Mechanistic basis of manual therapy in myofascial injuries. Sonoelastographic evolution control

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Summary  The term myofascia is referred to the skeleton of muscle fibres organized as an interconnected 3D network that surrounds and connects the musculoskeletal system. Extracellular matrix muscle is relevant in tissue structural support and transmission of mechanical signals between fibres and tendons. Acute and chronic musculoskeletal injuries (muscle strain) are one of the major problems faced by those who practice any type of sport, regardless of whether they are professionals or amateurs. Therapeutic boarding is of uncertain value in most cases because there are many contributing factors such as type, severity, functional implication of the damaged tissue, progression or risk of relapse. Different studies suggest that the musculoskeletal cell matrix is essential for the development, maintenance and regeneration of skeletal muscle. In this article, we highlight the action of "non-contractile" structures, in particular the myofascial system or muscle fascia, which can be responsible for the pathophysiology and healing process of muscular injuries. Manual therapy plays a predominant role in the treatment of these types of injuries and is key in the process of obtaining a scar capable of transmitting mechanical information. The scientific basis of this process is described in this article. Through real-time sonoelastography we have accurate information regarding the current stage of the repair process and, thus, guide our treatment at all times. Some new
concepts are introduced, including local elasticity, the relationship between fascial pretension and the different stages of the physiological myofascia repair process, scar modelling technique, and sonoelastographic evolution control.

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**Muscular fascia or myofascia. Myofascial interfaces**

The term myofascia refers to the muscular supporting connective tissue, skeleton of muscle fibres, or musculoskeletal cell matrix. As such, myofascia consists of three distinct layers of dense connective tissue (starting from the most external surface to the most interior): (1) the epimysium, which covers the muscle surface; (2) the perimysium, which consists of fascicles below the epimysium; and (3) the endomysium, which further divides these fascicles into muscle fibres (Purslow, 2010) (see Fig. 1). It is therefore possible to find an injury not just on a muscle fiber level but also at the endomysium, perimysium and epimysium level. Therefore, although an isolated continuity solution at some fascial septa is possible, it is evident that muscular fiber strain with its corresponding epimysium is inevitable. In other words, when there is a mechanical overcharge at this level, should we talk about muscular injuries or myofascial injuries? In this article, we highlight the action of "non-contractile" structures, the myofascial system or muscle fascia, and their decisive participation in the physiopathology and healing process of muscular injuries (Kaariainen et al., 2000).

Kjaer et al. (2006) suggest common signaling pathways that stimulate contractile and extracelular matrix (ECM) components. In fact, in developing skeletal muscle, an important interplay between muscle cells and ECM can be present. In addition to the fascial layers described above, recent scientific research has shown the existence of interfaces or connective bonds precisely connecting the fascial tunics between them and the muscle's cells. These links have been traditionally omitted in conventional descriptive anatomy because they are obliterated in normal dissections. In studies from Passerieux et al. (2006) loops were found between the perimysium and epimysium and named *epi-perimisial junctions*. Muscle physiology advances suggest that the perimysium plays a role in the cross-transmission of contraction forces (Huijing, 2009). This *interfascial trabecular system* may provide mechanically competent links for transmitting forces from the periphery to the inside of the muscle. In addition to the above loops, links called perimysial—perimysial *junctions* exist that facilitate the longitudinal slip of fascicles between themselves (Passerieux et al., 2007). However, the more relevant junctions appear to be the adhesion between the perimysium and the myofiber endomysium. These structures are introduced into the honeycomb structure formed by the endomysium and make their way to the muscle cell surface, forming "perimysial junctional plates" (for more information please refer to Passerieux et al., 2007). The importance of these structures lies in their ability to penetrate the inside of the muscle cell and stop at places with a high concentration of myonuclei and mitochondria. Thereby, as described later, external mechanical forces that could affect gene expression, are directed into the muscle cell and the musculoskeletal cell matrix (Kjaer, 2004).

**Myofascial repair. Local elasticity concept**

The muscle wound, healing after myofascial injury (muscle strain), is a complex process including three overlapping phases: degeneration and inflammation, muscle regeneration and fibrosis (Jarvinen et al., 2000; Serrano and Muñoz-Cánoves, 2010). Following acute tissue injury, infiltrating inflammatory cells and resident stem cells orchestrate their activities to restore tissue homeostasis. It is known that when myofascia is damaged, myogenic satellite cells activate, divide, differentiate, and finally fuse with healthy muscle fibres to repair the injured tissue and to enhance hypertrophy of muscle fibres (Jarvinen et al., 2005; Filippin
et al., 2009). Other non-muscle stem cells such as hepatocyte growth factor (HGF), fibroblast growth factors (FGFs), transforming growth factor–betas (TGF-betas), and others, are released during muscle repair and guide muscle regeneration (Smith et al., 2007; Karalaki et al., 2009).

