5 Integrating Dry Needling with New Concepts of Myofascial Pain, Muscle Physiology, and Sensitization

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Summary

Myofascial trigger points are a commonly overlooked cause of chronic neuromusculoskeletal pain and dysfunction. Examination for trigger points requires good palpation skills and understanding of the common referral patterns of myofascial pain. The unique neurobiology of muscle pain and the concepts of peripheral and central sensitization provide new insights into the pathophysiology of myofascial pain. Acupuncture dry needling is an effective technique for treating myofascial pain particularly when local twitch responses are elicited. Uncovering the biochemical profile of active myofascial trigger points and determining the local biochemical effects of needle insertion may help elucidate mechanisms behind the initiation and amplification of myofascial pain and how dry needling works.

Key Words: acupuncture, myofascial pain, dry needling, sensitization, myofascial trigger points, nociceptor, dorsal horn

1. INTRODUCTION

Musculoskeletal (MSK) pain is the most common manifestation of chronic pain. Use of the term *neuro*musculoskeletal pain is preferable for this form of chronic pain to convey accurately the notion that the perpetuation of pain depends upon fundamental and in some cases irreversible changes in the nervous system. Neuroplasticity is a normal adaptive change in the function and/or structure of the nervous system in response to a nociceptive signal. However, in chronic pain, these neuroplastic changes

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become maladaptive and can fundamentally alter one's pain threshold, pain intensity, and affective interpretation of pain.

The most common type of MSK pain is believed to be myofascial pain, or pain that arises from discrete hyperirritable palpable nodules in taut bands of muscle called myofascial trigger points (MTrP) (see Figure 1) *(1)*. The presence of this type of muscle tissue pain generator is still somewhat controversial given the lack of an objective test to verify its presence and, at this point, diagnosis depends upon the systematic palpation of the soft tissue by an experienced examiner following a thorough medical history. An active MTrP causes pain at rest and may often cause general motor dysfunction (stiffness and restricted range of motion). Gerwin et al. concisely summarized the diagnostic criteria for myofascial pain *(2)*:

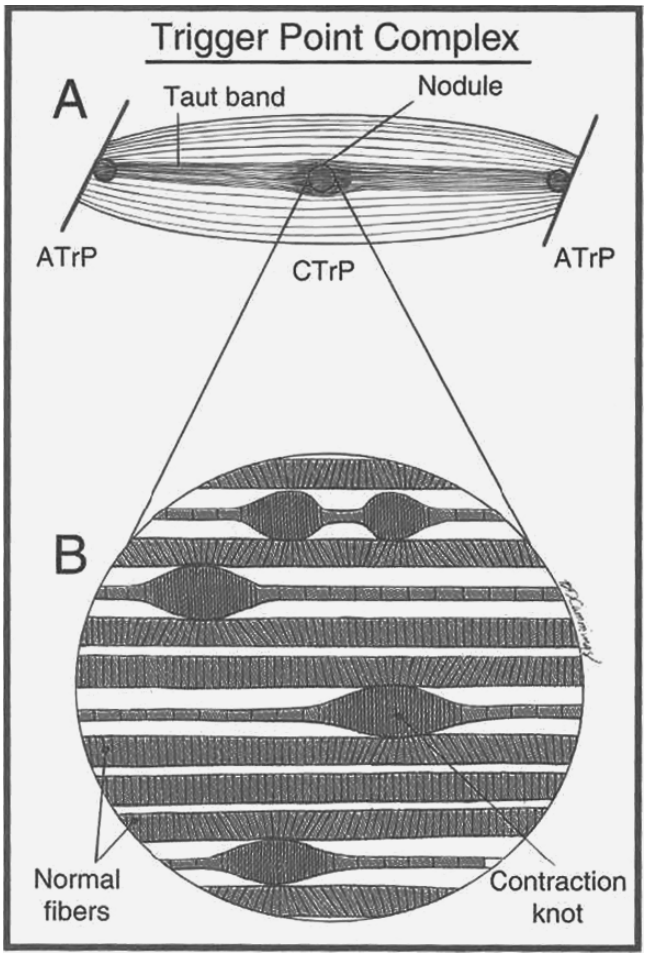

Fig. 1. Schematic of a trigger point complex in longitudinal section. The central trigger point (CTrP) and attachment trigger point (ATrP) regions can exhibit abnormal tenderness. The contraction knots are believed to cause the taut band and make a trigger point feel nodular. (Simons DG, Travell JG, Simons LS. 1999. Travell and Simons' Myofascial Pain And Dysfunction: The Trigger Point Manual. Vol 1. Upper Half of Body. Baltimore: Williams and Wilkins. Original is Figure 2.25 on page 70 of text.)

1. Localized pain in a taut band of muscle
2. Local twitch response to cross fiber stimulation of the taut band
3. Pain to deep palpation that is recognized pain
4. Referred pain to a characteristic distant region based on myofascial referral maps
5. Restricted movement in joints related to muscle
6. Weakness that is not caused by neurological compromise
7. Autonomic dysfunction

A latent MTrP often causes motor dysfunction without pain. Otherwise, latent MTrPs have all the characteristics of active MTrPs, although usually to a lesser degree. Normal muscle does not contain taut bands or MTrPs *(3)*.

