Myofascial Trigger Points: Pathophysiology and Treatment with Dry Needling

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Introduction

The aim of this article is to review the possible mechanisms related to the pathophysiology and treatment of myofascial trigger points with the technique of dry needling. Muscles in general and myofascial trigger points (MTrP’s) are a major source of persistent pain (Travell 1983). Myofascial pain is often associated with MTrP’s in muscles, tendons and fascia (Sola 1990, Simons 1999). It is characterized by regional muscle pain and described as dull or achy.

Definition of a Myofascial Trigger Point (MTrP)

The MTrP has been defined as a highly localised and hyper-irritable spot in a palpable taut band of skeletal muscle fibres (Travell 1983). It is associated with a number of important characteristics (Travell 1983, Simons 1988, Sola 1990),

1. Compression may elicit local or referred pain that is often familiar to the patient.
2. Rapid compression or needling of the muscle taut band may elicit a local twitch response (LTR). This is a brisk contraction of muscle fibres around the taut band.
3. Restricted range of stretch and an increased sensitivity to stretch may cause tightness and shortening of the involved muscle.
4. The muscle with a MTrP may be weak due to pain but there is usually little noticeable atrophy.
5. There may be associated autonomic phenomena such as an increased sudomotor and pilomotor response.
6. The MTrP may be active or latent. An active MTrP is one with spontaneous pain or pain response to movement, while a latent MTrP is sensitive only when directly stimulated.

It is known that a significant number of adults have latent trigger points, that is trigger points that only elicit pain on direct compression rather than spontaneously (Sola, 1990).
Location of Myofascial Trigger Points (MTrP’s)

All active and latent MTrPs, but not all acupuncture points, are tender. Tender and clinically relevant acupuncture points are called ‘Ah-Shi’ points. In Chinese, ‘Ah-Shi’ means ‘Oh Yes!’ (That’s the right spot). When the point is pressed the patient recognises their pain (Hong 2000). Consideration of MTrP location must therefore provide an adequate explanation for the possible mechanisms and structures responsible for this tenderness.

There have been a number attempts to provide a scientific explanation for the location of the traditional acupuncture point. The scientific dissection of the acupuncture point is an obvious first step in an attempt to find an explanation behind the mechanism of acupuncture. In 1966 the Austrian histologist Kellner found a statistically significant increase in the amount of sensorial elements at many although not all acupuncture points as compared to non-acupuncture points (Kellner 1966).

In 1987 the German anatomist Heine described a vasomotor-nerve bundle passing through the superficial fascia in 80% of all acupuncture points (Heine 1993). He put emphasis on the fact that, together with this bundle, a cylinder of proteoglycan containing ground substance protrudes towards the surface. The cylinder is topped by a denser proteoglycan layer. Proteoglycans, blood vessel, nerve, sensory receptors and muscle constitute a functional unit and interact. This ‘point-organ’ according to Heine has possibilities related to the proteoglycan network over and above the generally accepted nerve and blood vessel related functions (Heine 1993).

Recent work examining rabbit skeletal muscle has identified tender, taut bands similar to that found in human muscle. When these bands are compressed the rabbit will withdraw as if it has suffered pain or discomfort. Similar behaviour is not noted when other sites are palpated (Hong, 1994a). When these sensitive sites are stimulated mechanically with a needle a local twitch response has been observed. These twitch responses are more easily elicited at the sensitive spot in the muscle than at any other site. The local twitch response produced in rabbit muscle appears to be the same as that in human muscle when examined with electromyographic recording as well as when observed clinically, and is probably mediated through a spinal cord reflex (Hong, 1994a).

In rabbit skeletal muscle active MTrP’s were found to be mainly in the endplate zone (EPZ), an area associated with the motor point of the muscle (Simons, 1995). The total area of the EPZ is however much more than just the motor point itself (Fig. 1). A motor point being defined as,

‘The region of skin where an innervated muscle is most accessible to percutaneous electrical excitation at the lowest intensity’

In most muscles the EPZ corresponds to an area of tissue running transversely across a muscle in a direction which is perpendicular to the direction of the muscle fibres in the mid region between the muscle’s proximal and distal attachments (Coers 1959). In
general the motor point tends to be located near the centre of the EPZ and corresponds to the greatest concentration of endplates.

