

New Frontiers in the Matrix of *Neuro*-musculoskeletal Pain: Integrating Pain Mechanisms with Objective Physical Findings and Needling Strategies

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Goals and Objectives:

- To gain deeper understanding of the mechanisms of central and peripheral sensitization, and investigate the critical role of these neuroplastic changes in perpetuating chronic *neuro*-musculoskeletal pain
- Discuss the unique neurobiology of muscle pain
- Demonstrate that an active myofascial trigger point (MTrP) in the upper trapezius has elevated levels of inflammatory mediators, neuropeptides, catecholamines and cytokines – substances known to be associated with pain, sensitization and inflammation
- Discuss the limitations of digital palpation
- Introduce novel applications of ultrasound techniques to visualize MTrPs, measure their stiffness properties and local blood flow
- Demonstrate that MTrPs in the upper trapezius are stiffer than surrounding tissue and that active MTrPs can be distinguished from latent MTrPs by their high-resistance blood flow
- Summarize the reproducible physical manifestations of spinal segmental sensitization (SSS) associated with chronic *neuro*-musculoskeletal pain
- Review how improved quantitative and objective diagnostic techniques are used to determine the spinal segments involved in SSS (including dermatomes, myotomes and sclerotomes), and how such investigations are applicable in the diagnosis and treatment of chronic *neuro*-musculoskeletal pain
- Discuss and demonstrate modalities and needling techniques used to desensitize the involved segments, eliminate chronic myofascial trigger points and alleviate chronic *neuro*-musculoskeletal pain

Abstract

Chronic pain states are characterized by profound changes in neuronal excitability and architecture in the pain matrix. These neuroplastic changes occur in the spinal cord, thalamic nuclei, cortical and limbic areas and may alter the threshold, intensity and affect of one's pain experience. Spinal Segmental Sensitization (SSS) is a hyperactive state of the dorsal horn caused by bombardment of nociceptive impulses from sensitized and/or damaged tissue. Active (i.e., spontaneously painful myofascial trigger points [MTrPs]) are a very common source of persistent nociception and sensitization of dorsal horn neurons that often results in SSS and chronic pain. Furthermore, recent studies of the biochemical milieu (using novel microanalytical techniques) and viscoelastic properties (using office-based diagnostic ultrasound) have revealed fascinating objective abnormalities of MTrPs that help explain their role in myofascial pain syndrome and SSS.

The dynamic changes that occur during the initiation, amplification and perpetuation of SSS may explain the objective and reproducible segmental physical findings (e.g., dermatomal allodynia and hyperalgesia) and the effects observed following dry needling.

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This workshop and handout will integrate emerging knowledge from the pain sciences in a clinically accessible way by discussing how to identify findings suggestive of SSS in patients with chronic pain. In addition, modalities and needling techniques that desensitize the involved spinal segment will be discussed and demonstrated.

Distinct Neurobiology of Muscle Pain

Most current knowledge on pain mechanisms is derived from studies on cutaneous pain. In actuality, muscle pain has a unique neurobiology. Its distinctive characteristics are critical in explaining the clinical presentation of myofascial pain. Muscle pain can often be described as aching, cramping, deep and difficult to localize. It is distinguished from cutaneous pain in that muscle pain involves nociceptive-specific neurons in the brainstem and spinal cord[1, 2] and activates unique cortical areas that are associated with affective or emotional components of pain[3]. Although muscle nociception is inhibited more intensely by descending pain-modulating pathways[4, 5], persistent muscle nociception, compared to cutaneous nociception, is more effective at inducing maladaptive neuroplastic changes within the dorsal horn[6]. Such neuroplastic changes support the clinical observation that muscle pain is often difficult to resolve.

Characteristics, Evaluation, and Diagnostic Criteria of Myofascial Pain

Musculoskeletal pain is the most common manifestation of chronic pain. The term *neuro-musculoskeletal* pain is preferable when describing a chronic musculoskeletal pain state because it accurately implies fundamental alterations in the nervous system – sometimes irreversibly so. Myofascial pain arises from myofascial trigger points (MTrPs) (see Figure 1). An MTrP has been defined as a “hyperirritable spot, usually within a taut band of skeletal muscle or in the muscle’s fascia, that is painful on compression and that can give rise to characteristic referred pain, tenderness, and autonomic phenomena[7].” Accordingly, it has been found that active MTrPs have a significantly lower pain pressure threshold than latent MTrPs and normal, uninvolved muscle tissue[8]. Diagnosis depends exclusively upon history and physical examination. *The Trigger Point Manual* contains detailed instructions for examination which may be performed by a clinician trained in manual palpation techniques.

While MTrPs cause local pain upon palpation, it is also common for them to project pain to distant sites, such that myofascial pain is experienced in seemingly unrelated areas. Continued pressure over an active MTrP should increase local pain and mimic the patient’s reported referral pain patterns. A latent MTrP, though not spontaneously painful, is usually tender and may also be associated with referred pain upon palpation.

Another characteristic physical finding of the MTrP is the presence of a local twitch response (LTR). This involuntary, localized contraction of muscle fibers is both transient and rapid and can be elicited by manual palpation. In fact, the LTR is considered a criterion of an MTrP. While controversy exists over an official list of diagnostic criteria, Gerwin (1997) outlined essential findings of an MTrP: 1.) an exquisitely tender spot found in a taut band of muscle, 2.) an LTR and/or referred pain to distant sites upon manual palpation or needling of the tender spot, 3.) restricted range of motion, 4.) reproduction of the patient’s pain complaint through pressure on the MTrP, 5.) regional muscle weakness and 6.) autonomic symptoms[9]. The fourth criterion is only applicable for active MTrPs since latent MTrPs do not cause spontaneous pain.

Despite the fact that muscle makes up more than half of the human body by weight, there is no organized focus on student training or research in muscle pain. As a result of a lack of understanding, awareness and/or training, muscle pain is often overlooked. MTrPs are the most common, yet misdiagnosed and inadequately treated component of non-articular musculoskeletal pain disorders. Clinicians tend to treat the symptoms of muscle pain (e.g., with medications) rather than the cause, which are usually MTrPs. Muscle pain is often given little consideration because there is neither consensus on the diagnosis nor any standardized objective measures to verify the presence of MTrPs. To date, accurate diagnosis of myofascial pain depends exclusively upon the palpation skills, clinical acumen and experience of the examiner.