At the same time, the myofascial repair process leaves behind a connective tissue scar in the injured muscle. Fibrosis can be defined as the replacement of the normal structural elements of the tissue by distorted, non-functional and excessive accumulation of fibrotic tissue (Diegelmann and Evans, 2004). Thereby, because of an excessive proliferation of collagen, dense scar tissue can form within the injured muscle. Unfortunately, the gradual development of fibrotic scar tissue within the injured area hinders muscle regeneration, and ultimately leads to incomplete functional recovery in relation to decrease of contractile function and muscle extensibility, along with an increased risk of reinjury (Ciciliot and Schiaffino, 2010).

The fibrotic phase of the repair process depends on general factors including the type, severity, location and functional implication of the damaged myofascia; as well as the appropriate inflammatory response, and treatment protocol, including early mobilisation (dense scar formation in the injury area may prohibit muscle regeneration), immobilisation (improves the penetration of muscle fibres through the connective tissue but their orientation is not parallel with the uninjured muscle fibres) or mobilization started after short immobilisation (better penetration of muscle fiber through connective tissue and better orientation of regenerated muscle fibres) (Jarvinen and Lehto, 1993).

More specifically, in the balance of the processes of remodelling and fibrosis, the influence of particular growth factors, including transforming growth factor–beta1 (TGF–beta1) is relevant. This is a potent stimulator of collagen proliferation, leading to the formation of fibrotic scar tissue after injury (Desmouliere et al., 1993). Tomasek et al. (2002) suggest that TGF–beta1 induces myofibroblastic differentiation of fibroblasts both in vitro and in vivo. The resulting over-growth of myofibroblasts is responsible for the ensuing excessive accumulation of fibrotic tissue. Interestingly, TGF–beta1 can induce the differentiation of myogenic cells into fibroblasts in injured skeletal muscle, increasing skeletal muscle fibrosis (Li et al., 2004; Cencetti et al., 2010).

Furthermore, by means of mechanical stimuli being converted into chemical activity (Ingber, 1997) studies have shown that the mechanical properties of a cell’s microenvironment can have a great impact on cell structure and function (Engler et al., 2006). One of the recent paradigm shifts in stem cell biology has been the discovery that stem cells can begin to differentiate into mature tissue cells when exposed to intrinsic properties of the ECM, such as matrix structure, elasticity, and composition (Reilly and Engler, 2010). Local elasticity refers to the ability of a tissue to return to its original length after a stretch, measured in kPa in a defined area of soft tissue. In this context, recent findings (Wipff and Hinz, 2009; Hinz, 2009; Meyer-ter-Vehn et al., 2011) suggest that TGF–beta1 activation is partly controlled by tissue stiffness and myofibroblast contractile forces. This mechanical cue for myofibroblast differentiation establishes a vicious cycle because the excessive extracellular matrix secreting and remodelling activities of myofibroblasts are the cause and effect of further connective tissue contracture and stiffening (Hinz, 2009). In other words, fibroblasts can alter their morphometry and gene expression profile when grown on chemically equivalent surfaces, with different rigidities (Solon et al., 2007).

Fascial pretension and tensinal homeostasis

Development of the teseogy theory allows us to define the relationship between mechanics and biochemistry at the molecular level (Ingber, 2003). Teseogy, which is an abbreviation for “tension integrity”, is an architectural concept developed by Fuller (1961). Teseogy structures are based on the presence of discontinuous compression elements that balance the force generated or received by continuous tension elements. A significant characteristic of this teseogy model is the tensile prestress (pretension) of continuous tension elements, that ensures constant structure stability against both compression and traction forces (Ingber, 2008a). This pretension can be variable, and it varies, based on different mechanical demands, such as absorption and management of forces. Moreover, in living organisms, all components are arranged according to a hierarchical organization that spans from the helical DNA structure on the molecular level, to the cellular and nuclear cytoskeleton on the microscopic level to the musculoskeletal system on the macroscopic level, and finally to the entire organism. The bioteseogy model incorporates the compression-resistant bones of the skeleton and the surrounding tension-generating muscles, and tension-resistant fascia (Ingber, 2008a). This is a physically integrated framework that supports the weight of the body, allowing rapid adjustment to external forces, while permitting freedom of movement (Ingber, 2008a).