There is no standard of care for the treatment of soft tissue pain, in part, because both clinical and basic science research in this area still lags behind research on other painful conditions such as arthritis and neuropathic pain. Dry needling is a treatment technique that has its roots in acupuncture and has been found in clinical studies to be as effective as lidocaine injection in inactivating a myofascial trigger point (MTrP) and providing symptomatic relief *(4)*. Recent experimental data demonstrates that active MTrPs have a unique biochemical milieu that changes with acupuncture dry needling and this data provides important clues about the unique physiology of acupuncture points and the therapeutic effects of acupuncture stimulation *(5)*.

2. MYOFASCIAL TRIGGER POINTS VERSUS ACUPUNCTURE POINTS

In 1977, Melzack theorized that classic acupuncture and trigger point stimulation techniques are generally painful and produce analgesia based on hyper-stimulation of the peripheral nociceptive system, inducing a self-regulating pain modulating effect. He went on to speculate:

> *…the close correlations between trigger points and acupuncture points for pain is remarkable since the distribution of both types of points are historically derived from such different concepts of medicine. Trigger points are firmly anchored in the anatomy of the neural and muscular systems, while acupuncture points are associated with an ancient conceptual but anatomically non-existent system of meridians, which carry Yin (spirits) and Yang (blood). Despite the different origins, however, it is reasonable to assume that acupuncture points for pain relief, like trigger points are derived from the same kind of empirical observation: that pressure at certain points is associated with particular pain patterns, and brief, intense stimulation of the points by needling sometimes produces prolonged relief of pain. These considerations suggest a hypothesis; that trigger points and acupuncture points for pain, though discovered independently and labeled differently, represent the same phenomenon (6).*

Melzack noted a 71% correspondence between acupuncture points and Travell and Simons' MTrPs in terms of spatial location and referral patterns *(6)*. For example, the referral pattern of a common MTrP in the latissimus dorsi muscle tracks very closely together with the paired heart-small intestine meridian described in traditional Chinese medicine. A recent study by Birch has challenged the degree of spatial correspondence between acupuncture points and MTrPs and found a probable correspondence between MTrPs and *a shi* points, which are a different class of acupuncture points. However, his argument is based on an assumption that MTrPs should only be compared to acupuncture points that have a pain indication, which is a misunderstanding of the basic insight of Melzack's, which is based on spatial location alone *(7)*. The actual degree of

correspondence notwithstanding, acupuncture meridians, *a shi* points and Travell and Simons' grid of MTrPs may represent a guide or map of where to examine for common "active" acupuncture points, or points that when palpated or needled can influence the health and pain levels of an organism. An overview of the unique neurobiology of muscle pain and what makes a point "active," whether an acupuncture point or MTrP is needed to better understand this relationship.

3. THE UNIQUE NEUROBIOLOGY OF MUSCLE

Muscle pain has several unique characteristics when compared to cutaneous pain with important physiological consequences:

—Activation of muscle nociceptors causes an aching, cramping pain that is difficult to localize and refers to deep somatic tissues.
—Muscle pain activates unique cortical structures *(8)*.
—Muscle pain is inhibited more strongly by descending pain-modulating pathways.
—Activation of muscle nociceptors is much more effective at inducing neuroplastic changes in dorsal horn neurons *(9)*.

In what follows, we will elucidate what is known about the unique neurobiology of muscle nociception to help understand the biological basis of the development of "active" MTrPs and by extension acupuncture points.

3.1. Sensitization/Activation of Muscle Nociceptors

Muscle nociceptors can be activated mechanically, by deforming the axonal membrane of the nerve ending, or by chemical activation, from the release of sensitizing or pain producing substances from the surrounding tissues and immune cells (see Table 1) *(10)*. It is the latter mechanism of chemical activation that is of clinically greater interest, especially in chronic pain where often there is little gross swelling evident. Endogenous substances such as bradykinin (BK), prostaglandins (PG), and serotonin (5-HT) are not only very effective at sensitizing and/or activating muscle nociceptors but also cause vasodilation. Therefore, it is likely that the release of these substances will also lead to mechanoreceptor activation by distorting the normal tissue relationships. A sensitized muscle nociceptor lowers its normally high stimulation threshold into the innocuous range such that it will respond to everyday stimuli like light pressure and muscle movement *(11)*. Furthermore, at sufficient concentrations, BK and 5-HT can directly activate muscle nociceptors.

The nociceptor endings contain neuropeptides such as substance P (SP) and calcitonin gene-related peptide (CGRP) that, when released, produce vasodilation and plasma extravasation around the nociceptor and the release of sensitizing substances from the surrounding tissue. Whenever the nociceptor is activated by a noxious stimulus, the stored neuropeptides are released, which directly influence the local microcirculation by stimulating vasodilation and increasing the permeability of the microvasculature. More importantly, the secretion of the neuropeptides in sufficient quantity leads to a cascade of events, including the release of histamine from mast cells, BK from kallidin, 5-HT from platelets, and PGs from endothelial cells. The cumulative effect is the increased production and release of sensitized substances in a localized region of edema in the muscle tissue. Therefore, the muscle nociceptor is not merely a passive structure designed to record potentially noxious stimuli. Rather, muscle nociceptors play an active role in the