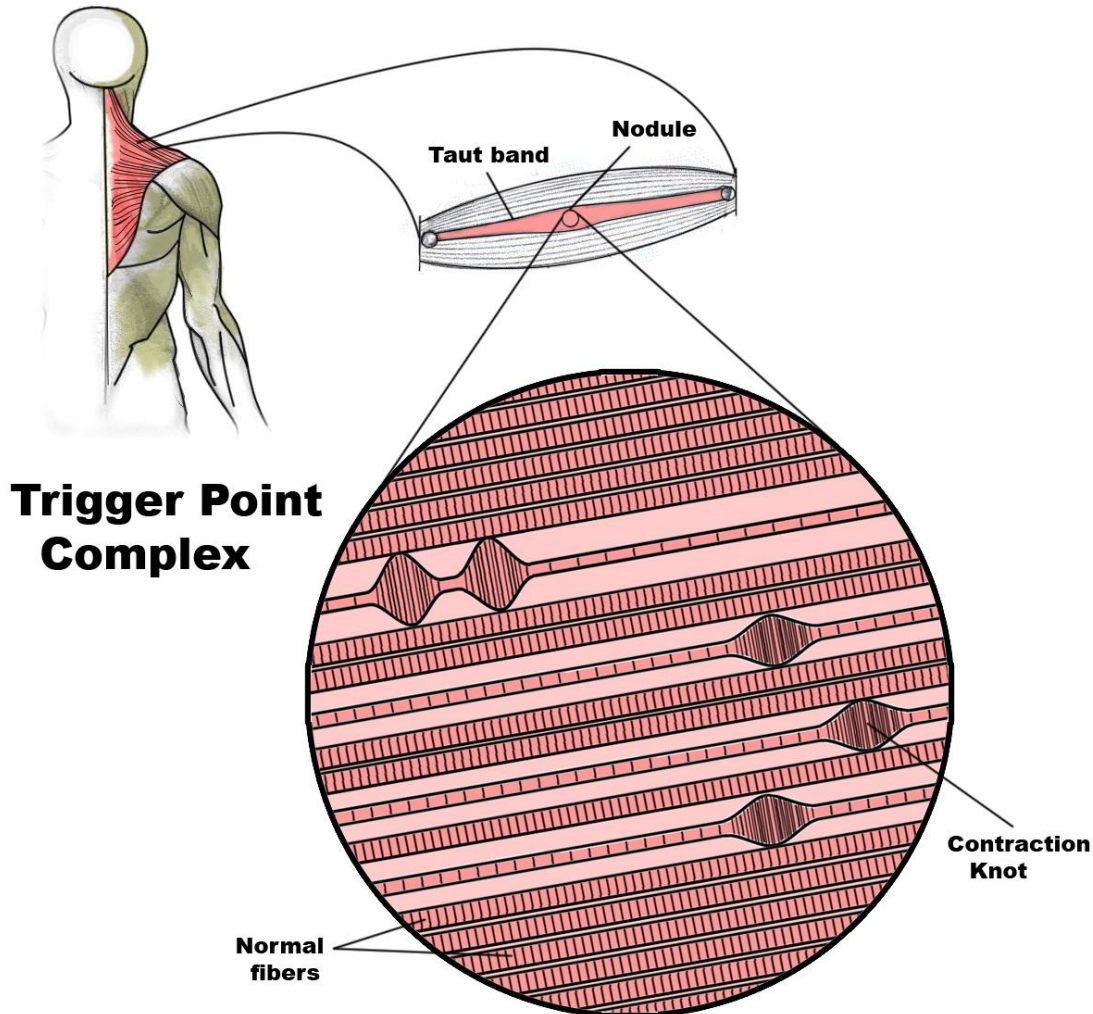


Figure 1. Schematic of a trigger point complex. A trigger point complex in a taut band of muscle is composed of multiple contraction knots (Adapted from Simons, D.G., Travell, J.G. *Myofascial Pain and Dysfunction: The Trigger Point Manual*, vol. 1; second ed., and Användare: Chrizz.)

Background on Sensitization

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Chronic pain syndromes (e.g., myofascial pain syndrome, fibromyalgia, etc.) exhibit profound neuroplastic changes, altering neuronal excitability and architecture in structures of the pain matrix (e.g., the spinal cord, thalamic nuclei, cortical areas, amygdala and periaqueductal gray area). This dynamic process can fundamentally alter pain threshold, pain intensity and emotional affect[10].

Signaling in the pain matrix may begin with activation of polymodal nociceptors, structures which can be sensitized by substances released from damaged tissue and the nociceptor terminals themselves. Prolonged noxious input may lead to long-term changes in gene expression, somatosensory processing and synaptic structure. For example, a continuous barrage of noxious input into the dorsal horn (a process termed “afferent bombardment”) results in the co-release of L-glutamate and substance P (SP). Released together, these two substances can lower thresholds for synaptic activation and open previously ineffective synaptic connections in wide dynamic range (WDR) neurons, thus inducing central sensitization[11, 12].

Sensitization up-regulates ion channel and receptor expression and increases the number of these membrane proteins on nociceptors and dorsal horn neurons. Under normal circumstances, a dynamic balance exists between pain’s role in facilitating and inhibiting function. Neurons conveying nociceptive information are controlled by a variety of inhibitory interneurons, structures critically involved in preventing the transition from acute to chronic pain[10].

An understanding of segmental distribution of sensory nerve fibers is a vital component in proper pain management[13]. Innervation patterns of the skin, muscles and deep structures occur at an early stage of human fetal development and little variability exists among individuals[14]. Accordingly, each spinal cord segment has a consistent segmental relationship to its spinal nerves. This allows clinicians to attribute the pattern of dermatomal, myotomal and sclerotomal hyperalgesia to dysfunction in its corresponding spinal segment[13, 15].

Spinal segmental sensitization (SSS) is a hyperactive state of the dorsal horn caused by bombardment of nociceptive impulses from sensitized and/or damaged tissue (e.g., somatic structures such as active MTrPs or visceral structures such as the gall bladder). Manifestations in the sensitized spinal segment include dermatomal allodynia (i.e., pain to a normally non-painful stimulus) and hyperalgesia (i.e., increased pain to a normally painful stimulus) in addition to sclerotomal tenderness and MTrPs within the involved myotomes [13, 15]. Hyperalgesia of central origin is so prevalent that in one study, it was found to be responsible for 61% of patients suffering from arthrosis. This suggests that both central and peripheral mechanisms are responsible for maintaining a chronic pain state in these individuals. Initially, hypersensitivity occurs at a local, affected site but it is possible for central mechanisms to then begin and persist separately from the peripheral process[16]. Further, segmental sensitization occurs through neuron hypertrophy as well as upregulation of excitatory neurons, prohyperalgesic peptides, and neurotransmitters at the dorsal horn. As a result, pain and inflammation occur as independent events, as one condition is not indicative of the other.

MTrPs are Associated with Peripheral Abnormalities

While the pathophysiology of myofascial pain remains enigmatic, various studies have begun to elucidate its underlying properties. Our research team has sought to determine if there are objective biochemical differences among active MTrPs, latent MTrPs, and normal muscle

tissue. To accomplish this, we developed a novel microdialysis needle (Figures 2A and 2B) with the same size, shape, and characteristics of an acupuncture needle. This microanalytical technique could safely and quantitatively measure the local biochemical environment of muscle *in vivo* using continuous, real-time sampling[12, 17, 18].

We chose to investigate the levels of biochemical substances (e.g., inflammatory mediators, neuropeptides, catecholamines, cytokines, etc.) that are released from and act on muscle, nerve, and connective tissue. These bioactive substances were selected because they are known to be associated with sensitization, pain, inter-cellular signaling and inflammation. Results from the upper trapezius muscle indicate that active MTrPs have a unique biochemical milieu compared to latent MTrPs and muscle without palpable MTrPs. Subjects with neck pain secondary to an MTrP had significantly elevated local levels of various endogenous substances, including substance P (SP), calcitonin gene-related peptide (CGRP), bradykinin (BK), serotonin/5-hydroxytryptamin (5-HT), norepinephrine (NE), tumor necrosis factor-alpha (TNF- α), and interleukin-1 β (IL-1 β) within the active MTrP compared to carefully matched controls[12, 17, 18]. Interestingly, compared to controls, subjects with active MTrPs in the upper trapezius also had elevated levels of these biochemicals in a remote, unaffected muscle (the gastrocnemius)[12, 18].