One of the most significant characteristics of this biotenseogy model can be fascial pretension that ensures constant structural stability. This notion of acquired pretension raises the following questions: in the healing process, could we consider fascial pretension increase to be a local etiological factor of increasing the fibrotic phase? In other words, what happens in the healing process when we start with excessive pretension of the myofascia? Additionally, can we measure the local pretension increase?

In this context, if physical forces play an important role in tissue development and remodelling it is necessary to know how fibroblasts respond to different pretension states in the extracellular matrix. The biotenseogy model can be applicable at the cellular level, considering the cell as a pretension structure with cytoskeletal filament networks that mechanically couple specific cell surface receptors, such as integrins, to matrix scaffolds (Ingber, 1998). During the in vitro culture of fibroblasts, in 3D collagen matrices with different states of substrate pretension, it was observed that fibroblasts have a high level of mechanical sensitivity (Rhee, 2009). Recent studies on fibroblasts in three-dimensional (3D) collagen matrices have revealed the importance of biomechanical conditions, in addition to biochemical cues, for cell shape, signaling and migration (Solon et al., 2007; Langevin et al., 2010).

When cultured on 3D high-tension matrices, fibroblasts exhibit stress fibres, permanent focal adhesions and signal...
activation of focal adhesions. In the same culture with 3D high-tension matrices, fibroblasts can adopt a laminar form by flattening and spreading (Gabbiani, 2003; Hinz, 2007; Chiquet et al., 2009). This laminar form will have important implications for cellular function by activating a proliferative phenotype with high level of collagen biosynthesis activity (Grinnell, 2003; Kjaer et al., 2006). Additionally, conjunctive presence of mechanical stress (high-tension matrix) and active transforming growth factor β (TGF-β) is essential to convert fibroblasts into contractile myofibroblasts, which contribute to the reconstruction of injured tissue by secreting new extracellular matrices, and by exerting high contractile force (Hinz, 2007). Conversely, fibroblasts grown on low-tension matrices (relaxed) adopt a dendritic form, in which the dendritic extensions expand to allow for physical communication among the fibroblasts. Low-tension cultures experience changes in cell function in the same way that changes occur in cell function and shape in 3D high-tension matrices cultures. In this case (low-tension), biosynthetic activity is inhibited, leading to a quiescent cellular state. Additionally, this relationship between fibroblasts and the extracellular membrane through focal adhesions that is mediated by integrin receptors involves a bidirectional relationship precisely conditioned by pretension (Grinell, 2008).

**Manual therapy in myofascial injuries**

If fibroblast behavior depends on surrounding mechanical processes, it is relevant to understand these processes as fully as possible, when treating myofascial injury. Most importantly, can pretension states be modified by manual therapy? Could cell and tissue physiology be influenced, using fascial therapy? And moreover, in the clinical context, to guide a correct treatment in myofascial injury, how might we measure the relevant local elasticity changes in myofascial tissues, before and after treatment?

In normal tissue response injury, wound healing is directed by chemical and mechanical signals. Once the regenerative phase has started, isometric tension is generated in the repair area that stimulates a local increase in collagen biosynthesis. Finally, it progresses to the contracture phase, which can be induced by myofibroblast contractile activity. When this final phase persists over time, it can create pathological situations if the contraction produced by the myofibroblasts smooth actin alpha-1 fibres also persists (Grinell, 2003).

It is therefore essential to return the matrix from a high-tension state, to a low-tension one. It might be possible to induce myofibroblast apoptosis, or to halt the transformation to myofibroblasts, although this hypothesis has not yet been proven (Hinz, 2007). Similarly, any manual action that returns the matrix to a low-tension state might be able to inhibit biosynthetic activity by the fibroblast (excessive pathologic collagen crossovers), which would disturb mobility between the fascial interfaces and, ultimately, would lead to hypertrophic scars.

In order to reduce fibrosis and facilitate muscle regeneration various techniques can be used to inhibit growth involved in the development of scar tissue. In this direction, some biologic approaches such as relaxin treatment (Negishi et al., 2005) or the use of antifibrosis substances such as decorin (Fukushima et al., 2001), gammainterferon (gamma INF (Foster et al., 2003) and others such as suramin (Chan et al., 2003) could inhibit TGF-β1, decrease myofibroblast proliferation, and promote proliferation and differentiation of myofibroblasts, so improving muscle strength in vivo. However, clinical trials are required in order to safely include this treatment in the standard management of muscle injuries.