Table 1
Chemical Sensitivities of Muscle Nociceptors*

Biochemical	*Source*	*Effect on muscle nociceptor*
Acid and potassium	Damaged cells, ischemia	Sensitization/activation
Serotonin	Platelets, mast cells	Sensitization/activation
Bradykinin	Kallidin	Sensitization/activation
Histamine	Mast cells	Sensitization/activation
Prostaglandins	Arachidonic acid, endothelial cells	Sensitization
Leukotriene D4	Arachidonic acid, tissue cells	Desensitization
Substance P	Primary afferent fiber	Sensitization
Cytokines	Damaged cells, myocytes, immune cells, mast cells	Desensitization sensitization/activation

*Adapted from references 10 and 11.

maintenance of normal tissue homeostasis by balancing the vasoconstrictive activity of the sympathetic nervous system and sensing the peripheral biochemical milieu. With tissue injury the secretion of SP and CGRP increases, leading to vasodilation, plasma extravasation, localized swelling and the release of sensitizing substances that can alter the responsiveness of the nociceptor. The sensitization of muscle nociceptors is clearly involved in animal models of muscle pain and may play a key role in the exquisite tenderness found when firm pressure is applied over an active MTrP *(11)*.

It is now known that the activation of a nociceptive ending is not primarily due to a nonspecific damage of the nerve ending by a strong stimulus. Rather, it is due to the binding of specific substances (e.g., BK, PG, 5-HT, etc.) to their paired receptors on muscle nociceptors (see Figure 2) *(11)*. Receptor responsiveness is dynamic. For example, the BK receptor changes when the tissue is pathologically altered. Ordinarily, BK binds to the B2 receptor, but with tissue inflammation a different BK receptor (B1) is synthesized in the cell body of the ending (in the dorsal root ganglion) and inserted into the nociceptor terminal membrane. Unlike the B2 receptor, which is constitutively expressed, the B1 receptor is inducible and is involved in sensitization of the peripheral nociceptor. Induction and binding of the B1 receptor can also lead to the production of pro-inflammatory mediators, including tumor necrosis factor-alpha (TNF-α) and Interleukin-1beta (IL-1β). Stimulation of B2 receptors leads to only transient increases in calcium concentration and as a result, the nociceptor is less prone to sensitization. However, the B1 receptor is considered less susceptible to desensitization mechanisms and its stimulation results in sustained elevations of calcium concentration, which can lead to sustained peripheral sensitization *(12,13)*.

This is an example of an adaptive peripheral neuroplastic change. However, if the change persists after the inflammation has subsided, it becomes maladaptive and may herald the transition from acute to chronic pain. One can imagine, then, that the degree to which muscle nociceptors in a MTrP become sensitized and/or activated will vary according to the balance of sensitizing substances in the muscle tissue and the threshold of their respective receptors. There may be a spectrum of nociceptor irritability based on this balance that distinguishes a normal muscle from a muscle with a latent MTrP from one with an active MTrP.

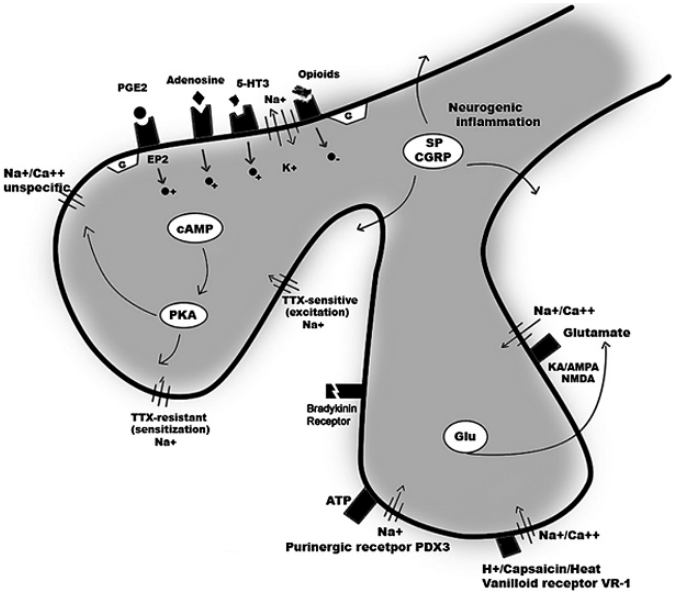

Fig. 2. Schematic drawing of a muscle nociceptor. Membrane receptor molecules for serotonin, bradykinin, prostaglandins, etc. and the intracellular events that increase the sensitivity of the nerve ending are illustrated. (Mense S. The Pathogensis of Muscle Pain. Current Pain and Headache Reports 2003; 7:419–425. Copyright by Current Science, Inc. Original is Figure 1 on page 420 of article.)

3.2. Central Sensitization

The pain and dysfunction from MTrPs may not be caused only by changes in the sensitization of the peripheral nociceptors but also by alteration in the responsiveness of the dorsal horn. A chronic active MTrP may be the nidus of on-going noxious input that sensitizes dorsal horn neurons potentially leading to increased pain or the spread of pain to other segments via central sensitization. Conversely, a sensitized central nervous system may lead to a lowering of the activation threshold of the peripheral nociceptors in a MTrP, which in turn can lead to the transition from a latent to an active MTrP (see Chapter 2). A review of basic muscle pain neurophysiology is helpful in understanding how central sensitization develops.

