Dry needling — peripheral and central considerations

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Dry needling is a common treatment technique in orthopedic manual physical therapy. Although various dry needling approaches exist, the more common and best supported approach targets myofascial trigger points. This article aims to place trigger point dry needling within the context of pain sciences. From a pain science perspective, trigger points are constant sources of peripheral nociceptive input leading to peripheral and central sensitization. Dry needling cannot only reverse some aspects of central sensitization, it reduces local and referred pain, improves range of motion and muscle activation pattern, and alters the chemical environment of trigger points. Trigger point dry needling should be based on a thorough understanding of the scientific background of trigger points, the differences and similarities between active and latent trigger points, motor adaptation, and central sensitize application. Several outcome studies are included, as well as comments on dry needling and acupuncture.

Keywords: Myofascial pain, Trigger points, Sensitization, Pain, Dry needling

Introduction

Over the years, dry needling has become a popular treatment technique in manual physical therapy.[1] Physical therapists and other healthcare providers in many countries employ dry needling in the clinical management of patients with myofascial pain and trigger points. In the USA, approximately 20 states and the District of Columbia have approved dry needling by physical therapists, which is a dramatic increase since 2004, when only four states approved dry needling.[2] In 2009, the American Academy of Orthopaedic Manual Physical Therapists adopted a position statement that dry needling is within the scope of manual physical therapy. The advantages of dry needling are increasingly documented[3] and include an immediate reduction in local, referred, and widespread pain,[4–7] restoration of range of motion and muscle activation patterns,[5,8,9] and a normalization of the immediate chemical environment of active myofascial trigger points.[10,11] Dry needling can reduce peripheral and central sensitization.[4]

Popular explanations of myofascial pain tend to be relatively simplistic and do not always offer a well-evidenced theoretical foundation to direct clinical treatment strategies.[1] Historically many researchers and clinicians have considered a vicious cycle hypothesis, known as the pain–spasm–pain cycle, which postulated that muscle pain would cause spasm of the same muscle, and in turn would cause more pain leading to more spasms.[12] The concept is based on the assumption that pain would excite alpha-motor neurons and possibly even gamma-motor neurons. There is, however, experimental and human evidence that both alpha- and gamma-motor neurons generally are inhibited by nociceptive input from the same muscle.[13–17] Animal data confirmed that a change in muscle spindle sensitivity may alter proprioceptive functioning, but there is no evidence of facilitation of spindle activity.[18] In other words, muscle pain does not appear to cause an increase in fusimotor drive.[19] Nevertheless, proponents of this concept continue to suggest that trigger points are the result of dysfunctional muscle spindle activation.[20] Although the pain–spasm–pain cycle is frequently referenced, it is a refuted concept based on an outdated and simplified understanding of the structure and function of alpha- and gamma-motor neurons.[21,22]

The updated pain-adaptation model may reflect more accurately the current thinking. According to this model muscle pain inhibits alpha-motor neurons leading to activation of antagonists and an overall decrease in motor function.[23] Even so, these patterns are not universally applicable either. Martin *et al.* demonstrated that muscle nociception resulted in excitation of both elbow flexor and extensor

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muscles,[24] while others found that the activity of motor neurons is not necessarily uniformly decreased.[25–29] A new motor adaptation model has been proposed.[22]

Although various needling approaches are commonly referred to as 'dry needling', it is important to realize that there are significant differences between schools of dry needling, their specific needling techniques, underlying philosophy or rationale, and duration of training programs. Each approach appears to address particular aspects of the total picture. Different dry needling techniques have been promoted to treat various forms of soft tissue dysfunction.[30–32]