Grinell (2008) has reported that: “physical manipulation of fascia has the potential to change the cell-matrix tension state and also may influence localized release of cellular growth factors. As demonstrated by our research on fibroblast–collagen matrix interactions, such changes could lead to profound and rapid modulation of structural, functional and mechanical interactions between fibroblasts and the extracellular matrix and, as a result, contribute to the reorganization of fascia that results from bodywork practice”.

According to different authors, the relatively low level of forces used by manual therapists would be insufficient to cause microfailure of the collagen, except perhaps in very thin or loose tissue (Chaudry et al., 2008). Thus it seems probable that a large part of the benefits of myofascial therapies may be due to neurophysiological effects (Cantu and Grodin, 2001). In this sense, Schleip (2003) suggests that, in fascial techniques, the manual induction process would be able to cause deep modulations, at different levels of the nervous system, through stimulation of interstitial receptors present in fascia, leading a global decrease in sympathetic tone that would produce a local vasomotor reaction, modifying, in short term the tissue viscoelasticity (thixotropic reaction).

Other authors suggest the possibility of causing structural changes in fibrosis and fascially restricted areas/ myofascial restricted areas, through the decrease of cross-links between collagen fibres, increasing gliding functions between fascial layers, as well as inducing microfailure of collagen fibrils, as their tensile strength is exceeded. All of these changes might be associated with the transformation of ground substance, from a densified state (gel) to more fluid (sol) state (Simmonds et al., 2012; Tozzi, 2012) (Fig. 1).

With these concepts in mind, the authors of this paper have developed a scar modelling technique (Table 1), that attempts to reverse the matrix state from high to low tension, with controlled mechanical stimuli through the combined use of torsion, shear, traction, axial and compressive vectors on scar tissue. All this is done in order to generate a maintained tension, against a barrier, until a release of tension is perceived. (Pilat, 2003) (Fig. 2a–c). We suggest that the pursuit of tensional homeostasis by the therapist’s manual treatments, guided by the liberation of “jumps” of accumulated elastic energy in the interfaces, could cause the 3D reorganization of fascial interfaces on a macroscopic level (through junctions described above as fascial loops) (Passerieux et al., 2006), resulting in tensional normalization on the microscopic level (tensional reharmonization between the cytoskeleton and extracellular membrane through receptor integrins). This reharmonization would act to normalize cell function and provide medium-term remodelling of the extracellular matrix. Obviously, further clinical trials are necessary to test this hypothesis.

Finally, in clinical practice, it is important to be alert to the relationship development of hypertrophic scars and high-
risk re-injury, and poor treatment outcomes (Engebretnsen et al., 2010). Eventually, the scar will have its own char- acteristics determined by its degree of resistance and elas- ticity. These characteristics contribute to the quality of the scar, which is key in maintaining the integrity of force transmission. It must be strong enough to transmit forces and to sustain the loads of contraction, and elastic enough to absorb forces and to prevent tearing under externally applied strains (Purslow, 2002). At this point, Heiderscheit et al. (2010) suggest the relevance of fascial stiffness increase as an etiological factor in muscle injuries, since it is known that fascia demonstrates viscoelastic behavior (Yahia et al., 1993; Chaudry et al., 2008) and plays an important role in transmitting and deforming mechanical forces between muscles (Huijing, 2009). So, in this context, could the pretension states related to hypertrophic scar and fascial stiffness, be modified by fascial therapy to decrease risk of re-injury, while improving myofascial force transmission? If so, how might we measure local chronic fibrotic scars?

Real-time sonoelastography and sonoelastographic evolution control

We propose the use of real-time sonoelastography (RTSE) as an image test to evaluate our hypotheses. Ophir et al. (1991) described the principle of strain imaging (elastogra- phy). In order to reduce time consuming calculations Pesavento et al. (2000) developed a fast cross sectional technique based on real-time elastographical imaging that can provide very rough information about the assessment of elasticity of biological tissues. Palpation is a well-known subjective method used by physicians to assess organs stiffness (Garra, 2007). In our context, ultrasound elasticity imaging can be considered as an extension of the ancient art of body palpation that can provide very valuable objective data in prevention and treatment of myofascial injury in order to: (a) measure local fascial pretension as a relevant etiological factor of increasing fibrotic phase in myofascial wound healing; (b) measure the mechanical dose we apply to the tissues and their reaction to the myofascial therapy, before and after treatment and (c) measure fascial stiffness by hypertrophic scars, as a rele- vant etiological factor of muscle injury and relapse.