The primary peripheral sensing apparatus in muscle involves the group III (thinly myelinated, low-threshold fibers, morphologically identical to Aδ fibers in the skin) and group IV (unmyelinated, high-threshold fibers, identical to C fibers in the skin) afferent nerve fibers. These fibers cause aching, cramping pain when stimulated with micro-neural techniques.

The central projections of these fibers share several important characteristics:

a) *reduced spatial resolution* due to a lower innervation density of muscle tissue compared to the skin, thus making it more difficult to localize muscle pain;
b) *convergence of sensory input* from skin, muscle, periosteum, bone and viscera into lamina IV and V of the dorsal horn onto the wide dynamic range neuron, making it difficult to distinguish the origin of the pain compared to cutaneous nociception;
c) *divergence of sensory input* into the dorsal horn with sustained noxious input leads to the opening of previously ineffective connections (this is especially true of group IV

fibers in animal models, which then begin to respond to lower levels of stimulation, i.e., mechanical allodynia) *(11)*.

Compared to normal muscle and muscle with latent MTrPs, a muscle with active MTrPs is more tender and mechanically sensitive, suggesting that peripheral nociceptors are already sensitized. Once sensitized, the group IV afferents will fire at lower thresholds, even though they are normally high-threshold nociceptors. For example, in animal models, injection of bradykinin into muscle will cause the group IV afferents to respond to much lower levels of stimulation (i.e., they become sensitized) *(14)*. Muscle tenderness is mainly due to the sensitization of muscle nociceptors by protons, BK, prostaglandins, and serotonin. Presumably, then, peripheral sensitization by these substances contributes to the tenderness seen in active MTrPs and may contribute to the pain that individuals with active MTrPs describe. For example, in an active MTrP, the stretch of muscles with normal movement may now be sufficient to activate nociceptors that normally are high threshold and would not respond to this type of mechanical activation, thus causing pain.

With the lowering of the activation threshold, the peripheral nociceptors in muscle will fire more readily and can induce central sensitization. In animal models of pain, a nociceptive input from skeletal muscle is much more effective at inducing neuroplastic changes in the spinal cord than is input from the skin *(9)*. Experimentally induced myositis in animal models causes a marked expansion of the response of second-order neurons in the muscle's target area of the dorsal horn. Hoheisel et al. found that noxious input from the gastrocsoleus (L5) muscle after a localized inflammatory reaction was created, also activated second-order neurons in the L3 segment. This segment would ordinarily not be activated by noxious stimulation of the gastrocsoleus in non-inflamed muscle *(15)*. The expansion of the receptive field in the dorsal horn is a result of a central sensitization—i.e., the L3 dorsal horn neurons have become hyperexcitable via previously ineffective afferent inputs due to the continuous nociceptive drive from this L5 muscle and now the L3 segment responds to an input from muscle that it previously did not *(11)*.

The expansion of the receptive field in the spinal cord with myositis-induced excitation is clinically relevant because it can help explain the unusual referral patterns seen in MPS. For example, MTrPs in the suboccipital muscles may refer to the frontal region of the head and MTrPs in the piriformis may cause sciatica (see Chapter 1). In addition, this observed phenomenon in animal models of muscle pain could begin to explain the spread of muscle pain to other segments that many patients experience over time with chronic myofascial pain syndrome (MPS). It also can explain the hyperalgesia many patients report because many of these neurons are hyperexcitable. How do these myositis-induced changes in the spinal cord occur? In what follows, we will show that the changes represent a type of rewiring of the nervous system in response to sustained peripheral drive of an irritable muscle nociceptor (e.g., a MTrP).

3.3. Synaptic Connections in the Dorsal Horn

There are at least two functional types of synaptic connections in the dorsal horn. One is an *effective* synapse—where action potentials arriving at the pre-synaptic portion of the synapse exert a strong influence on the post-synaptic or 2nd order neuron. There are a much larger number of *ineffective* synapses between primary afferents and second-order neurons—ineffective because they don't influence the post-synaptic neuron in a way that will cause it to fire under normal circumstances. These ineffective synapses

are multi-segmental, and there is anatomical evidence that deep somatic afferents can ramify and enter the dorsal horn at up to six to seven segments (see Chapter 2).

The excitatory amino acid glutamate is the pre-synaptic transmitter for nociceptive information in dorsal horn neurons and can act on N-methyl-D-aspartate (NMDA) and the alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors at the post-synaptic site. Under normal conditions, only the AMPA channel is active—so, if one sustains a blow to a muscle, then a short train of nociceptive impulses from the injured muscle will cause the pre-synaptic site in the dorsal horn to release glutamate. This causes a brief activation of the AMPA receptor and post-synaptic neuron. An ineffective synapse doesn't have AMPA channels and so even though the train of impulses may reach segments of the dorsal horn that are normally thought to be outside the myotome of the injured muscle, the second-order neurons will not fire at these levels. However, with an intense or prolonged noxious input, SP will start to co-release with glutamate. If this noxious barrage continues and sufficient quantities of SP are released, the NMDA channel will become responsive to glutamate and open up, allowing the entry of calcium into the cell. The rush of calcium ions into the second-order neuron activates enzymes, leading to a cascade of events that eventually causes the *de novo synthesis* of AMPA receptors in what were previously ineffective synapses. In this way, the release of SP in the dorsal horn in sufficient quantities will increase the efficacy of synaptic connections in the spinal cord, allowing the multi-segmental spread of noxious input. This explains how action potentials emanating from nociceptors in an L5 muscle can then excite neurons in the L3 segment *(11)*.