**Fig.1 Relation of Endplate zones (EPZ), Endplates and Taut Bands**

Other researchers have demonstrated that acupuncture points are identifiable by measuring skin resistance to the passage of an electrical current. Points are seen to correspond to areas of low electrical resistance, and therefore may be more sensitive to stimulation with a needle (Saita 1973). In addition studies have also demonstrated that traditional acupuncture points have a 71% correlation with MTrP’s (Melzack, 1977).

There is now compelling evidence to show the MTrP is to be found at a site where a branch of a muscle’s motor nerve enters the muscle and terminates in a number of motor endplates (Mense 2001). At this site there is also a neurovascular bundle, containing large and small sensory nerves, the latter having terminal nociceptors, and blood vessels with closely associated autonomic nerve fibres. This neurovascular point correlates with the motor point of the muscle (Gunn 1976, 1977).

This would be consistent with the findings of increased neural elements and lowered skin resistance at acupuncture points previously described. So it would seem that the MTrP, the motor point and the traditional acupuncture point may in many cases be the same thing (Melzack, 1977, Simons 1999).

This apparent link between the MTrP, the motor point and the traditional acupuncture point has led to a modern, scientific based attempt at a nomenclature that is able to replace the centuries old Chinese system. Gunn (1976, 1977) classifies acupuncture points into four groups as follows:

- **Type I** Motor points.
- **Type II** Focal meetings of superficial nerves in the sagittal plane.
• Type III  Areas lying over superficial nerves or plexuses.
• Type IV  Musculo-tendinous junctions.

Type I points, muscle motor points, are particularly used in the technique of Intramuscular Stimulation (Gunn, 1976). Examples of common motor points of the anterior thigh and leg are detailed in figures 2 and 3.

Fig.2 Motor Points of the Anterior Thigh
The location and radiation of symptoms from MTrP’s has been charted by Travell and Simons (1983) and a clear overlap can be seen between MTrP’s, motor points and acupuncture points. This is demonstrated when comparing Fig 2, Motor Points of the Anterior Thigh and Fig 4 which outlines the site and radiation of symptoms from MTrP’s in Vastus Medialis and Vastus Intermedius.
Laboratory Investigations

Laboratory investigations are only helpful in diagnosing myofascial pain in so much as they exclude other pathologies. Blood tests, including full blood count, erythrocyte sedimentation rate, rheumatoid factor and antinuclear antibody are normal. Radiographic imaging shows no specific abnormality. Although Gunn (1977a, 1980) describes MTrP’s
in muscles as being related to a radiculopathy at the spinal segment which supplies that muscle, the commonest cause of which is spondylosis. As spondylosis represents the structural disintegration and morphologic alteration that occurs in the intervertebral disc, with concurrent patho-anatomical changes in the surrounding structures these changes would be visible on plain radiograph.

In a thermographic study by Fisher (1984) there was found to be an increased temperature change associated with tender MTrP’s and in a further study by Sola (1956) there was a reduced skin conductance found overlying MTrP’s. A lower skin conductance over trigger points fits with Gunn’s Type I acupuncture point, the motor point, the definition of which is ‘the area of skin overlying a muscle that requires the minimal amount of electrical stimulation to induce a contraction’ (Gunn 1976, 1977).

EMG assessments have been used in attempts to diagnose MTrP’s but are generally unrevealing, except in endplate potentials, which are common in midfiber trigger points, less common in taught bands, and not present outside of an endplate zone. It is suggested that endplate potentials may be characteristic of MTrP’s (Simons 2002).

Clinically increased trigger point activity and consequent myofascial pain is often described by patients as being exacerbated by emotional stress. This is supported by McNulty (1994) who demonstrated that emotional stress was seen to trigger increased EMG activity in trigger points compared with adjacent tissue, which remained electrically silent.

**Pathophysiology of Myofasical Pain and associated MTrP’s**

Recent evidence has afforded some insight into myofascial pain and associated MTrP’s providing a more scientific explanation for its pathophysiology and subsequent treatment (Simons 1997, Simons 1999, Hong 1998). It has been suggested that the mechanism of MTrPs formation is a phenomenon of central sensitisation in the spinal cord, but that the central sensitisation is a response to peripheral activation. This peripheral activation may be induced by a variety of causes such as trauma and ischaemia and be more probable in patients whom have an existing degree of neuropathy.