RTSE is based on Young’s, or shear elasticity modulus measurements, and the principle that each biological tissue has its own elastic characteristics, quantified according to the formula for the Young’s modulus, which relates the compression of a material when an external pressure is applied, to its deformation (Sarvazyan, 2001).

In recent years, elasticity imaging has attracted attention as a technique that directly reveals the physical property of tissue, making it possible to determine the change of tissue hardness caused by pathology (Frey, 2003). The original application of RTSE was to detect and classify tumors, based on the fact that these are significantly harder than surrounding tissues. Thus, when a mechanical compression or vibration is applied, the tumor (hard tissue) deforms less than the surrounding tissues (Bercoff et al., 2003).

This information is represented on a monitor as a chromatic spectrum called an elastography image (elastogram) over the conventional ultrasound image: areas in red represent the most elastic areas (fluids), areas in blue represent the most rigid ones (bone), while areas in green can represent firm areas with intermediate consistency/stiffness (Lalitha and Balaji, 2011). In addition, it is possible to compile an elastography score, based on a 5 point scale according to the different colors displayed in the elastogram (minimal score — 1 — for scars with elasticity similar to the surrounding myofascial tissue and maximal score — 5 — for hypertrophic scars with no deform- ability). In this way it is possible to achieve a subjective assessment of elasticity tissue (Wang et al., 2010). In brief: sonoelastography is an echogram that is colored according to the elasticity of the depicted tissue (see Figs. 3 and 4).

Some elastography equipment allows calculation of the strain ratio. For example, by choosing a selected damaged area, a region of interest (ROI), a comparative relationship is established, based on a constant hardness numerical value (bone to lesion strain ratio) or constant soft numerical value (fat to lesion strain ratio). In this way, it is possible to obtain objective numerical relative elasticity data by evaluating the compliance difference between the local lesion and the surrounding tissues (Cho et al., 2010) (see Fig. 5).

Although RTSE is not yet usually used in routine clinical physical therapy practice, it has been shown to be useful in the differential diagnosis of breast cancer, showing a high sensi- tivity, specificity and histopathological correlation in charac- terizing malignant lesions of the breast (Andreea et al., 2011). However, variability in specificity between sites and observers is possibly due to individual technique differences in per- forming elastography and measuring lesions (Barr et al., 2012). In addition RTSE has also been used to diagnose different pathologies, including thyroid (Lim et al., 2012), cervical (Thomas et al., 2007) and prostate (Walz et al., 2011).
cancers, as well as degrees of hepatic fibrosis in patients with chronic liver disease (Rath et al., 2011) (see Fig 6).

In recent years, RTSE has emerged as a way of characterizing the mechanical properties of the tissues of the musculoskeletal system, for example showing good feasibility and reproducibility in assessment of lateral epicondilitis (De Zordo et al., 2009a, b). Other authors suggest the relevance of RTSE in measurement of elasticity in healthy achilles tendon (De Zordo et al., 2009a, b; Drakonaki et al., 2009) as well as in achilles tendinopathy (De Zordo et al., 2010; Sconfienza et al., 2010) and myofascial trigger points (Sikdar et al., 2009).

The authors of this paper propose the use of fascial therapy (scar modelling technique) and RTSE, in combination, for appropriate assessment, treatment and monitoring of myofascial injuries. Therefore, in fascial therapy, when we manually evaluate the status and fascial tonicity of the affected tissues, we usually do a “fascial quick scan”. In other words, we generate a mechanical wave manually, with the intention of spreading it through tissues. The wavelike elastic rebound is palpated and evaluated by the therapist’s hand. This provides a subjective evaluation, based on the individual’s years of clinical experience and study. When performing an assessment with RTSE we use a similar approach by generating a controlled deformation through the probe, to study the elastic behavior based on its transmission through tissues. This information is analyzed by the equipment software, which then provides a color-coded elasticity map containing objective and storable information (elastogram). Moreover, as described above, with a strain ratio, we can quantitatively objectify the elastic behavior of myofascial tissue during the repair process, thereby evaluating the effects of manual therapy during the treatment process (see Figs. 7 and 8).