Together, these studies demonstrated and confirmed that the clinical distinction between active and latent MTrPs is associated with a highly significant objective difference in the local biochemical milieu. High concentrations of the biochemicals found have the ability to cause both peripheral and central sensitization. These findings may help to explain why active MTrPs are acutely painful, tender, and a source of referred pain. Our biochemical studies have helped to establish the clinical importance of palpating and identifying active MTrPs. They also suggest that myofascial pain is an objective entity in the spectrum of clinical pain states and may also explain why specific treatments are effective. For example, studies have found that an injection of the serotonin antagonist tropisetron was found to be more effective than lidocaine in relieving pain from MTrPs[19, 20]. Fittingly, our research indicates elevated local levels of 5-HT within individuals suffering from active MTrPs. CGRP, which was also found to be elevated in these individuals [12, 17, 18], has implications for activity within the neuromuscular junction. CGRP enhances the release of acetylcholine (ACh) from the motor end plate, decreases the effectiveness of acetylcholinesterase[21, 22], and upregulates the ACh-receptors in the muscle. As ACh activity becomes more effective, the frequency of miniature endplate potentials increases as does the development of persistent focal muscle fiber contraction, a defining characteristic of the MTrP[23].

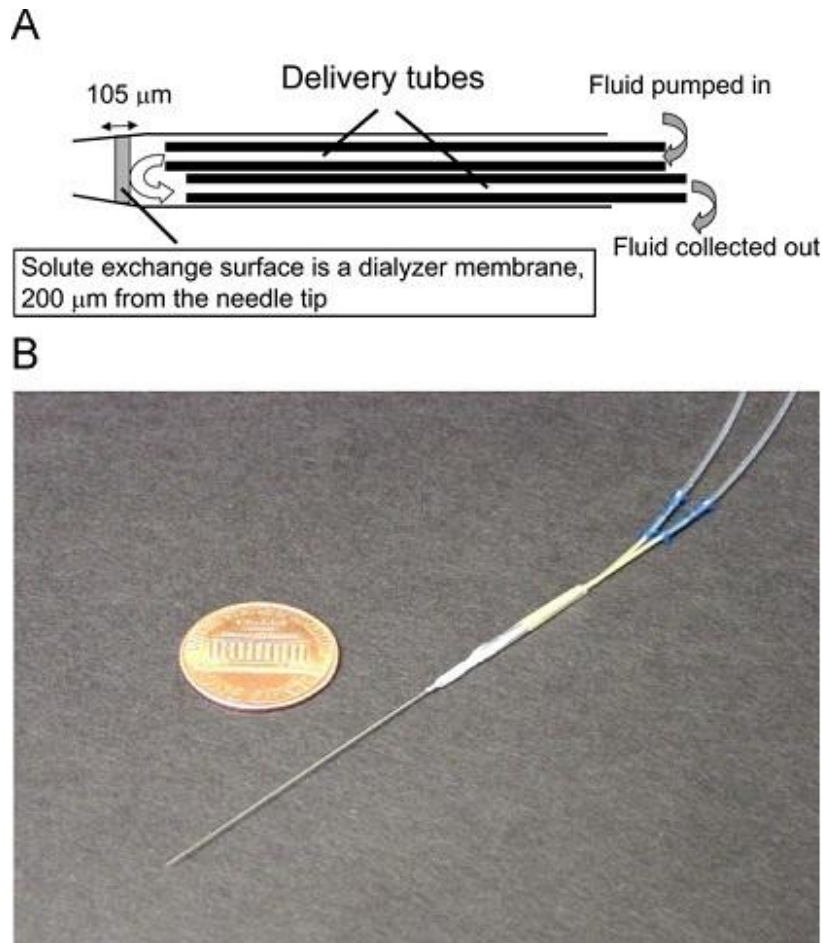


Figure 2. (A) Microdialysis schematic. (B) Photo of needle. (Reproduced with kind permission by the American Physiological Society and Elsevier, Ltd., from Shah *et al.*, 2005, 2008.).

Peripheral to Central Sensitization in Muscle Pain

Sensitization of both peripheral and central afferents is responsible for the transition from normal to aberrant pain perception in the central nervous system that outlasts the noxious peripheral stimulus. In animal models of pain, nociceptive input from skeletal muscle, as compared to cutaneous nociceptor activation, is much more effective at inducing neuroplastic changes in the spinal cord [6]. Continuous input from peripheral muscle nociceptors may lead to changes in function and connectivity of sensory dorsal horn neurons via central sensitization. For example, sustained noxious input from an active MTrP may sensitize dorsal horn neurons leading to allodynia, hyperalgesia, temporal summation of pain[24] and expanded referral pain patterns. A possible explanation for this phenomenon is increased synaptic efficiency through activation of previously silent (ineffective) synapses at the dorsal horn.

This concept of opening previously ineffective connections was demonstrated in a rat myositis model. Experimentally-induced inflammation unmasked receptive fields remote from the original receptive field, indicating that dorsal horn connectivity expanded beyond the original neurons involved in nociceptive transmission[25]. In this study, nociceptive input resulted in central hyperexcitability and this finding helps to explain referred pain patterns common to MPS. Central sensitization may also facilitate additional responses from other receptive fields as a result of convergent somatic and visceral input at the dorsal horn[26] via wide dynamic range

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(WDR) neurons (Figure 3). Furthermore, afferent fibers have the ability to sprout new spinal terminals that broaden synaptic contacts at the dorsal horn and may also contribute to expanded pain receptive fields[27]. This change in functional connectivity may occur within a few hours, even before metabolic and genetic alterations occur in dorsal horn neurons[28].

There is a biochemical basis to explain the development of peripheral and central sensitization in muscle pain. Continuous activation of muscle nociceptors leads to the co-release of L-glutamate and SP at the pre-synaptic terminals of the dorsal horn. In addition to activation of alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors by L-glutamate at the post-synaptic terminal, SP facilitates activation of previously dormant N-methyl-D-aspartate (NMDA) receptors. This leads to maximal opening of calcium-permeable ion channels, which hyperexcites nociceptive neurons and causes apoptosis of inhibitory interneurons[29]. Consequently, a persistent noxious barrage from the periphery can create long-lasting alterations in the central nervous system. Metabolic and gene induction changes, such as cyclo-oxygenase 2 (COX-2) induction in dorsal horn neurons, are maximal at several hours after an initial noxious stimulation and bolster functional changes after peripheral tissue injury[30].