Previous discussion has already highlighted that there is evidence to show that a MTrP is to be found at a site where a branch of a muscle’s motor nerve enters the muscle and terminates in a number of motor endplates (Mense 2001). At this site there is also found the neurovascular bundle which contains both sensory and autonomic components. These sites corresponding to motor points (Type I acupuncture points described by Gunn (1976)), frequently also correspond to traditional acupuncture points (Melzack 1977).

There is also evidence to suggest that the commonest reason for MTrP activity arising is trauma to the muscle. This trauma maybe a direct injury to the muscle or as the result of an indirect injury such as muscle overload during prolonged poor posture (Baldry 2002).
Gunn (1980) advocates a theory to explain myofascial pain based on the peripheral activation of MTrP’s and neuromuscular supersensitivity due to peripheral neuropathy, a condition often associated with spondylosis, although research to support this theory is weak. Gunn’s theory is based on the fact that neuropathy will lead to an increased sensitivity to acetylcholine (ACh) at the neuromuscular junction (Fambrough 1974, Katz 1964, Lomo 1976). Although the term neuropathy is used to describe this condition Gunn claims that in many cases the symptoms of neuropathy are found in the distribution of both the anterior and posterior rami of the affected segmental nerve. This denotes a radiculopathy (neuropathy at the nerve root) and is probably a more accurate description (Thomas, 1984).

Radiculopathy as a cause of myofascial pain is not a condition familiar to most clinicians, whereas in cases of cervical disc prolapse compression of the nerve and the resultant radicular symptoms are well described. Neuropathy may be defined as a disease that causes abnormal function in the peripheral nerve (Gunn 1980). Structural changes are not usually seen on imaging such as MR scan within the nerve itself and the neuropathic nerve still conducts nerve impulses, synthesises and releases transmitted substances and evokes action potentials and muscle contractions.

Of all the possible causes of radiculopathy, such as trauma, metabolic, toxic and degenerative changes, the commonest is the steady and slow attrition due to degenerative change and it is this often mild degenerate change coupled with minor injury that becomes chronic pain (Gunn, 1980). At the spinal level this is correctly termed spondylosis and represents the structural disintegration and morphologic alteration that occurs in the intervertebral disc, with concurrent patho-anatomical changes in the surrounding structures.

Frykholm (1951) showed that the meningeal coverings of the nerve roots are often affected by these spondylitic changes, and that dural sleeve thickening and fibrosis can occur. This produces stenosis, and surgical release of the dural sheath can provide pain relief.

The way in which a radiculopathy affects muscles is attributed to Cannon and Rosenblueth’s Law of Denervation (Cannon and Rosenblueth, 1949). This law states,

“When a unit is destroyed in a series of efferent neurons, an increased irritability to chemical agents develops in the isolated structure or structures, the effect being maximal in the part directly denervated”.

All denervated structures are able to develop super-sensitivity; specifically of interest to myofascial pain is the fact that this includes skeletal muscle. Cannon and Rosenblueth’s original work was based on total denervation for the development of super-sensitivity and not merely a neural irritation. However, Sharpless (1975) demonstrated that any circumstance that impedes the flow of motor impulses for a period of time is able to rob the effector organ of its excitatory input. A term he described as disuse super-sensitivity
It is the supersensitivity in muscle that is perhaps the most important feature of radiculopathy and myofascial pain as it will lead to muscle shortening in the muscles supplied by the affected spinal segment (Gunn 1997). Often the whole muscle becomes sensitised to palpation; a term sometimes referred to as allodynia or myalgic hyperalgesia depending on the severity of the condition.

In addition to this model of neuropathy the chronic nature of many myofascial pain syndromes is also related to the phenomena of neuroplasticity and central sensitisisation. Although neurones in the central nervous system form synapses and pathways that are frequently dedicated to specific functions, pain mechanisms are not hard wired or fixed. Instead they are soft wired or plastic, changing according to previous and current input and other intrinsic factors. Neuroplasticity is the development of these pathways in response to a continued neural input and can produce pathological pain (Coderre 1993).

Central sensitisation is increased sensitivity of the central nervous system nociceptive neurones that receive sensory information from the body and is a result of neuroplastic change. In the myofascial pain syndrome this plasticity may come about due to the sensory afferent bombardment that follows the activation of MTrP’s and sensitisation of Group IV nociceptors.