We believe that the elasticity measurement procedure that we have developed, named sonoelastographic evolution control (SEEC), is highly applicable for clinical use. We propose the following steps.

**Palpatory identification**

Through palpation, the therapist finds a possible scar or hypomobile area by attempting to relate it to the information that obtained when taking the case history.

**Ultrasound location**

Although we utilize ultrasonography (US) preferentially in the transverse and longitudinal cuts, we can also use some

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**Figure 2** (a) Scar modelling technique based in axial and compressive vectors. (b) Contact phase: initial vector compression maintained by the finger flexor tone of the second, third and fourth fingers. (c) Stimulation phase using spiroid/circular vector to generate a maintained tension against a sense of resistance.

**Figure 3** Elastography evaluates the deformation produced by an injury. Tissue hardness is indicated by a color scale.
dynamic applications to study movement behavior. Once we have localized the area of interest, we draw a mark on the skin framing the probe’s margins, and then collect the ultrasound images. Additionally, based on the work of Langevin et al. (2009), using ultrasound we assess not only inflammation and fibrosis, but also fascial thickness. It seems that repetitive motion and mechanical stress over hyperfibrotic scars, following myofascial injury, may result in excessive deposition of collagen, increasing fibrosis and adhesions between fascial interfaces. In order to assess the

Figure 4  Sonoelastography in real-time is a non-invasive method that utilizes a system to obtain images using an ultrasound device combined with a pressure sensor. This allows identification of the elasticity of a tissue region that has a different elasticity, compared with the surrounding tissues. Tissue hardness is indicated by a color scale.

Figure 5  Strain ratio study in different regions of interest of the same elastogram with regard to a constant reference value (0.01). (A) 61.71; (B) 39.99; (C) 8.60.
dynamic performance and force transmission of the myofascial system, we suggest the use of dynamic ultrasound as an image test. This is capable of providing valuable information regarding myofascial interface sliding capacity, when subjected to active or passive movement, movement against resistance, or manual treatment by the therapist.

Elastography mode

The elastography mode is used to define the elastic behavior of the intrinsic elastic characteristics of the tissues in the studied area. What is being searched for is continuity or, if this is not observed for the loss of homogeneity in the tissue’s color scale, relative to surrounding tissues that are histologically identical (see Figs. 9 and 10). In current clinical practice, it is sometimes difficult, or even impossible, to distinguish pathological tissue from surrounding healthy tissue on conventional ultrasound. Damaged inflamed or fibrotic areas often present the same echogenicity. Moreover, it is well known that inflammation and fibrosis lead to changes in tissue elasticity. Therefore, because distinct tissue softening, linked to musculoskeletal disorders, can be detected in real-time strain imaging (De Zordo et al., 2009a, b), we propose the use of RTSE as an ideal method for objectifying the type of injury, and the state of repair. Further, according with our hypothesis, we think RTSE could be an ideal method for objectifying the status changes produced in soft tissues, after fascial manipulation, providing information regarding the daily evolution of muscle injuries, and the responses to manual therapy. As explained below, this might allow application, in an effective manner, of the type of treatment required. This proposes that, depending on the tension degree in the damaged area (based on the elasticity curve concept), progress can be judged as the matrix returns from a high-tension state to a low-tension one, or vice versa.

Measurement

It is possible to obtain an elastography score and/or a strain ratio of a studied area — a numerical value of the relative elasticity of a region of local lesion (scar), relative to surrounding tissues. Additionally, histograms can provide graphical representations, showing a visual impression of the distribution of numerical data. The next step is treatment. Scar modelling technique is applied, according to the application principles described above, until the therapist produces a “change of status”.

In our clinical practice we then repeat the process, or start from the elastography mode, where we obtain new recordings. We compare the new results to those obtained initially. In this way, we can study elastic behavior of tissues over time with respect to the reference values we have accumulated.

Discussion

In this article, we describe our clinical experience in the treatment of muscle injuries with manual therapy and, specifically, with scar modelling techniques. To assess the
type of muscle injury, in clinical practice different imaging techniques are used, such as ultrasonography (US) and magnetic resonance (MR). Sometimes, ultrasound cannot distinguish pathological tissue from healthy tissue because injured areas, involving inflammation or fibrosis, present the same echogenicity. While some authors suggest the important role of MR to evaluate the risk of recurrent muscle injury (Koulouris et al., 2007), others have found that the MR use is limited in identifying individuals at risk for reinjury (Heiderscheit et al., 2010). In this article, we propose the use of RTSE and, in addition, dynamic ultrasound, not only to improve the clinical use of diagnostic imaging techniques in muscle injuries, but also to direct in a very accurate way, the kind of treatment required in relation to the mechanical properties of the injured area, as well as to measure the mechanical response of myofascial tissue following fascial therapy. In addition, the use of RTSE can offer relevant clinical information, facilitating control and decreasing the high risk of relapse, secondary to a hyperfibrotic dense scar.