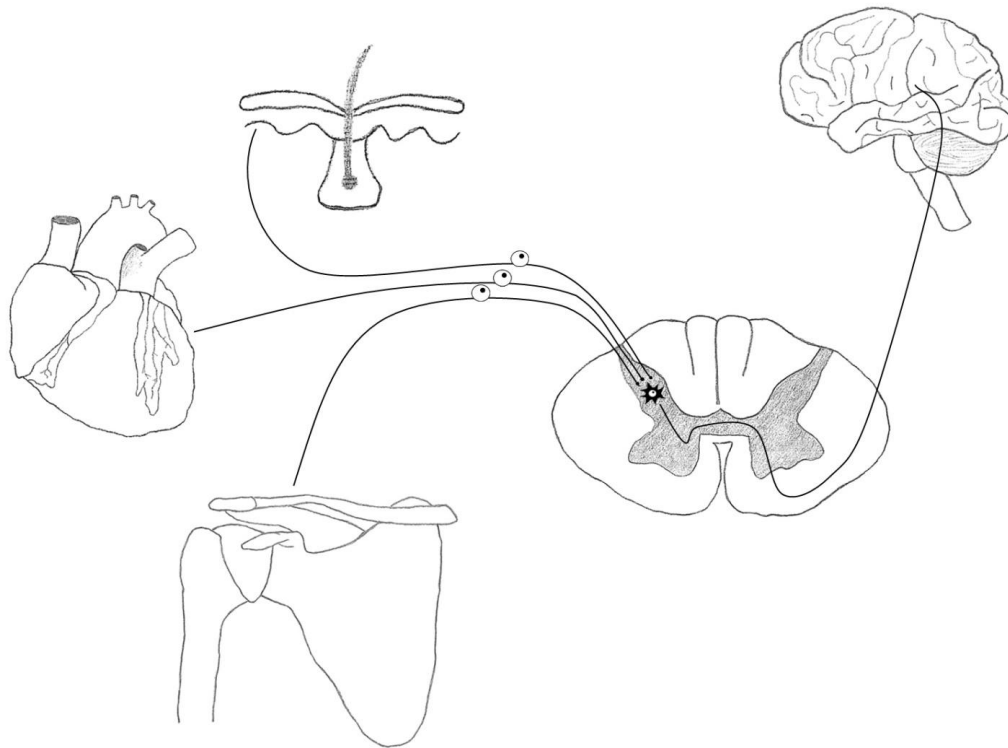


Figure 3. Wide dynamic range neuron. A WDR neuron receives convergent input from cutaneous, visceral, and deep somatic afferents and subsequently sends signals to the thalamus. As such, the WDR neuron and higher-level brain centers can be driven by various inputs. Accordingly, central sensitization may facilitate responses from other structures (e.g., the shoulder, heart and skin) which share convergent input.

(Adapted from: Willard, F., "Basic Mechanisms of Pain." *Future Trends in CAM Research, in Integrative Pain Medicine: The Science and Practice of Complementary and Alternative Medicine in Pain Management*, J.F. Audette, Bailey, A., Editor 2008, Humana Press Inc.: Totowa.)

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Higher Brain Centers Dynamically Modulate Muscle Pain

As aforementioned, persistent afferent input from active MTrPs preferentially activates and sensitizes WDR neurons in the dorsal horn. The stimuli then ascend the spinothalamic tract to reach higher brain centers. In addition to activating the thalamus, muscle afferent input preferentially activates the limbic system (i.e., the anterior cingulate gyrus, insula and amygdala), which plays a critical role in modulating muscle pain and the emotional or affective component to persistent pain[3]. Increased activity in the limbic system leads to greater fear, anxiety, and stress. Furthermore, Niddam et al. (2007) demonstrated increased limbic system (i.e., anterior insula) activity in patients with upper trapezius myofascial pain syndrome[31].

There is a dynamic balance between supraspinal descending facilitation and inhibition. For example, the rostral ventral medulla (RVM) is a relay area between the periaqueductal gray (a structure located in the midbrain) and the spinal cord. The RVM contains a population of “on” cells and “off” cells which can either increase or decrease the level of pain, respectively. It does so through projections that modulate activity in the dorsal horn. Following initial tissue injury, the “on” cells serve a useful and protective purpose designed to prevent further damage. Under ordinary circumstances, tissue healing would lead to a decrease in “on” cell activity and an increase in “off” cell activity. However, in chronic musculoskeletal pain conditions, there appears to be an overall shift to a decrease in inhibition, presumably due to an imbalance of “on” cell and “off” cell activity[11].

Muscle pain also impairs diffuse noxious inhibitory control (DNIC)[32]. Disrupted descending inhibition in chronic musculoskeletal pain may lead to an increased pain sensitivity of muscle tissue[33]. Current data suggest that MTrPs are not merely a peripheral phenomenon but rather, they activate and sensitize WDR neurons in the dorsal horn and higher brain centers and may, in turn, be dynamically modulated by these structures[31, 34].

Dry Needling and the Local Twitch Response

Dry needling is an effective, non-pharmacological treatment of MTrPs which has approached acceptance as the “standard of practice” for deactivating active MTrPs. It may be performed using either a superficial or deep dry needling technique. Elicitation of one or more local twitch responses (LTRs) is a goal of dry needling and often benefits those with pain secondary to MTrPs. Though the mechanism of an LTR is unknown, studies suggest a biochemical component. Five minutes after the induction of a single LTR, our group found a dramatic change in the biochemical milieu of the upper trapezius muscle. Within minutes of the LTR, the initially elevated levels of SP and CGRP within the active MTrP drastically decreased to levels approaching that of normal uninvolved muscle tissue. The reduction of these biochemicals in the local muscle area may be due to a small, localized increase in blood flow and/or nociceptor and mechanistic changes associated with an augmented inflammatory response[17, 18]. Though not designed as a treatment intervention, the results of these studies are provocative in that the substances analyzed are known to be associated with sensitization, persistent pain, and spinal facilitation. In an animal model, it appears that dry needling may, in fact, activate the descending inhibitory pain system and cause local deactivation of the MTrP[35].

Limitations of Digital Palpation

Current diagnostic standards for myofascial pain rely on palpation for the presence of MTrPs in a taut band of skeletal muscle[7]. However, proper diagnosis requires a highly skilled clinician and some studies have found low inter-rater reliability among examiners in their attempts to identify MTrPs[36, 37]. Although digital palpation is considered the gold standard for diagnosis, it does have several limitations. Specifically, digital palpation does not 1) provide an objective, reliable and sensitive method of diagnosis and measurement of treatment efficacy; 2) provide quantitative comparisons of the tissue properties before and after treatment; 3) objectively differentiate among active MTrPs, latent MTrPs, and palpably normal tissue; 4) objectively discriminate between superficial and deep MTrPs; and 5) permit objective study of the natural history of MTrPs.

Visualization and Characterization of MTrPs

Accordingly, there is a need to develop objective, repeatable, and reliable diagnostic tests for evaluating MTrPs and determining treatment outcome measures. Such measures can be used to properly diagnose MTrPs, understand their natural progression, and overcome the subjectivity and limitations of digital palpation. Accordingly, our group has applied three types of ultrasound diagnostic imaging techniques—grayscale (2D ultrasound), vibration sonoelastography (Figure 4), and Doppler—to differentiate tissue characteristics of MTrPs in the upper trapezius muscle compared to surrounding soft tissue.

These office-based measures are readily available, portable, and inexpensive imaging modalities, suitable for use in a clinician’s office. We have demonstrated that ultrasound elastography can serve as an objective image-based measure of MTrPs. Using ultrasound, MTrPs can be imaged and appear as focal hypoechoic (darker) areas with a heterogeneous echotexture. MTrPs also have reduced vibration amplitude on elastography, indicating a localized area of stiffer tissue compared to surrounding soft tissue (Figure 4)[8, 38].

Our studies have also revealed that MTrPs have a unique vascular environment. Doppler ultrasound was able to show differences in the microcirculation in and around active MTrPs compared to latent MTrPs and normal tissue. For example, blood flow waveform characteristics can be used to differentiate active and latent MTrPs. Blood flow reversal in diastole was associated with active MTrPs, indicating a very high resistance vascular bed. This may be due to a blood vessel compression by a local muscle contracture (e.g., an MTrP) and/or biochemically-mediated vasoconstriction of the local blood vessels [38, 39]. Further analysis has also demonstrated that active MTrPs have a significantly larger surface area than latent MTrPs and normal sites[8].