4. MYOFASCIAL TRIGGER POINTS AND DRY NEEDLING

Myofascial pain associated with MTrPs is a common cause of non-articular musculoskeletal pain. In a community-based chronic pain clinic, Gerwin et al. found that MTrPs were the primary source of pain in 74% of 96 patients with musculoskeletal pain *(16)*. Similarly, Fishbain et al. found that MTrPs were the primary source of pain in 85% of 283 patients consecutively admitted to a comprehensive pain center *(17)*. Myofascial trigger points should be considered in the differential diagnosis of any musculoskeletal condition and are often an overlooked cause of pain in individuals with co-existing joint or visceral disease, including cervical disc lesions, hip osteoarthritis, temporomandibular joint disorders, pelvic pain, headaches and epicondylitis *(18–22)*.

The author's preference for treatment of MTrPs is to use a 32-gauge acupuncture needle for dry needling. An acupuncture needle has a rounded tip compared to the beveled edge of a hypodermic needle and is therefore less painful and less traumatic to tissue. Furthermore, it affords the clinician superior proprioceptive feedback that is very helpful in guiding the needle toward the active MTrPs, which are often firm and initially resistant to needle passage.

In clinical practice, identifying the precise location of the MTrP during injection or dry needling is more important than the type of anesthetic solution injected. The local twitch response (LTR) is a valuable clinical indicator that confirms the accurate location of the MTrP. Furthermore, it is essential to elicit local twitch responses while needling to obtain the desired clinical effect *(4)*. Lewit determined that the effects of dry needling are primarily due to the mechanical stimulation of a MTrP. In his study, treating MTrPs with an acupuncture dry needling technique resulted in immediate pain relief in nearly 87% of the treated sites. Lewit also found that pain relief was permanent

in over 31% of the subjects (20% experienced several months, 22% several weeks and 11% several days of pain relief). Only 14% of subjects had no pain relief *(23)*. There is evidence that needling MTrPs in one muscle group may eliminate MTrPs in muscles that belong to the referred pain area of the treated MTrPs *(24)*. How does dry needling work? In order to answer that question, a better understanding of the pathophysiology of MTrPs is needed.

5. UNCOVERING THE BIOCHEMICAL MILIEU OF MYOFASCIAL TRIGGER POINTS

A team at the National Institutes of Health designed a clinical protocol to assess the local biochemical milieu of MTrPs. A novel 32-gauge microdialysis needle was fabricated that is capable of collecting small volumes (~0.5 μl), at sub-nanogram levels of solutes <75 kDa, from muscle tissue. With such a device it was possible to study the local muscle biochemical milieu in subjects with and without pain and with and without MTrPs at GB-21 in the upper trapezius muscle. This is the most common MTrP in the body and has a unique referral pattern of pain (Figure 3). Furthermore, this needle has the same size, shape and handling characteristics of an acupuncture needle and allows simultaneous sampling of the local biochemical milieu of muscle before, during and after a local twitch response is elicited with the same needle *(5)* (Figure 4).

Three subjects were selected based on history and physical examination to be in each of 3 groups (total 9 subjects): Group 1—*Normal* (no neck pain, no MTrP); Group 2—*Latent* (no neck pain, MTrP present); Group 3—*Active* (neck pain, MTrP present). Samples were obtained continuously with the microdialysis needle at regular intervals, including at the time of needle insertion, elicitation of a local twitch response, and post twitch.

The main outcome measures were concentration levels of protons (pH), substance P (SP), calcitonin gene-related peptide (CGRP), bradykinin, serotonin, norepinephrine, tumor necrosis factor-alpha (TNF-α), and Interleukin -1beta (IL-1β) determined by analysis of samples.

Overall the amounts of SP, CGRP, bradykinin, serotonin, norepinephrine, TNF-α, and IL-1β were significantly higher in the *Active* group than either of the other two groups ($p < 0.01$). The pH was also significantly lower in the *Active* group than the other two groups ($p < 0.03$). In the *Active* group, the amounts of SP and CGRP were significantly lower at the end of sampling (post twitch) than at baseline ($p < 0.02$) (Figures 5, 6).

Subjects with active MTrPs and greater pain levels (i.e., lower pressure pain sensitivities) had lower pH levels in the vicinity of their MTrPs. A positive correlation has previously been shown between pain and local acidity *(25)*. In a rat model, Sluka et al. found that repeated injections of acidic saline into one gastrocnemius muscle produced *bilateral*, long-lasting mechanical hypersensitivity (i.e., hyperalgesia) of the paw *(26)*. Sluka found that the degree of nociceptor activation correlated with increasing acidity of the saline injected. The hyperalgesia was reversed by spinally administered μ- or ∂-opioid receptor agonists *(27)* or N-methyl-D-aspartate (NMDA) or non-NMDA ionotropic glutamate receptor antagonists *(28)*. This model demonstrates secondary mechanical hyperalgesia is maintained by neuroplastic changes in the CNS. Furthermore, the persistent mechanical hyperalgesia was not caused by muscle tissue

