was studying in the 1970s, the mechanism long escaped any explanation. Scientists had showed that it was not mediated by the brain, by the spinal cord, or by any known hormonal mechanism. Only by considering the role of the ECM and by using advances in scientific techniques could this important physiological mechanism be explored.

Hocking concludes: “Our current study suggests an important new paradigm wherein tensile forces from actively contracting skeletal muscle alter the conformation of fibronectin fibrils surrounding the vascular wall and transiently expose matricryptic FNIII-1 sites that, in turn, initiate a biochemical signal and thus, signal a change in arteriolar diameter. The ability of tissue strain to alter the conformation of fibronectin and expose a cryptic cell-binding domain represents a simple, yet elegant means of converting a mechanical signal into a biochemical response. As such, this novel mechanotransduction pathway may be just one example of a common approach to transmitting mechanical forces, including pulsatile flow and shear stress, from the ECM to cells.” These extracellular fibers turn over rapidly, up to 50% in just 24 hours, again demonstrating an active ever-changing nature—and providing another potential mechanism of action for therapies.

To conclude, I share with you some questions prepared by Dr. Carla Stecco in preparation for her keynote address:

It is commonly thought that everything ... to be known about the human anatomy has already been revealed, and that anatomical variables present the only real news in this field. However, if we consider fascial anatomy, nothing is more far from the truth. Indeed, for many years, the fasciae have been considered only as a ‘white envelope for the muscles,’ and very little attention has been given to their macroscopic and histological anatomy.... We hope to find some answers to

several rather specific questions concerning fascial anatomy:

- Is deep muscular fascia a dense, regular connective tissue similar to an aponeurosis, as suggested by some authors, or is it an irregular loose connective tissue?
- Inside the deep fasciae can we recognize different regular sublayers, or are the fasciae composed of intertwined bundles of collagen fibers?
- How are the deep fasciae related with the underlying muscles?
- Does the superficial fascia exist? Does it have a specific structure, or not?
- How do the various fasciae appear under ultrasound, MRI, and CT scans? Could these instruments help us to understand the structure of the fasciae in living people? And what about the relationship between fasciae and surrounding structures?
- What is the role of the ECM, and in particular of the hyaluronic acid component?
- Could fasciae be considered elastic tissue? What is the percentage of elastic fibers within fasciae? Are there regional variations?
- Are fasciae innervated? And what type of receptors could be recognized within fasciae?
- What is the relationship between the fasciae and muscle spindles?
- Do the fasciae possess a basal tonus?
- Do fasciae have the capacity to actively contract?
- Do fasciae have a role in proprioception and in peripheral motor coordination?

Answers to these questions could contribute to clinician's understanding of the biomechanical behavior of the fasciae, their role in acute and chronic myofascial pain syndromes and of the real effectiveness of different therapies.

I encourage readers to review the definitions of fascia proposed by Langevin and Huijing, and to come to the congress with specific questions in which the specific fascial structure of interest is defined. As proposed by those eminent scientists, those structures are the dense connective tissue, the areolar connective tissue, the superficial fascia, the deep fascia, the intermuscular septa, the interosseal membrane, the periosteum, the neurovascular tract, the epimysium, the intra- and extramuscular aponeuroses, the perimysium, and the endomysium. The full article is available online at <http://www.ijtm.org/index.php/ijtm/article/view/63/80>.

And so, clinician and scientist alike, I invite you to share your observations and to participate in yet another exciting conference to be held March 28 – 30, 2012, in Vancouver, British Columbia, Canada. Registration for the Third International Fascia Research Congress (Fascia Research—What Do We Notice? What Do We Know?) opens March 1, 2011, with abstract submissions due by July 1, 2011.

Pre- and post-congress workshop descriptions, program details, and information on ordering the program books and DVDs from previous congresses can be found at <http://www.fasciacongress.org/2012>. The program book for 2012 will include full-text scientific papers (some new; some older, but forgotten) presenting entirely new research that expands the basis for the scientific study of fascia, and abstracts of the latest research to be presented in papers and posters. The program book for each congress includes materials that are entirely different from those in previous congress books; the content encompasses a careful selection of the latest scientific literature that is most important for fascia research (and which is still somewhat readable for people outside that immediate field). A summary of 2007 program book is available online<sup>(8)</sup>, as is a summary of the 2009 book<sup>(9)</sup>.

## CONFLICT OF INTEREST NOTIFICATION

The author declares that there are no conflicts of interest.

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