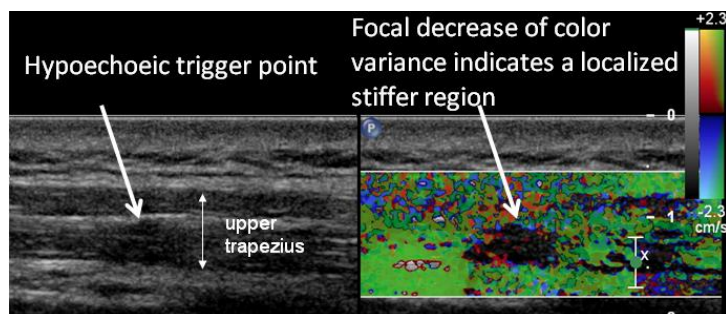


Figure 4. (A) Upper trapezius muscle with a palpable MTrP. A hypoechoic region and a well-defined focal decrease of color variance indicating a localized stiffer region are visible.

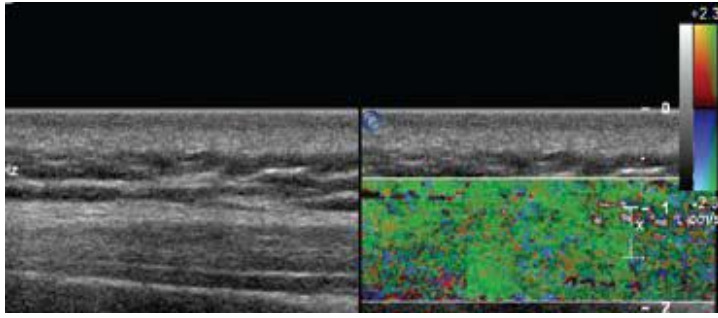


Figure 4. (B) Normal upper trapezius muscle. A myofascial trigger point is not palpable and the normal muscle appears isoechoic and has uniform color variance.

Spinal Facilitation

Spinal facilitation is an increase in activity of spinal cord neurons due to the bombardment of nociceptive stimuli into the dorsal horn (Figure 5)[40]. Under normal circumstances, activation of primary afferent nociceptors in the dorsal horn is modulated by inhibitory mechanisms either locally or via descending pathways from the cerebral cortex or brainstem. However, persistent nociceptive afferent input may result in inhibitory neuronal cell death, wind-up and sensitization of secondary order neurons in the dorsal horn. Circuits in the spinal cord (i.e., dorsal horn, ventral horn and lateral horn) may develop lowered thresholds of activation, causing them to be more easily activated by minimal or no input at all. The ensuing spinal facilitation is characterized by:

- 1) Increased ventral horn outflow that stimulates anterior motor horn cells, resulting in increased muscle tone in the myotome corresponding to its segmental level of afferent barrage;
- 2) Increased lateral horn outflow which results in autonomic reflexes that enhance nociceptive activity; and
- 3) Increased dorsal horn outflow that causes anti-dromic electrical activity along a sensory nerve (also known as “dorsal root reflexes”).

Dorsal root reflexes activate dorsal root ganglion cell bodies to increase production and release vasoactive neuropeptides (e.g., SP, CGRP and somatostatin) both centrally and peripherally. Upon release into the peripheral tissue, they may exacerbate a local inflammatory process by stimulating vasodilation and plasma extravasation. This results in local tissue tenderness and mechanical hyperalgesia. Furthermore, adjacent spinal segments may become progressively sensitized upon bombardment of the central nervous system[24].

An underappreciated anatomical fact is that primary afferent nociceptive fibers actually trifurcate upon entering the dorsal horn (Figure 6). That is, one branch enters the dorsal horn at that segmental level, one branch ascends, and one branch descends along the dorsal margin of the dorsal horn. Furthermore, some visceral afferents have been found to span the entire length of the spinal cord[41, 42]. These often overlooked anatomical considerations have enormous implications in the initiation, perpetuation and amplification of spinal facilitation and persistent pain states.

Consider the following clinical scenario: An individual develops severe acute painful cholecystitis. The sustained noxious input is of sufficient intensity and duration to destroy inhibitory neurons at the segmental level of entry into the dorsal horn (T6). Fortunately, removal

of the gall bladder completely alleviates the pain. Years later, the individual develops a minor but acutely painful back injury while lifting heavy boxes. The resultant afferent nociceptive input immediately enters the lower thoracic/upper lumbar segments and ascends/descends the spinal cord. While ascending the cord, but prior to reaching T6, presumably intact and functional inhibitory neurons suppress the nociceptive input and prevent the sensation of pain at those segmental levels. However, upon reaching the T6 segment, the nociceptive signal is able to activate dorsal horn neurons at this level since the local inhibitory neurons are dysfunctional and/or dead. These result from prior cell death following the original intense gall bladder stimuli associated with the acute cholecystitis. In fact, a common clinical manifestation is reproduction of the identical pain pattern caused years before, in this case by the acute gall bladder disease. Under these circumstances, the patient may become distressed and even complain of acute gall bladder pain even while acknowledging that the organ had been removed, thus resulting in a type of “phantom” pain. Accordingly, practitioners of osteopathy often interpret the re-emergence of an old pain pattern as the possible harbinger of new disease. The cause could be musculoskeletal in origin (as in this example) or due to an underlying visceral problem or disease (e.g., peptic ulcer disease, pre-clinical cardiac ischemia, etc.)

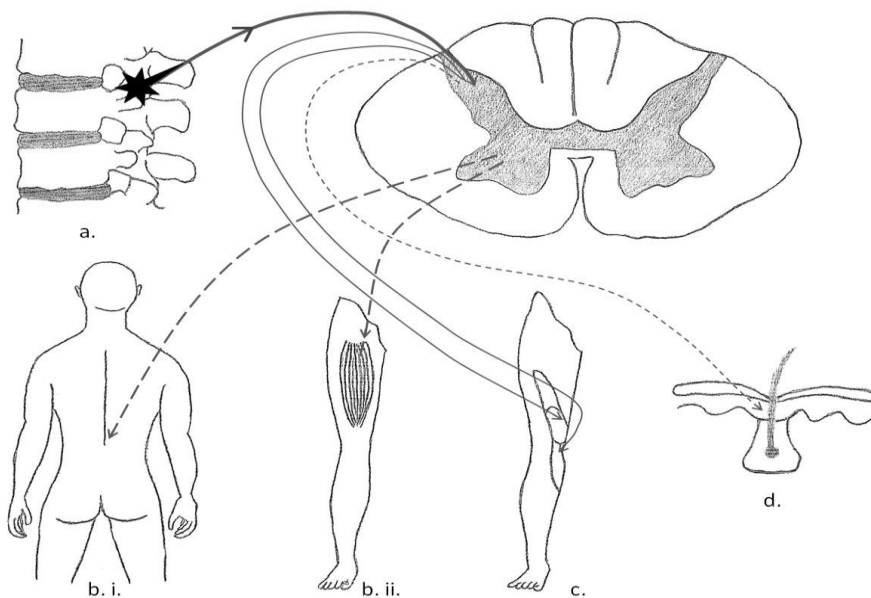


Figure 5. Facilitated spinal segment.

a. Nociception originating in the L4 facet joint synapses on the dorsal horn.

b. At the L4 segmental level, a motor neuron within the ventral horn becomes activated, causing a reflex spasm of muscles innervated by the same segment such as (i.) the paraspinal muscles and (ii.) the rectus femoris muscle.

c. Dermatomal and sclerotomal structures sharing the L4 segmental level may become sensitized and painful as a result of dorsal root reflexes.

d. Dorsal root reflexes in the L4 segment may also sensitize cutaneous structures, rendering them more painful.