Noxious afferent input activates N-methyl D-aspartate (NMDA) receptor ion channel mediated reactions in the dorsal horn that sensitize both nociceptive and wide dynamic range (WDR) spinal neurones. Long lasting potentiation of nociceptive processing within the dorsal horn then produces the phenomenon of central sensitisation (Neugebauer 1990). Spontaneous activity in nociceptive afferents, expansion of receptive fields and decreases in firing threshold all play a role in inducing exaggerated responses to normally non-noxious afferent stimuli (Cervero 1996).

Additional evidence supports the existence of WDR neuronal mediated reflex muscle response induced by central sensitisation (Morgan 1996). Subthreshold synaptic potentials from combined mechanoreceptor and nociceptor afferents produce summation on the motorneuron pool, resulting in a reduction in the threshold, and thus an increase in the overall excitability of the alpha-motorneurons (Woolf 1995).

In addition to neuropathic and neuroplastic changes in the peripheral and central nervous system Simons (1999) has proposed an integrated theory to explain a mechanism of MTrP genesis at the motor endplate. Simons considers that a MTrP is essentially a region of multiple dysfunctional motor endplates, and that each dysfunctional endplate is associated with a section of muscle fibres that are contracted forming the tender taught band that can be palpated during clinical examination.

The connection between the dysfunctional motor endplate and the taut contraction knot in the muscle is thought to be related to what has been termed the ‘Energy Crisis Theory’ outlined in Fig.5 (Simons1999).}
The hypothesis is that there is an excessive production of ACh from the dysfunctional motor neurone terminal into the synaptic cleft occurring under normal resting conditions. This excessive release of ACh may be stimulated by either a direct trauma to the muscle or as the result of an indirect injury such as muscle overload during prolonged poor posture (Baldry 2002). The excessive ACh activates ACh receptors in the post-synaptic membrane of the neuromuscular junction and produces an increase in the number of miniature endplate potentials (MEPP’s). This increase may be further heightened if the innervating nerve is neuropathic resulting in a degree of neuromuscular supersensitivity to released ACh. (Gunn 1980).

The MEPP’s are detectable as spontaneous electrical activity and initiate a sustained depolarisation of the post-synaptic membrane of the muscle fibre. Hubbard and Berkoff (1993) reported that spontaneous electrical activity (SEA) could be recorded from a trigger point region. SEA consists of continuous, low amplitude, noise-like action potentials (10-50 microvolts, occasionally up to 80 microvolts). It may be accompanied by intermittent large amplitude spikes (100-600 microvolts) especially from more active trigger points. In previous animal studies electrical activity similar to SEA was recorded in the endplate zone when the endplate was irritated either mechanically or biochemically. This activity was shown to be a consequence of excessive acetylcholine release (Ito, 1974). SEA was not directly related to spinal cord activity, since an animal study demonstrated that transection of peripheral nerves or spinal cord did not induce any obvious change in SEA over a period of an hour (Hong 1998).

![Diagram of the neuromuscular junction and energy crisis theory](image-url)
It remains unclear whether these potentials are indeed spontaneous and some authors refer to them as end plate noise (EPN) rather than SEA (Simons 1996). However, a search of the physiological literature has indicated that these noise-like potentials represent grossly abnormal endplate activity as compared to the normal MEPP discharge pattern (Simons 2002). Simons (2001) reviews in detail the experimental basis for the understanding that EPN and spikes result from an abnormal increase (up to three orders of magnitude) in the rate of spontaneous release of ACh from the motor end plates of nerve terminals.

Simons (2002) compared the prevalence of motor endplate potentials (noise and spikes) in active MTrP’s, endplate zones, and taut bands of 11 muscles in 10 subjects an attempt to assess the specificity of endplate potentials to MTrP’s. The results demonstrated endplate noise occurring in all 11 muscles at MTrP’s, 4 muscles at endplate zones (but outside the MTrP), and in no taut bands outside the endplate zone (Fig. 6). The conclusion was that endplate noise seems to be characteristic of, but not restricted to the region of the MTrP.

Fig.6 Recording of endplate noise and spikes observed in a trigger point. The upper trace shows 20-50μV continuous endplate noise component with two 100μV superimposed high amplitude spikes. The lower trace is recorded at the same amplification but from a site approximately 1cm to the side of the trigger point. The lower trace shows no electrical activity beyond baseline noise (Simons 2002).