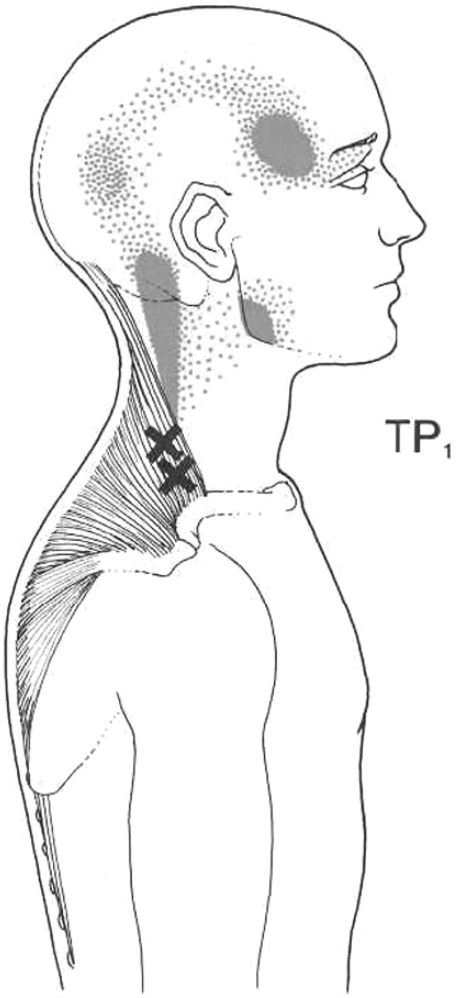

Fig. 3. Referred pain pattern and location of (X) of central trigger point 1, identical to GB-21, in the upper trapezius muscle. (Simons DG, Travell JG, Simons LS. 1999. Travell And Simons' Myofascial Pain and Dysfunction: The Trigger Point Manual. Vol 1. Upper Half of Body. Baltimore: Williams and Wilkins. Original is Figure 6.1 on page 279 of text.)

damage and did not require continuous nociceptive input from the site of injury. That is, the hyperalgesia persisted even after the cessation of acid administration *(25)*.

Therefore, an acidic milieu alone (without muscle damage) is sufficient to cause profound changes in the properties of nociceptors, axons, and dorsal horn neurons (i.e., the pain matrix). This model elegantly demonstrated that secondary mechanical hyperalgesia is maintained by neuroplastic changes in the central nervous system. Mechanical hyperalgesia is a hallmark of a MTrP. An acidic pH is well known to stimulate the production of bradykinin during local ischemia and inflammation and may further explain the cause of pain in patients with an active MTrP.

Significantly elevated levels of SP and CGRP were also found in the vicinity of the active MTrPs. As mentioned in the previous section, prolonged nociceptor activation is known to greatly increase the amount of SP that is transported orthodromically from

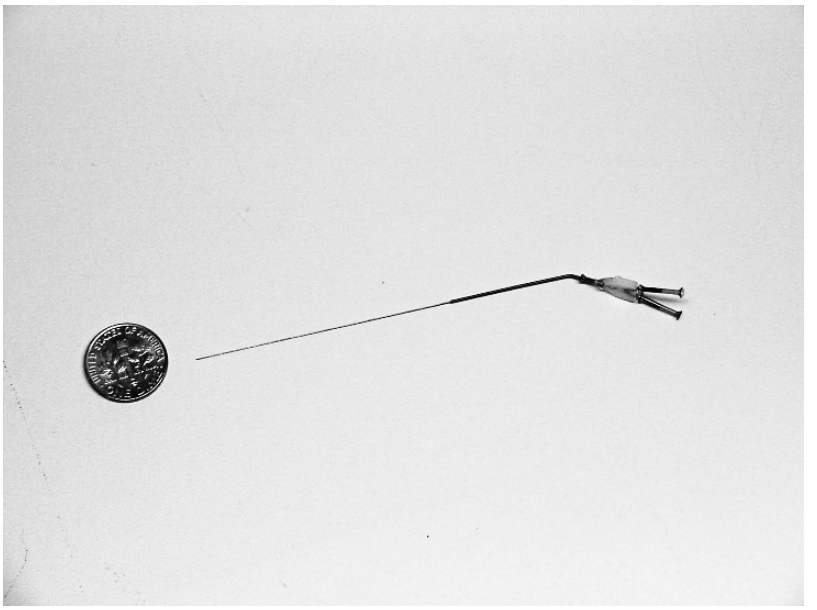

Fig. 4. Microdialysis needle. This 32-gauge device has the size, shape, and handling properties of an acupuncture needle and, likewise, is designed to be minimally invasive. (Shah JP, Phillips TM, Danoff JV, Gerber LH. An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. J Appl Physiol 2005 Nov;99*(5)*:1977–84. Epub 2005 Jul 21. Original is Figure 2 on page 1978 of article.)

the dorsal root ganglion into the dorsal horn of the spinal cord and this process can lead directly to neuroplastic changes. However, in this study, it was demonstrated that the neuropeptides SP and CGRP were transported antidromically along the primary afferent axon and secreted in the peripheral muscle tissue leading to concentrations that were significantly elevated in the subjects with active MTrPs. SP causes mast cell degranulation with the release of serotonin (in addition to histamine) and up regulation of pro-inflammatory cytokines such as TNF-α. Increases in TNF-α can in turn stimulate the production of norepinephrine.