It is known that differences in extracellular matrix elasticity can be a determinant of healing processes in muscle regeneration and during the fibrotic phase of muscle repair. In an initial phase there is a need to maintain the initial reflex contracture as an ideal tension habitat to increase biosynthetic activity, and movement of fibroblasts to the scar region. Additionally, the use of biological agents such as autologous growth factors (platelet-rich plasma) can accelerate the healing process, releasing high doses of growth factors to damaged tissue (Creaney and Hamilton, 2008). Recent findings suggest the important therapeutic potential of intramuscular injection of muscle-derived stem cells that can improve and accelerate skeletal muscle healing, by increasing angiogenesis and decreasing scar tissue formation (Ota et al., 2011; Gharaibeh et al., 2012). In the physical therapy context, with a focus on muscle remodelling, different authors (Valero et al., 2012) suggest that eccentric strength training can stimulate the activation and proliferation of satellite stem cells that participate in the skeletal muscle regeneration. In order to obtain complete restoration of the pre-injury status of damaged tissue, excessive collagen proliferation should be avoided, in order to prevent...
formation of hyperfibrinous scarring, with high risk of relapse (Desmouliere et al., 1993). Since scar tissue is stiffer than the contractile tissue it replaces and can alter the mechanical environment of muscle fibres, evaluation of the mechanical properties of the extracellular matrix is suggested. It is known that activation of TGF-β1 and excessive extracellular matrix secreting and remodelling activities of myofibroblasts can, decisively, increase skeletal muscle fibrosis (Li et al., 2004) — processes that are partly controlled by the mechanical properties of the microenvironment cells, and tissue stiffness (Wipff and Hinz, 2009; Hinz, 2009).

The authors propose the need of modifying what we call "elasticity curve" that registers the myofascial tissue during the repair and fibrosis processes (see Fig. 11). In those cases in which a scar shows significantly higher stiffness rates than surrounding tissues, it would be necessary to control the use of treatment protocols such as eccentric exercise, that can potentially increase local pretension in the injured area, and favor an increase of TGF-β1, related not only to muscle repair but also to fibrosis increase. Also those techniques based on biological agents such as autologous growth factors (platelet-rich plasma) that, as cited previously, can be related to local release of growth factors that lead to significant collagen proliferation. On the contrary, it is proposed, in this context (high-tension matrix) the use of scar modelling techniques that encourage the extracellular matrix muscle to return from high-tension state to low-tension one. To confirm this proposal we suggest the use of RTSE and SEEC, including strain ratio and elastography imaging (elastogram) as clinical tools that allows the evaluation of changes in local elasticity, secondary to treatment.

During the evolution of the healing process, following a muscular injury, the use of RTSE can offer valuable information, allowing adaptation of the treatment plan to the recovery process — from the outset until the return to physical activity. In other words, early mobilization (exercises with appropriate loading starting at 3–6 days post-injury) is needed to optimize the regeneration process, and to recover the extensibility and strength of the injured...
skeletal muscle to pre-injury levels. In contrast, a minimum period is required to produce a scar of sufficient strength and elasticity to bear the forces induced by remobilisation, without re-rupture (Jarvinen et al., 2007). Thus, the parameters usually used to assess treatment advice in relation to evolution of the patient, image diagnosis with MRI, or ultrasound tests, of strength and extensibility, and the manual therapist’s perceptions, and the sensations described by the patient, could be usefully supplemented with RTSE and SEEC, including strain ratio and elastography imaging.

Moreover, the high rate of recurrent injuries suggests that our current understanding of myofascial injury and reinjury risk may be incomplete (Opar et al., 2012).