The sustained depolarisation caused as a result of the MEPP’s causes a continuous release of calcium from the sarcoplasmic reticulum resulting in a sustained shortening of the muscle sarcomeres. The excessive demand for the production of ACh in the motor neurone terminal, the continuance of the depolarisation, the ongoing release and uptake of calcium, and the sustained shortening of the sarcomeres leads to a local increase in the demand for energy.

In addition to this increased energy demand the sustained muscle contraction may compress local vessels and so compromise the blood supply leading to a subsequent decrease in oxygen and nutrient supply. The increased demand and decreased supply of energy results in a local energy crisis (Simons 1999). It may be that the local hypoxia results in a breakdown of the calcium pump that under normal circumstances returns calcium to the sarcoplasmic reticulum. This pump is driven by adenosine triphosphate which may become depleted due to the local hypoxia. Failure to remove calcium and
return it to the sarcoplasmic reticulum would result in a sustained contraction and muscle shortening.

Local changes resulting from the energy crisis may lead to release of neuroactive substances that sensitise both sensory and autonomic nerves within the neurovascular bundle. This is supported by Shah (2004) who in a study using a microanalytic technique detected the presence of neuropeptides at active trigger points. These neuropeptides were similar to those seen in neural inflammation and suggest that myofascial pain is linked to the presence of localized inflammatory agents.

The sensitisation of both sensory and autonomic nerves within the neurovascular bundle may result in the tenderness elicited when MTrPs are palpated and account for the referred pain from MTrPs. Hong (2000) observed that the referred pain patterns of some MTrPs are similar to the traditional meridian connections of acupuncture points. The consistent pattern of referred pain in a specific MTrP suggests that there are fixed connections between certain sensory neurons in the spinal cord. These are probably the same as the connections between acupuncture points along a meridian (Hong 2000).

In addition peripheral sensitisation of nociceptors leads to an afferent barrage of both nociceptive and WDR spinal neurones activating NMDA receptors and precipitating central neuroplastic changes. Neuroplastic changes in dorsal horn synaptic responses to acute nociceptive pain have been demonstrated to occur within hours of tissue injury (Neugebauer 1990).

**Effect of Dry Needling on Myofascial Trigger Points**

Clinically MTrP injection of local anaesthetic and/or corticosteroid has been used as an effective and valuable procedure to deactivate an active MTrP and subsequently relieve the pain and tightness of the involved muscle. During MTrP injection, as soon as the needle penetrates to a sensitive site, an LTR can usually be elicited (Hong 1997, Gunn 1980). Dry needling, that is needling the MTrP without injection of any drug, has been reported to be as effective as injection of local anaesthetic (Hong 1997).

Chen (2001) demonstrated a significant decrease in SEA in MTrP’s in rabbit skeletal muscle following dry needling that elicited LTR’s (Fig.6). He concluded the elicitation of LTR’s, rather than the trauma effects of the needling were the primary inhibitory factor on SEA during dry needling.

**Fig. 7** Electromyographic recording of spontaneous electrical activity from myofascial trigger points in the biceps femoris muscle of a rabbit before (*left*) and immediately after dry needling (*right*). (Chen 2001).
Gunn (1997) describes disuse sensitivity as leading to the formation of MTrP’s and subsequently to muscle contracture and shortening. Release of these muscles is usually necessary to restore joint range and relieve pain. Typically when several of the most painful MTrP’s are released in a muscle then relaxation of the entire muscle follows.

The needle is said to cause a decrease in SEA and subsequent relaxation via either a direct local electrical stimulus or via a reflex mechanism. As the needle is introduced into muscle tissue it disrupts the cell membrane of individual muscle fibres and causes a brief discharging described as “insertional activity”. This insertional activity may produce amplitudes as high as 2mv. This can be prolonged in states of neuropathy (> 300ms) and is further augmented by manipulation of the needle. The insertional activity can case a shortened muscle to visibly fasciculate and subsequently relax (Gunn 1978). In addition as the muscle is injured by the needle then a “current of injury” follows, this current of injury was first described in 1797 by the Italian scientist Galvani. Injury potentials of several microamperes are generated and can persist and provide stimulation for days until the miniature wounds heal (Gunn 1978, Jaffe 1985).

A study by Lomo (1976) demonstrated that denervation supersensitivity in animal muscle may be reduced or abolished by electrical stimulation. Hypersensitivity as assayed by the sensitivity of muscle extrajunctional membrane to acetycholine diminished at a rate, which depended on the amount and pattern of the stimuli.