In support of this effect of the elevated TNF-α, significantly elevated levels of serotonin and norepinephrine were found in subjects with active MTrPs. The increased levels of norepinephrine may be associated with increased sympathetic activity in the motor end plate region of the MTrP. In one study, intra-arterial injection of phentolamine, an α–adrenergic antagonist, decreased the spontaneous electrical activity from the locus of a myofascial trigger spot in rabbit skeletal muscle *(29)*. It is believed that this locus in the rabbit animal model is equivalent to the human MTrP. This finding supports the hypothesis that the autonomic nervous system is involved in the pathogenesis of MTrPs.

Interestingly, both SP and CGRP concentrations dropped significantly after the one local twitch response was elicited. The sudden drop in concentration of these analytes may be due to a local increase in blood flow following the LTR or some direct effect on the ability of the nociceptors to secrete neuropeptides, perhaps via mechanical hyperpolarization of the nerve ending. This effect also leads to the question of whether the commonly observed (at least temporary) decrease in pain after the

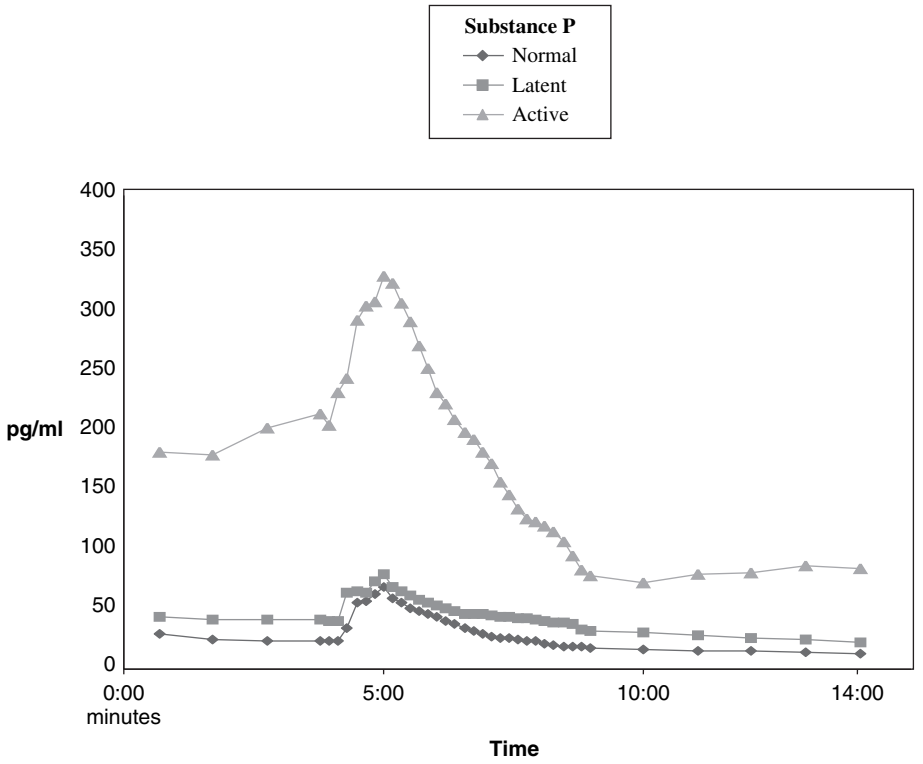

Fig. 5. Concentration of Substance P (SP) over time. A local twitch response (LTR) was elicited at 5 minutes in subjects in the Active and Latent groups. Only in the Active group was this followed by a rapid decline in SP to a concentration below that compared to initial needle insertion

release of a MTrP is causally related in some way to the change in concentration of these neuropeptides. Physiologically, the LTR may also induce chemical changes in the local environment of the nociceptor and thereby interfere with the responsiveness of the nociceptor membrane channels, raising its firing threshold and reducing pain transmission.

These experimental findings pose additional questions as to whether MTrPs are local phenomena, dependent upon a process of segmental sensitization initiated by some form of local trauma, which ultimately persists; or whether individuals who develop active MTrPs have a more systemic neuroendocrine dysregulation that would lead to sites remote from active or latent MTrPs to have similar biochemical profiles to the MTrP itself. These remote sites should be evaluated to help distinguish whether MTrPs have unique biochemical profiles or are associated with a more widespread phenomenon. To answer this question an unaffected muscle without MTrPs, the gastrocnemius, was identified and sampled for its biochemical milieu in subjects with active, latent, and absent MTrPs in the upper trapezius in a new series of subjects using the same paradigm as aforementioned.