(Adapted from Romero Ventosilla, P., *Consecuencias clínicas de la Estimulación Sensorial persistente: Sensibilización Espinal Segmentaria*, (Personal Communication), 2010.)

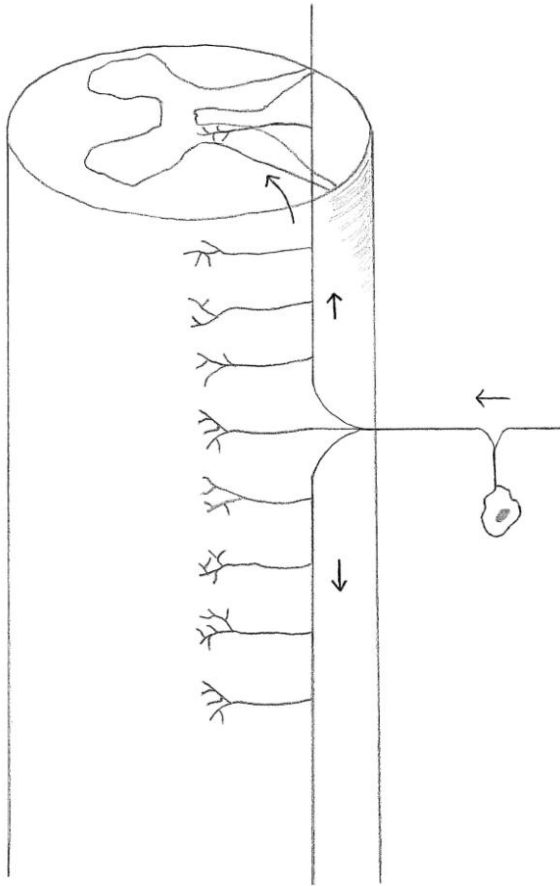


Figure 6. Trifurcation of a neuron. Upon entry into the dorsal root of the spinal cord, primary afferent neurons have the ability to trifurcate; one branch enters at that segmental level while other branches may ascend and descend along the dorsal margin of the dorsal horn in Lissauer's tract.

Diagnosis and Implications of Spinal Segmental Sensitization in the Clinic

Spinal segmental sensitization is consistently associated with musculoskeletal pain states, underscoring its significance. For example, involvement of thoracic spinal levels (e.g., T1-T12) in SSS facilitates and perpetuates abdominal pain and somatovisceral symptoms commonly mimicking gastrointestinal conditions, such as peptic ulcer disease. The development or activation of MTrPs is one of the clinical manifestations of SSS. In other words, a latent MTrP that is located along a sensitized segment (i.e., myotome) may become an active MTrP (i.e., associated with a spontaneous pain complaint).

Many treatments for myofascial pain such as physical therapy and trigger point injection procedures are directed at the peripheral pain generators, e.g., active MTrPs. Oftentimes, the segmental dysfunction is overlooked and practitioners fail to recognize the presence of SSS. As a result, many individuals may only experience temporary deactivation of MTrPs and pain frequently recurs.

An accurate diagnosis of pain distribution requires identification of the sensitized spinal segment. SSS is determined by findings of allodynia, hyperalgesia and measurable pressure pain sensitivity over the sensory, motor and skeletal areas along with viscera supplied by a particular spinal segment (i.e., the dermatome, myotome, sclerotome and viscerotome, respectively).

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Furthermore, these objective and quantitative findings help the clinician to identify the tissues and likely pain mechanisms involved in their patients' chronic pain. These segmental findings are not only reproducible, but they are often indicative of the severity of the sensitized state and provide important clues about the underlying pathogenesis of the pain syndrome.

The requisite examination skills are easy to learn and of fundamental importance to the evaluation and management of a chronic pain complaint. Furthermore, their application before and after treatment, aimed at desensitizing the involved spinal segment, provides the clinician and patient meaningful, objective and reproducible physical findings to guide treatment outcomes.

Imamura *et al.* (2008) systematically evaluated individuals with refractory, disabling pain associated with knee osteoarthritis (OA), who were scheduled to undergo total knee replacement. The authors speculated that the pain experienced by these patients may be associated with the presence of central nervous sensitization rather than peripheral inflammation and injury. The presence of hyperalgesia was evaluated, and the impact of pressure pain threshold (PPT) measurements on pain, disability and quality of life was assessed in these patients and compared to age-matched healthy controls. PPT measurements were obtained for the subcutaneous dermatomes of the lower extremities and over the vastus medialis, adductor longus, rectus femoris, vastus lateralis, tibialis anterior, peroneus longus, iliacus, quadratus lumborum and popliteus muscles and the supraspinous ligament. They found that the group with knee OA had significantly lower PPT over all evaluated structures versus healthy control subjects. Lower PPT values were correlated with higher pain intensity, higher disability scores and poorer quality of life, except for the role-emotional and general health status. Combined PPT values over the patellar tendon, at the S2 subcutaneous dermatome and at the adductor longus muscle were the best predictors for the visual analog scale and Western Ontario and McMaster Universities Osteoarthritis Index pain scores. They concluded that patients with knee pain due to OA who were scheduled for total knee replacement showed hyperalgesia of nervous system origin that negatively impacted pain, knee functional capacity and most aspects of quality of life[16].

In order to determine the presence of SSS, the patient is asked to identify with one finger the location of his/her principal pain complaint and indicate the intensity of pain from 1-10. Adjacent dermatomal levels are examined paraspinally by:

- Scratching the skin with the sharp edge of a paper clip or Wartenberg pinwheel – this noxious stimulus is applied across dermatomal borders and the patient is instructed to simultaneously report any sharpening or dulling in the sensation of pain during the procedure. An increased painful response is indicative of hyperalgesia.
- Picking up the skin between the thumb and forefinger and rolling the tissue underneath, also known as a “pinch and roll” test (Figure 7) – this non-noxious stimulus is applied across dermatomal borders and the patient is instructed to simultaneously report any sensation of pain. The sensation of pain is indicative of allodynia, a finding that is the most sensitive indicator for the diagnosis of sensitization.

Adjacent myotomal levels are examined by:

- Palpating segmentally related musculature for tender spots, taut bands and MTrPs.
- Applying a pressure algometer to measure the local tenderness (i.e., PPT) along the myotome.

Adjacent sclerotomal levels are examined by:

- Palpating segmentally related tendons (e.g., tendonitis), entheses (e.g., enthesitis), bursae (e.g., bursitis) and ligaments (e.g., supraspinous ligament sprain).

- Applying a pressure algometer to measure the local tenderness (i.e., PPT) along these sclerotomal structures.

(Note: PPT is the minimum pressure that elicits pain and is considered abnormal if it is at least 2kg/cm² lower than a normosensitive control point.)