In addition rotation of a needle grasped by muscle shortening can produce intense stimulation. Rotational motion is converted to linear motion which shortens the muscle fibres locally. This shortening activates muscle spindles and Golgi Tendo Organs and may cause subsequent muscle relaxation via local spinal reflexes.

**Needling Techniques**

Baldry (2002) described the MTrP as a site of activated and sensitized nociceptors which when palpated gave rise to a flexor withdrawal reflex (jump sign) and, in some cases, the
utterance of an expletive (shout sign). He stated that dry needling needed to be of sufficient strength to abolish both signs and that a superficial technique was adequate in 90% of patients. The remaining 10% of patients, in addition to having nociceptive MTrP pain also suffered form nerve root compression pain and responded to deep dry needling. Baldry’s superficial technique involves insertion of needles (0.3mm x 30mm) into the tissues overlying the MTrP to a depth of no more than 10mm. needles are left in situ long enough for both the ‘jump sign’ and the ‘shout sign’ to be eliminated. An average time of 30 seconds is described.

The use of a superficial technique is supported by Macdonald (1983) who examined superficial needling (to a depth of 4mm) on MTrP’s in patients complaining of chronic low back pain, demonstrating significant benefit to patients using this technique. However, numbers were small, 8 in the superficial group and 9 in a placebo group (transcutaneous electrical nerve stimulation). The study also used electroacupuncture if no improvement was noted from superficial needling, making assessment of the effects of needling alone difficult.

Edwards (2003) using a randomized controlled, trial examined superficial needling in conjunction with active muscle stretching comparing it to active stretching in isolation or no treatment. The trial concluded that superficial needling followed by active stretching was more effective than stretching alone in deactivating MTrP’s and also more effective than no treatment (measured by Short Form McGill Pain Questionnaire and Pressure Pain Threshold). Stretching without prior MTrP deactivation was also stated as increasing MTrP sensitivity.

In a study by Lewit (1979) needle effectiveness on the relief of myofascial pain was examined, the study supported the use of deep dry needling for the deactivation of MTrP’s. This was supported by Ceccheerelli (2001) who used a randomized controlled study to demonstrate deep needling to be superior when compared to superficial needling in the treatment of myofascial shoulder pain. This study did however include needling of both MTrP’s and more traditional acupuncture points and therefore makes it difficult to assess the significance of either one.

Gunn (1997) uses a technique he terms intramuscular stimulation (IMS) to relieve myofascial pain by dry needling muscles, with acupuncture needles, at tender motor points. Points he has termed Type I acupuncture points (Gunn 1976). During the technique of IMS a 40mm needle will routinely be used, the absolute size being dependent on the muscle being needled and the depth of the motor point being treated. This departure from classical acupuncture allows stimulation of deeper motor points by using a manual plunger for insertion, oscillation, and twirling of the needle.

In a single-blinded crossover trial Chu (2004) examined a group of patients complaining of chronic low back pain. The study compared two experimental groups, one receiving electrical twitch-obtaining intramuscular stimulation (ETOIMS) using a monopolar EMG needle electrode inserted into multiple sites in paraspinal muscles from T10 to S1. The other experimental group received intramuscular stimulation (IMS) without any electrical
stimulation. The control group received superficial skin stimulation only. The trial concluded that although ETOIMS provided a greater immediate and sustained pain relief than muscle stimulation or skin stimulation without electrical stimulation the difference was not statistically significant.

In a systematic review by Cummings (2001) 23 trials studying needling for myofascial pain concluded that no specific method demonstrated superiority and therefore patient comfort should guide treatment. Most of the trials studied in this review used a deep needling technique.

**Conclusions**

Many patients suffering from myofascial pain will have associated MTrP’s which are readily detectable in a clinic without the need for sophisticated investigative procedures. These MTrP’s may be due to both peripheral and central sensitization mechanisms. Probably some of the proposed mechanisms are relevant in some conditions, and in some patients, and others are relevant in other conditions and other patients. For example if no tissue injury exists, either direct or indirect the peripheral mechanisms are less likely to have a significant role. In accurately diagnosing a patient’s pain as being related to MTrP’s the clinician therefore needs to be aware of all the possible causative factors that have allowed the development of the MTrP’s and be attentive to the fact that these will no doubt be different in successive patients.

**References.**