Preliminary unpublished findings confirmed that at needle insertion in the upper trapezius muscle the concentrations of the analytes such as SP, CGRP, serotonin, bradykinin, TNF-α and so on were significantly higher in the *Active* group when compared to the *Latent* and *Normal* groups. Significant differences in analyte levels at

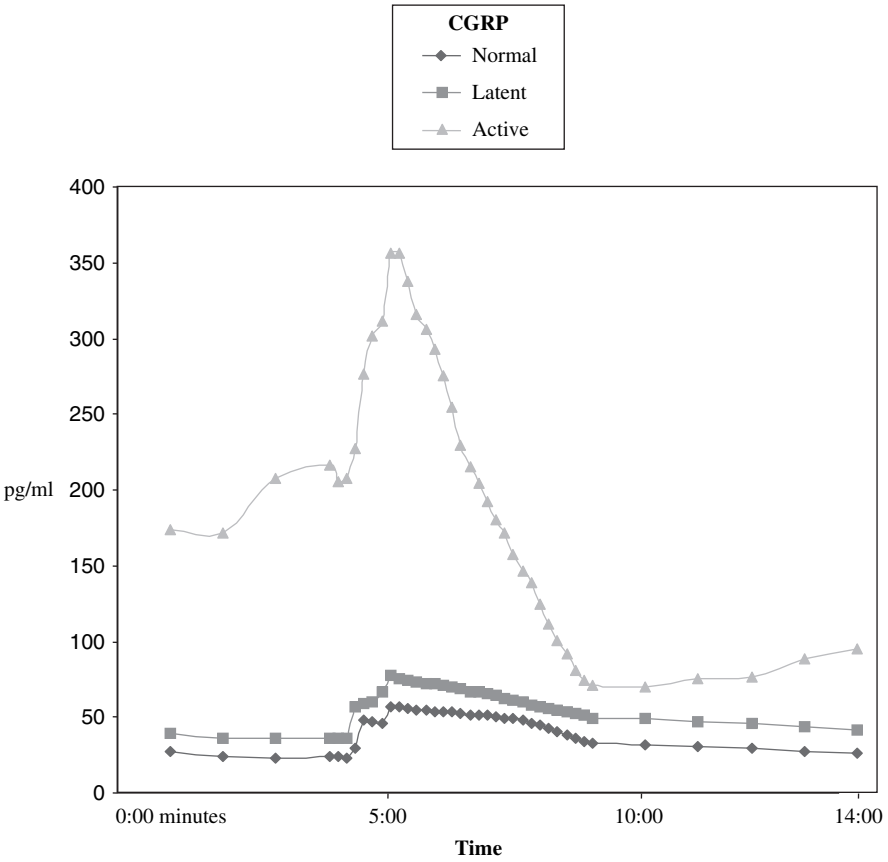

Fig. 6. Concentration of calcitonin gene-related peptide (CGRP) over time. A local twitch response (LTR) was elicited at 5 minutes in subjects in the Active and Latent groups. Only in the Active group was this followed by a rapid decline in CGRP to a concentration below that compared to initial needle insertion

needle insertion were demonstrated between the area sampled in the trapezius (active MTrP) and the normal, non-painful gastrocnemius in the same subject, suggesting that the vicinity of the active MTrP exhibits a unique biochemical milieu of substances associated with pain and inflammation. Further studies and analyses are on going.

6. CONCLUSION

Myofascial trigger points are a common cause of neuromusculoskeletal pain and dysfunction. Active myofascial trigger points function as dynamic foci of peripheral nociception that can initiate, accentuate, and maintain central sensitization and chronic pain states. Continuous nociceptive input from myofascial trigger points can increase excitability of dorsal horn neurons (causing allodynia and hyperalgesia) and open ineffective synapses—resulting in new receptive fields and referral of pain.

Until now, the distinction between active and latent myofascial trigger points depended solely upon the palpation skills and clinical acumen of the examiner and the subjective responses of the patient. Active and latent myofascial trigger points share the same physical findings—i.e., a hyperirritable nodule in a taut band of muscle. As

a result clinicians must rely on historical factors such as the description of pain at rest and provocative tests, attempting to reproduce the patient's pain and referral pattern with deep palpation. In clinical practice, the diagnosis of myofascial pain is confirmed retrospectively if, after identification and treatment of the myofascial trigger points, the patient's pain is eliminated or reduced. However, given the lack of high quality randomized controlled studies using standard treatment protocols for MPS, the benefit an individual patient may receive by local treatment is often attributed to the placebo effect.

The application of a minimally invasive micro-analytical technique to sample the biochemical milieu of active versus latent MTrPs demonstrates that there is now objective data to confirm the clinical distinction between subjects with active myofascial trigger points from those with either latent myofascial trigger points or muscle without myofascial trigger points in the upper trapezius. Furthermore, the biochemical milieu of the *Active* group is distinguished by elevated concentrations of a variety of biochemicals (inflammatory mediators, neuropeptides, catecholamines cytokines, etc.) that can sensitize and/or activate muscle nociceptors, thereby providing an enhanced explanatory model of why they have pain.

In essence, then, by learning to carefully examine and distinguish between active and latent myofascial trigger points when evaluating patients with chronic pain and dysfunction one can, in effect, identify the presence of a facilitated nervous system. This which involves changes both in the peripheral nociceptor and centrally in the dorsal horn leading to central sensitization and pathological neuroplastic changes in pain processing.

This data also supports that acupuncture dry needling may be an effective technique for deactivating myofascial trigger points. Uncovering the biochemical profile of active myofascial trigger points and determining the local effects of needle insertion may help elucidate mechanisms behind the initiation and amplification of myofascial pain, allowing targeted pharmacologic treatments. It may also explain how acupuncture dry needling and other physical modalities work at the local level.

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