Contemporary schools approach dry needling from a broad pain sciences perspective.[30,32,33] For example, Ma has developed a dry needling approach based on clinical applications of pain sciences and he maintains that his 'integrative systemic dry needling' is required to restore and maintain normal physiology of soft tissues and to reduce systemic stress to improve homeostasis.[32,33] To date, there are no research studies of Ma's needling approach. The 'intramuscular stimulation' dry needling approach developed by Gunn is one of the first medical dry needling approaches. Gunn considers myofascial pain to be secondary to neuropathy.[31] A few studies demonstrated the efficacy of intramuscular stimulation, but there are no studies that validate the underlying theoretical assumptions.[1,34,35] Dommerholt and Huijbregts focused on dry needling of trigger points, which occasionally has been interpreted erroneously as a more 'local' approach.[30] Trigger point dry needling has local and widespread effects[5,7] and influences remote parts of the body.[6,36,37] A superficial and a deep technique have been developed, whereby proponents of superficial needling suggest that the intervention targets primarily peripheral sensory afferents, while deep trigger point dry needling targets mostly dysfunctional motor units.[38,39]

To better appreciate the potential therapeutic role of dry needling, a review of the current research on myofascial trigger points follows within the context of pain sciences. The therapeutic effects of dry needling can only be understood against a pain management background. Therefore, review will focus on sensory and motor mechanisms relevant to dry needling, and indirectly on the application of dry needling. Unless indicated otherwise, references to dry needling in this article should be interpreted as trigger point dry needling based on the work of Travell, Simons and Lewit.[7,40,41]

Dry needling is relatively easy to learn for qualified healthcare providers, which may include manual physical therapists, physicians, dentists, chiropractors, and acupuncturists. A solid background and education in anatomy, physiology, and pain sciences are prerequisites. To use dry needling as an effective therapeutic modality, clinicians must learn how to identify trigger points. Dry needling requires training and practice in order to develop the sensitivity to appreciate subtle changes in tissue compliance and an awareness of the structures in the vicinity of the trigger points.[42] Most complications can be avoided by knowing the local anatomy, and by careful identification of the anatomical landmarks relevant to the muscle that is to be needled. Dry needling requires a well-developed kinesthetic awareness and visualization of the pathway the needle takes within the body.[3] Several studies have shown that experienced physicians, physical therapists, and chiropractors can reach acceptable degrees of inter- and intrarater reliability.[42–49] In a recent study, experienced clinicians reached good agreement, but inexperienced clinicians did not reach acceptable levels of agreement in spite of having completed a brief training program to improve standardization of the research protocol.[48] Trigger points can be verified objectively using magnetic resonance or ultrasound elastography[50–52] or with intramuscular electromyography,[53–55] but these techniques are not yet easily applicable to clinical practice at this time.

Active and Latent Myofascial Trigger Points

Trigger points are divided into active and latent trigger points. Active trigger points feature spontaneous local and referred pain away from the trigger point, while latent trigger points do not cause spontaneous pain. After stimulation with digital pressure, however, latent trigger points do evoke local and referred pain. In other words, both active and latent trigger points cause allodynia at the trigger point site and hyperalgesia away from the trigger point following applied pressure. Referred pain from active trigger points may mirror the formation of new effective central nervous connections, meaning that afferent fibers from trigger point nociceptors may make new effective connections with dorsal horn neurons that normally only process information from remote body regions.[56,57] A nociceptor is a receptor specialized in detecting stimuli that objectively can damage tissue and subjectively are perceived as painful.[56] In clinical practice, a trigger point is considered active if the elicit pain is familiar to the patient.

Active trigger points featured significantly lower pain thresholds with electrical stimulation in the muscle, the overlying cutaneous and subcutaneous tissues. In latent trigger points, the sensory changes did not involve cutaneous and subcutaneous tissues.[58,59] Several studies have shown, however, that latent trigger points do provide nociceptive input into the dorsal horn even though they are not spontaneously painful.[60–66] It is not entirely clear why this occurs.

Mense speculated that certain regions within a muscle may only be connected via ineffective synapses to dorsal horn neurons, which supply regions remote from the muscles with trigger points. This would explain why latent trigger points may not trigger spontaneous pain. Once these ineffective synapses are sensitized, referred pain would follow.[57] Latent trigger points can quickly become active trigger points. Because of increased synaptic efficacy in the dorsal horn, these trigger points would start featuring spontaneous pain. It appears that whether a trigger point is active or latent depends at least partially on the degree of sensitization.