The multifactorial origin of myofascial injuries is related to different risk factors including, among others, anatomical factors, such as biarticular organization, fiber-type distribution, muscle atrophy, strength imbalances and fatigue (Opar et al., 2012). Further, previous injury and formation of dense fibrotic scar tissue can be a significant injury and reinjury risk. In this way dense scar tissue can be formed within the damaged muscle, hindering muscle regeneration and, potentially, provoking a decrease of contractile function and muscle extensibility, as well as alteration of the transmission of forces from the myofascial tissue to the tendon (Ciciliot and Schiaffino, 2010). In this context, recognising the secondary myofascial stiffness of hypertrophic scars as a relevant etiological factor of muscle injury and relapse, we propose the use of RTSE and SEEC, including strain ratio and elastograms, to control the resistance and elasticity degree of the scar tissue. We also propose that a direct relationship must exist between local elasticity in the scar and physiological force transmission, which means that when there is higher sonoelastographic similarity between the local elasticity of the scar and the elasticity of the surrounding tissues with identical histological characteristics, there will be better physiological transmission of mechanical forces. Thus, when we find higher similarity between local elasticity and the surrounding tissues, the chances of relapse can decrease proportionately.

In connection with the ability of the RTSE to assess mechanical changes, before and after treatment, in our initial experience we have found that short-term changes may be related to an important secondary vasomotor response to treatment (thixotropic reaction). Subsequently, in the short term, a normalization of the degree of elasticity is commonly observed (see Fig. 12). In clinical settings, in relation to chronic scarring, we have observed that the greater the mechanical response that a scar has to treatment, the better prognosis there will be.

![Figure 13](attachment:image.png)

**Figure 13** Elasticity curve with strain ratio measurements of a scar after 3 months of evolution. It is possible to see differences between pre-treatment and immediate effects in elasticity changes (thixotropic reaction) after 50 min of scar modelling technique. Interestingly, structural changes in long-run elasticity can be seen after 4 days, particularly after a second treatment with scar modelling technique.
regardless of the adjustment of the elasticity index in the short term. It is important that we develop protocols to evaluate elasticity long-term. The use of RTSE, secondary to treatment, allows the assessment of structural changes in the medium and long term, unrelated to the immediate thixotropic reaction (short-term effect), involving stiffness decrease and tension normalization in the scar environment (i.e., the long-term effect) (see Fig. 13). In our clinical experience an interesting correlation is commonly observed between the structural changes noted after treatment in the medium term (4–8 days), and the perceived self-improvement of the patient, regarding increased extensibility, feeling of strength and functional capacity (Fig. 13).

Several limitations need to be addressed. A number of studies have investigated the sensitivity and specificity of RTSE in assessing the mechanical properties of RTSE involving various tumor types, such as breast tumor (Andreea et al., 2011). In parallel, the evaluation and treatment of musculoskeletal injury, for example, lateral epicondilitis, has shown a high reproducibility (De Zordo et al., 2009a, b). Furthermore, in the clinical context of myofascial injury it has been demonstrated that the strain ratio is signally lower in the regeneration-fibrotic phase, and in hyperfibrotic scars in relation to surrounding tissues. Moreover, in clinical contexts we have observed a direct relationship between elastographic changes after fascial therapy and improvements in clinical symptomatology, expressed by patients. However, high-quality clinical trials, with larger samples, are required to prove the sensitivity, reproducibility, intra-interobserver reliability, accuracy and validity of use of RTSE in assessment, treatment and monitoring of myofascial injury. Additionally, in clinical practice, in a grade I (mild) muscle strain there may have certain limitations in specifically characterizing and grading the elasticity of small scars with similar elasticity to the surrounding myofascial tissue. In contrast, the greater the degree of injury and subsequent repair process (dense hyperfibrotic scarring), the more efficient is the ability of RTSE to detect differences in elasticity between the scar and peripheral tissue. Further, because in our current clinical practice we use manual compression this technique can be seen to be operator-dependant. However, new equipment, with controlled tissue compression systems and image segmentation, could improve elasticity image capability. Also, in our clinical experience it is sometimes difficult to visualize the region of interest (ROI) after fascial techniques. This may be because of the 3D tissue reorganization related to small “landslides” involving the release of accumulated elastic energy in the fascial interfaces.

Conclusion

Skeletal muscle injuries cause loss of activity and increased risk of recurrent injury. We propose the use of RTSE and fascial therapy (scar modelling technique), in combination, for appropriate assessment, treatment and monitoring of myofascial injury. The use of RTSE allows objectifying of the type of injury, and the state of repair. Further, we think RTSE could be an ideal method for objectifying the status changes, produced in soft tissues after scar modelling technique, allowing treatment to be directly linked to the degree of tension in the damaged area. In addition, randomized clinical trials are required to check the potential of RTSE to evaluate and monitor myofascial injury.

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References