These examination techniques are used to determine the depth and breadth of the segmental manifestations of an individual's pain syndrome. Dermatomal, myotomal and sclerotomal segmental findings often overlap, making diagnosis and treatment of the sensitized segmental level relatively straightforward. However, in some cases they do not. For example, the affected dermatomal levels may be different from the affected myotomal and/or sclerotomal levels. When such differences arise, the affected segmental levels (whether dermatomal, myotomal or sclerotomal) most closely corresponding to the principal pain complaint should be treated first with paraspinal needling, a technique which addresses the centrally sensitized component of pain.

If the patient experiences little or no pain relief, then the additional segmental levels may be needled paraspinally until the patient reports a decrease in pain. This subjective decrease in pain is typically accompanied by an objective improvement in the segmental findings. However, effective management involves identification and treatment of both the peripheral and central components of sensitization. Accordingly, the clinician should identify and eradicate all foci of nociceptive bombardment (i.e., peripheral sensitization) responsible for initiating and/or perpetuating the centrally sensitized segmental findings. Active MTrPs are a very common source of peripheral nociceptive bombardment which may lead to central sensitization and perpetuation of the pain complaint. If left unresolved, active MTrPs (or other peripheral pain generators) will re-sensitize the dorsal horn, resulting in the re-emergence of segmental findings (i.e. allodynia and hyperalgesia) and the reproduction of the same pain complaint even after paraspinal needling treatment.



Figure 7. “Pinch and roll” test. The skin and subcutaneous tissue is gently pinched between the thumb and forefinger and rolled vertically across dermatomal borders. Elicitation of a painful response is indicative of allodynia.

Modalities and Manual Therapies

Modalities and manual therapies are often clinically effective at deactivating active MTrPs and desensitizing sensitized spinal segments and are commonly employed as a first line of treatment before attempting more invasive therapies. For example, various forms of electrical stimulation including microcurrent, transcutaneous electrical nerve stimulation (TENS), percutaneous electrical nerve stimulation (PENS), manual therapies and spray and stretch are commonly used to treat myofascial pain and SSS. However, if pain relief is only partial or pain persists despite several treatments using such various modalities, then needling and injection techniques should be considered, particularly in chronic cases in which the physical examination

reveals severe and persistent allodynia and hyperalgesia, suggesting dense dermatomal, myotomal and sclerotomal manifestations of SSS.

Paraspinal Dry Needling Techniques

Fischer *et al.* (2002) developed a technique utilizing injection of 1% lidocaine into the paraspinal muscles adjacent to the spinous processes. A 25-gauge needle, of sufficient length to reach the deep layers up to the vertebral lamina, is inserted in the sagittal plane. Injection is performed between the levels of the spinous processes corresponding to the affected segmental levels of sensitization as identified on physical examination. The needle is inserted through the paraspinal muscle to a maximal depth but before contacting the vertebral lamina. The needle is aspirated (in order to avoid blood vessels) and then approximately 0.1mL of anesthetic is injected; the needle is then withdrawn to a subcutaneous level and redirected in the caudal direction, ending about 5mm from the previous deposit of anesthetic solution. One continues this procedure, going as far as the needle reaches. The same procedures are then repeated going in the cephalad direction. The result of this technique (which Fischer calls a “paraspinous block”) is to effectively block the medial branch of the posterior primary rami at affected segmental levels[43].

Coincidentally, acupuncture practitioners utilize similar anatomical locations (e.g., traditional Chinese medicine “Hua Tuo Jia Ji” points). According to the *Acupuncture Energetics* textbook definition, these are “a collection of points on either side of the ligament attaching the transverse processes of the vertebral column, from T1 to L5, described as one-half Cun lateral to the spinous process; to be needled as deeply as possible into the ligaments just as they are accessible lateral to the midline; very useful as local points for axial or peripheral pain problems, and as points to reinforce the qualities of the back Shu points at the same vertebral levels” for needle insertion in order to achieve pain relief[44]. Many clinicians, including myself (as I have received training in both medical acupuncture and Fischer’s injection techniques), have adapted a dry needling technique to Fischer’s model of paraspinous blocks. Instead of a hypodermic syringe, my preference is to insert acupuncture needles sagittally into the paraspinal muscles as aforementioned (Figure 8). The needle is manipulated by using an up and down (i.e., “pistoning” action) accompanied by clock-wise and counter-clock wise rotations. Multiple acupuncture needles may be similarly inserted and manipulated creating a “paraspinous block” at each of the affected segmental levels, corresponding to the principal pain complaint. Furthermore, the segmentally corresponding supraspinous ligaments may be needled using a superficial needle insertion technique accompanied by just a clockwise and counter-clockwise rotation of the needle (i.e., “pistoning” of the needle should be avoided). The needles may be left in place for 15-20 minutes (similar to standard practice in acupuncture) while the relevant dermatomes, myotomes and sclerotomes are re-examined to determine whether the selected treated segments eliminate the objective signs of SSS (i.e. allodynia, hyperalgesia, etc.). This finding is often accompanied by a significant reduction in pain.

There are several advantages associated with this dry needling technique when compared with Fischer’s injection technique. First, affected segments may be treated without concern for the possible side effects associated with injection of local anaesthetics. In addition, more segments may be treated in one visit with dry needling because the number of injections is limited by the total dosage of anaesthetic that may be administered at one time. Furthermore, acupuncture needles are minimally invasive and better tolerated by patients because they have rounded-tips, designed to painlessly pass around cells, blood vessels and other tissues. On the

other hand, hypodermic needles are bevel-edged and sharp, designed to cut through blood vessels (thereby damaging to cells and tissues); this attribute results in more inflammation, bleeding and pain. Due to its fine construction and smaller diameter, an acupuncture needle provides the practitioner superior kinesthetic feedback when compared to a hypodermic needle. This permits more accurate manipulation and easier placement of the needle at desired tissue depths.

Though many practitioners can attest to improvement in pain levels as a result of dry needling in the paraspinal muscles, these merely arise from clinical observation. To date, there have been no randomized, double-blinded, placebo-controlled clinical trials examining the effects of paraspinal dry needling. A recent study offers a method that may be used to determine the effectiveness of this treatment. Mayoral del Moral *et al.* designed a study in which subjects scheduled for total knee replacement surgery were examined several hours before surgery. They were then randomly assigned to one of two groups: true dry needling or sham dry needling. Upon the induction of general anesthesia but before surgery began, those assigned to true dry needling of MTrPs were treated by a physiotherapist. Since subjects were unconscious at the time of true or sham treatment, they were unaware of their group assignment. Post-surgery, the true dry needling group reported less pain, demanded significantly less analgesics and rated their visual analog scale significantly better than the sham needling group[45]. A similar protocol could be used to systematically assess the outcome of paraspinal dry needling and lend support to its use. Knee joint and related muscles are innervated by the L3-L4 segmental levels. Accordingly, it can be speculated that paraspinal needling within these segments prior to total knee replacement for osteoarthritis may provide better pain relief than surgery alone and could also be used as an effective and conservative first line of treatment.