Evidence suggests that the first phase of trigger point formation consists of the development of contractured muscle fibers or a taut band, which may or may not be painful.[67] While the exact mechanisms of the taut band formation are not well defined, an excessive release of acetylcholine at the motor endplate, combined with an inhibition of acetylcholine esterase, an upregulation of nicotinic acetylcholine receptors, and other modulating factors are hypothesized to trigger the development of localized muscle contractures.[68,69] This is expressed in the 'integrated trigger point hypothesis' developed by Simons[69] and recently expanded by Gerwin *et al.*[68] and by McPartland and Simons.[70]

Characteristic of taut bands and trigger points is that they do not require an electrical activation of the alpha-motor neuron, but get activated by a spontaneous release of acetylcholine from the motor endplate.[68] Endplate dysfunction has been confirmed by multiple animal model and human studies.[55,71–79] Kuan and colleagues found a correlation between the irritability of trigger points and the prevalence of endplate noise,[39] and confirmed that blocking the release of acetylcholine with administration of botulinum toxin reduced the prevalence of endplate noise.[80] Several other studies have also shown that the administration of botulinum toxin can reduce the activity of trigger points.[81–87] Therefore, trigger points are found in close vicinity of motor endplates, which are spread out throughout the entire muscle.[88–90] Active trigger points are clustered around motor endplates and feature more endplate noise than latent trigger points, which once again supports that active trigger points are more sensitized.[39,65,91] There is some evidence that trigger points may have more 'jitter' than normal muscle,[72,92] but not all studies confirmed this.[93] Neuromuscular jitter is produced by fluctuations in the time for endplate potentials at the neuromuscular junction to reach the threshold for action potentials.[72]

Motor Aspects of Trigger Points

Trigger points are thought to develop especially following unaccustomed eccentric and concentric loading,[68] but also occur after low-load repetitive tasks and sustained postures,[94,95] with respiratory stress, such as over-breathing,[96,97] and in association with visceral pain and dysfunction.[98–101] It is conceivable that initially the taut band formation reflects a normal physiologic, protective, and stabilizing mechanism, for example, associated with damage or potential muscle damage, joint hypermobiity, visceral dysfunction, or abnormal breathing patterns. Prolonged contractures are likely to lead to the formation of latent trigger points, which can evolve into active trigger points. Once active trigger points exist, there will be a constant nociceptive input into the dorsal horn, which can perpetuate altered motor control strategies, lead to further muscle overload or even disuse, and result in the development of peripheral and central sensitization.[57,102,103]

From a motor perspective, the development of trigger points may be dependent on perceived or actual tissue damage, but there are only a few scientific studies of the activation patterns of trigger points. Muscle pain can modulate joint function and stability and increase the risk of joint injury.[104–106] Joint dysfunction, as seen for example with osteoarthritis, can also cause muscle hyperalgesia.[107] Treatment of trigger points around the involved joint is effective in reducing the pain associated with arthritis.[108,109] This brings up the question whether typical motor adaptations are common with myofascial pain. Surprisingly, little is known about motor adaptation and myofascial pain.

Hodges and Tucker recently proposed a new motor adaptation theory,[110] and although they did not consider the influence of trigger points, several key aspects of their theory may actually apply to trigger points. Hodges agreed that the vicious pain cycle and pain adaptation hypotheses are inadequate models of motor adaptation.[22] Instead, he proposed that a redistribution of activity within and between muscles must occur. Adding trigger points to the new theory, it is clear that they change the activity within muscles. In this respect, it is also noteworthy that not all regions within a muscle are equally prone to the development of trigger points.[94] The intramuscular pressure is not evenly distributed, which may contribute to intramuscular hypoxia and trigger point formation.[111] Trigger points do alter the activity between muscles.[6,36,112–115] Lucas and colleagues found altered movement activation patterns in shoulder abduction in subjects with latent trigger points in their shoulder musculature.[8,9] As reviewed previously, latent trigger points do not feature spontaneous pain, but they do provide nociceptive input. In the evaluation of patients with trigger points, clinicians should assess which modifications a particular patient has made, subsequently attempt to

determine why the adaptation was made and lastly, why it did not lead to satisfactory resolution of the pain problem.[116]

Hodges further postulated that 'pain would change the mechanical behavior such as modified movement and stiffness, which would lead to 'protection' from further pain or injury, or threatened pain or injury'.[22] Patients with myofascial trigger points have characteristic taut bands, which may be considered as a means to splint a body region.[120] Muscles harboring trigger points cause restrictions in range of motion.[5,117–119] Trigger points are commonly observed in muscles crossing an arthritic joint, although frequently trigger points occur even near non-arthritic joints.[108,109] Perhaps trigger points are a means of assisting sustained increased contractures.[120] In addition, myofascial trigger points inhibit overall muscle function, leading to muscle weakness without atrophy. Patients with myofascial pain commonly present with abnormal breathing patterns, such as hyperventilation, which leads to respiratory alkalosis.[96] Chaitow reviewed that under these circumstances, muscles are prone to develop trigger points, fatigue, and cramping.[96] Of interest is that myofascial treatment programs that include correction of breathing patterns are highly successful even with chronic pain patients.[121,122]

Hodges has also suggested that inhibition or facilitation of agonist and antagonists occurs, which is a common pattern seen in patients with myofascial pain. He proposed that the motor adaptation 'is not explained by simple changes in excitability, but involves changes at multiple levels of the motor system and these changes may be complementary, additive or competitive',[22] which applies to myofascial pain as well. Lastly, myofascial trigger points may offer some short-term benefit, but in the long run, they are disabling and a source of much unnecessary human suffering.

Muscle Pain and Trigger Points

Muscle pain is not always appreciated as a primary entity and frequently is only considered as a secondary phenomenon to tendonitis, whiplash, inflammation, or injuries to joints or nerves.[123–131] Nevertheless, muscle pain is a common phenomenon recognized by the International Association for the Study of Pain.[132] Muscle pain is associated with many chronic pain conditions. It is difficult to pinpoint and diffuse in nature. Muscle pain is inhibited strongly by descending pain-modulating pathways and under normal circumstances, there is a dynamic balance between the degree of activation of dorsal horn neurons and the descending inhibitory systems.[133] Muscles refer to deep somatic structures, but not to skin, although many neurons with muscle input also have additional receptive fields in the skin. A receptive field is defined as the body region from which a neuron can be excited or inhibited.[56]

Considering the relevancy of myofascial trigger points from a pain science perspective, it is not surprising that pain management specialists consider myofascial pain and trigger points to be clinically important.[134,135] Trigger points are peripheral sources of persistent nociceptive input, which can excite muscle nociceptors.[4,57,63,65,136–139] Nociceptive input from muscle is particularly effective in inducing neuroplastic changes in the spinal dorsal horn and likely in the brainstem.[140,141] Dry needling may be instrumental in reversing such neuroplastic changes by removing a constant and intense nociceptive source. Nociceptive input enters the spinal cord primarily via thinly myelinated group III or unmyelinated group IV afferent fibers.[57] Since dorsal horn neurons are convergent neurons, meaning that they receive information from many other sources, including joints, viscera, fascia, and the skin, not all input will lead to action potentials.[142] Each spinal neuron has multiple synaptic contacts, which can be excitatory or inhibitory, effective or ineffective, continuous, active or silent. The final outcome is determined by the combined input from all different sources.[57]