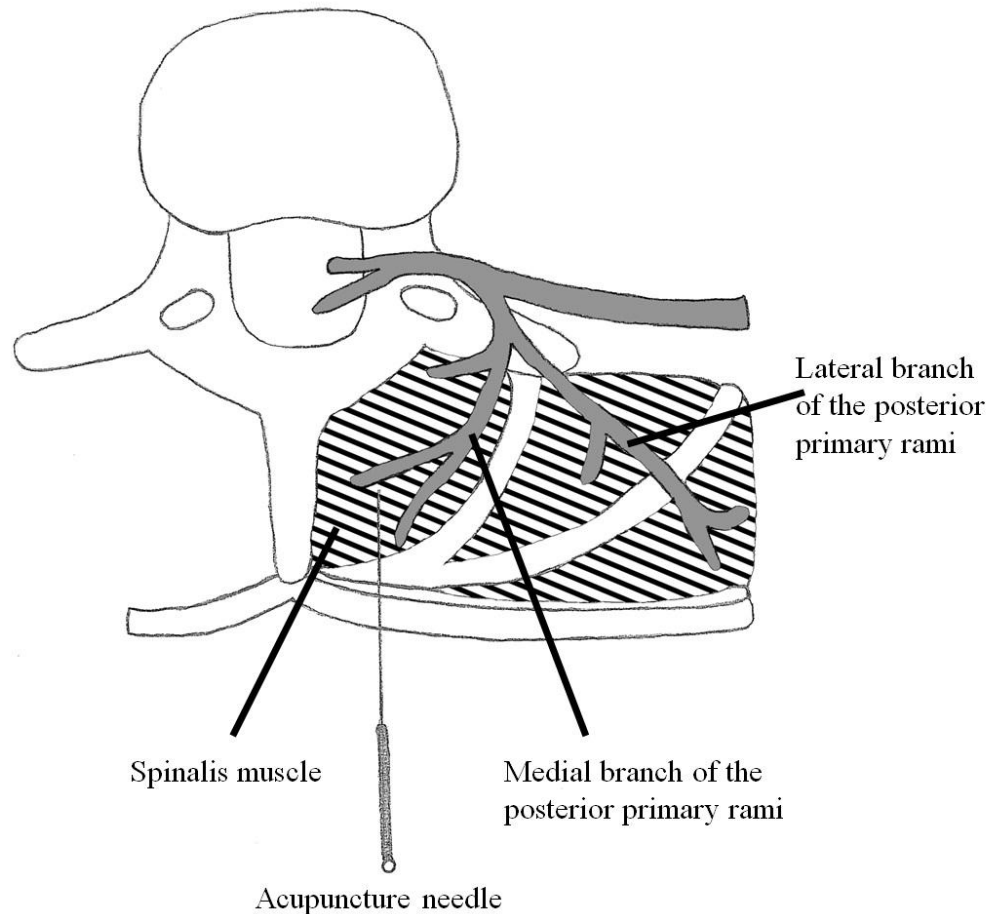


Figure 8. Paraspinal dry needling. An acupuncture needle is inserted sagittally into the spinalis muscle and then manipulated as described. Multiple acupuncture needles may be inserted to create a “paraspinous block” at each affected segmental level.

Conclusion

Current understanding of chronic pain mechanisms, particularly in the unique properties of the neuraxis, is changing rapidly as knowledge emerges in molecular and cellular biology. For example, active MTrPs function as dynamic foci of peripheral nociception that can initiate, accentuate and maintain central sensitization and chronic pain states. Continuous nociceptive input from MTrPs can increase excitability of dorsal horn neurons, leading to hyperalgesia and allodynia, and open previously ineffective synaptic connections, resulting in new receptive fields and pain referral[12].

Although MTrPs are a ubiquitous and under-diagnosed component of many acute and chronic pain complaints, they are also a common physical finding in asymptomatic individuals. This dichotomy challenges pain management practitioners to learn how to carefully palpate the soft tissue in order to distinguish active from latent MTrPs. Making this distinction is critical in order to adequately identify and treat a myofascial component of pain.

Histological, neurophysiological, ultrasound imaging, and somatosensory studies of MTrPs have found objective abnormalities. Novel biochemical sampling techniques demonstrate

that *active* MTrPs have elevated levels of bradykinin, serotonin, substance P, CGRP, norepinephrine, IL-6, IL-8, TNF- α , and IL-1 β . Active sites also have a more acidic pH. Together with observed motor and sensory abnormalities, these studies implicate peripheral and central mechanisms in the development of myofascial pain and associated MTrPs. These biochemical findings validate the clinical observation that active MTrPs are a source of nociceptive foci that continuously bombard dorsal horn neurons, leading to central sensitization and SSS.

Dorsal horn neurons may undergo neuroplastic changes as a result of chronic nociception. However, these changes may also occur in higher centers of the brain if pain is left unresolved for an extended period of time. Alterations in cortical areas may help to maintain and amplify the pain state and thereby create a vicious cycle that is increasingly difficult to resolve. At this point, removal of the etiological factors may be insufficient to relieve pain[24].

Once the presence of central sensitization has been established in a patient with chronic pain, the central nervous system should be targeted in addition to the musculoskeletal component, which is primarily treated with anti-inflammatory agents. Thorough understanding and identification of central nervous system sensitization has the ability to provide innovative and cost-effective therapeutic tools to control pain, reduce disability and improve quality of life. Comprehensive management should focus on the removal of perpetuating factors (e.g., active MTrPs) and by addressing SSS early in its development through methods such as electrical modalities, manual therapies, paraspinal dry needling, paraspinal blocks, centrally-acting pharmacologic agents, biofeedback, behavioral therapy, etc.

Spinal segmental sensitization offers an important paradigm to explain the nature of *neuro*-musculoskeletal pain. Though it essentially has the same origin as peripheral sensitization, the central phenomenon is distinguished by its clinical characteristics. The presence of specific dermatomal, myotomal and sclerotomal distribution patterns are objective, reproducible and reliable hallmarks of SSS. Paraspinal dry needling provides physiotherapists and other clinicians a clinically effective and minimally invasive treatment for *neuro*-musculoskeletal pain.

There is a need to develop objective, repeatable, and reliable diagnostic tests for evaluation and treatment outcome measures for MTrPs. Such measures can be used to properly diagnose and understand the natural history of MTrPs and to determine the underlying mechanisms and relevance to the development and resolution of myofascial pain. They may also be used as outcome measures in treatment trials of various interventions including manual therapies, electrical modalities, etc. Do manual techniques result in “softening” and eventually elimination of the MTrP? Do they improve blood flow in the vicinity of MTrPs, presumably washing out noxious, sensitizing and painful biochemicals? These office-based ultrasound techniques will help answer these questions.

Future Directions

Our group is currently developing a model for the peripheral and central mechanisms involved in myofascial pain. Now that we have identified objective differences which distinguish active MTrPs from latent MTrPs and normal tissue, we plan to further study the nature of MTrPs and surrounding soft tissue over time. Although painful MTrPs activate muscle nociceptors that, upon sustained noxious stimulation, initiate peripheral and central sensitization, what is their etiology and pathophysiology? What is the mechanism by which the pain state begins, evolves,

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and persists? What are the levels of anti-inflammatory substances, analgesic substances and muscle metabolites in the local biochemical milieu of muscle with and without MTrPs? How does a tender nodule progress to a myofascial pain syndrome? Which soft tissues are involved? Are there objective measures for assessing therapeutic outcomes? What is the mechanism by which active MTrPs contribute to SSS? What effects do local and central treatments of MTrPs have on SSS?

Future clinical research studies should focus on identifying the mechanisms responsible for the pathogenesis and pathophysiology of both myofascial pain, SSS and *neuro-*musculoskeletal pain by linking the symptoms and objective physical findings to the physical properties and biochemical changes in the muscle tissue.

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