Sustained contractures of taut bands cause local ischemia and hypoxia in the core of trigger points.[143] Recent Doppler ultrasound studies confirmed significantly different blood flow waveforms and a greater vascular output resistance in active trigger points when compared to latent trigger points and normal muscle tissue.[144] Outside the immediate environment of active trigger points, an increased vascular bed was observed, which is consistent with the measurement of increased oxygen saturation levels outside the core of trigger points.[143,144] Hypoxia may trigger an immediate increased release of acetylcholine at the motor endplate.[71] As a side note, myofascial tension, as seen in trigger points, may also enhance the excessive release of acetylcholine, which suggests the presence of a self-sustaining vicious cycle.[145,146]

Low oxygen levels lead to a significant drop in pH. In active trigger points, the pH may be well below 5, which is more than sufficient to excite muscle nociceptors.[11,147–149] Muscle nociceptors are dynamic structures than can be modified depending upon the local tissue environment. They play an active role in the maintenance of normal tissue homeostasis by sensing the peripheral biochemical milieu and by mediating the vascular supply to peripheral tissue. A low pH activates acid sensing ion channels (ASICs) and transient receptor potential vanilloid (TRPV) receptors, which in turn contribute to mechanical hyperalgesia and central sensitization.[150–153] Various

kinds of ASICs play different roles in the development of hyperalgesia,[154] i.e. ASIC3 is important for inflammatory pain and ASIC1a is involved in central sensitization and in processing noxious stimuli.[153] Repeated intramuscular injections of acid saline in rats activated N-methyl-D-aspartate (NMDA) receptors in the brainstem and other parts of the central nervous system.[155] A low pH downregulates acetylcholine esterase and triggers the release of several nociceptive substances, such as calcitonin gene-related peptide (CGRP), adenosine triphosphate (ATP), bradykinin (BK), serotonin (5-HT), prostaglandins (PGs), potassium, and protons.[156] ATP is one if the most important activating substances of muscle nociceptors by binding to P2X3 receptors. There are many interactions between these substances. For example, the combination of ATP and acid increases the pH sensitivity of the ASIC3 receptor.[157] Combinations of BK and 5-HT produce more muscle hyperalgesia than each chemical alone.[158,159] BK, PG, and 5-HT are not only very effective at sensitizing or activating muscle nociceptors, but they can also cause local vasodilation, which can lead to mechanoreceptor activation by distorting the normal tissue relationships. A sensitized muscle nociceptor has a lowered stimulation threshold into the innocuous range and will respond to harmless stimuli like light pressure (allodynia) and muscle movement (mechanical hyperalgesia). Most data are derived from animal studies as there are only few human research on muscle nociceptor activation.[160,161]

Central Sensitization and Trigger Points

Central sensitization has been described in association with many chronic pain syndromes,[162] such as endometriosis,[163] low back pain,[164] irritable bowel syndrome,[165] surgical pain,[166] whiplash,[167,168] shoulder impingement,[169] and fibromyalgia,[167,170,171] and as such, sensitization is not specific for myofascial trigger points. Trigger points are, however, involved in nearly every pain syndrome[131] and it is likely that central sensitization involves trigger points, as has been shown for whiplash,[172] tension-type headaches,[139,173–175] chronic primary headaches,[176] migraines,[177,178] lateral epicondylalgia,[179,180] breast cancer surgery,[136,181–184] fibromyalgia,[4,137,185] and temporomandibular disorders,[186] among others.

Awareness and recognition of the presence and underlying mechanisms of central sensitization are critical in manual physical therapy.[187] In clinical practice, it can be challenging to objectively determine whether a patient's musculoskeletal pain involves central sensitization. There is some evidence that an impaired nociceptive flexion reflex may be a valid indication of altered central nervous system processing.[188] As Lim *et al.* summarized, the nociceptive flexion reflex is a physiological measure that is commonly made from the biceps femoris muscle following electrical stimulation of the sural nerve. It involves the lowest noxious stimulation intensity required to trigger a reflex without stimulating peripheral nociceptors.[188]

Patients had significantly worse outcomes when they presented with relatively high levels of central sensitization, including hyperalgesia and referred pain, before subacromial decompression surgery.[169] Dry needling and trigger point injections commonly elicit and eliminate local and referred pain patterns or areas of secondary hyperalgesia.[178,189–191] As a side note, the effects of injections are comparable to dry needling.[192] The outcomes of subacromial decompression would conceivably have been much improved after central sensitization would have been addressed with trigger point therapy including dry needling, injections, or manual inactivation.[169,193–196] The same applies to the other listed diagnoses, i.e. trigger point needling decreased the overall sensitivity in patients with fibromyalgia and decreased pain and increased range of motion in whiplash, post-mastectomy, and temporomandibular patients.[4,5,172,197]

Patients with a hypersensitive trigger point in the upper trapezius muscle exhibited significantly enhanced somatosensory and limbic activity and decreased activity in the dorsal hippocampus compared with control subjects.[198] Using functional magnetic resonance imaging, Niddam *et al.* showed that pain following the insertion of a needle into a trigger point combined with electrical stimulation is mediated through the periaqueductal gray in the brainstem.[199] Central sensitization is the mechanism of referred pain from trigger points, which Travell and Simons described for most musculoskeletal muscles.[40,200] The mechanisms of muscle referred pain have been described in detail by Hoheisel, Mense, Arendt-Nielsen, and Graven-Nielsen, among others, and involve sensitization and an expansion of receptive fields.[56,201–207]

The immediate environment of active trigger points is characterized by significantly increased levels of substance P (SP), CGRP, BK, 5-HT, norepinephrine, tumor necrosing factor-alpha, and interleukin-1beta compared to latent trigger points and normal muscle tissue.[11,147,208] These chemicals sensitize and activate not only muscle nociceptors, but can also activate glia cells. Whether trigger points stimulate glia cells is not clear, as different studies show conflicting results. Chacur and colleagues demonstrated that chronic muscle lesions can activate microglial cells,[209] but others suggested different mechanisms.[210–213] Irrespective of the mechanism, myofascial trigger points become sources of ongoing nociceptive input

into the dorsal horn and contribute to and maintain central sensitization including referred pain.[57,139] Subjects with active trigger points in the upper trapezius muscle presented even with slightly increased levels of the same substances in the medial gastrocnemius muscle, possibly due to widespread sensitization.[11]

Unfortunately, glutamate levels could not be measured with the microdialysis methodology used previously, however, others have demonstrated increased intramuscular levels of glutamate associated with myalgia[214–219] and it is very likely that glutamate is involved with trigger points. Glutamate can activate the NMDA and alpha-amino-3-hydroxy-5-methyl-4- isoxazolepropionic acid (AMPA) receptors. Under normal circumstances, only the AMPA receptor is active, but the receptor does not respond to brief noxious stimuli. With prolonged and intense nociceptive input, SP is also released, which makes the NMDA receptor responsive to glutamate. As a result, an influx of Ca^{2+} ions initiates a cascade of events that results in the new synthesis of AMPA receptors at what were previously ineffective synapses. The new AMPA receptors do respond to brief noxious input. The release of SP in the dorsal horn can increase the efficacy of synaptic connections in the spinal cord, allowing the multi-segmental spread of noxious input, which clinically is known as referred pain.[220]

There are many other mechanisms involved in muscle pain and peripheral and central sensitization, such as serotonergic mechanisms. For example, the serotonin antagonist tropisetron inhibited the pronociceptive or pain-promoting effect of serotonin at trigger points.[221] Other relevant substances include nerve growth factor,[222] which can also stimulate TRPV receptors,[223] brain-derived neurotrophic factor,[224,225] and nitric oxide,[226,227] but a detailed discussion of their potential roles is beyond the scope of this review.

Dry Needling and Trigger Points

There is overwhelming scientific evidence that trigger points are not just peripheral phenomena limited to muscles. Treatments directed at inactivating trigger points do have an impact on central processes by removing a common and peripheral source of persistent nociceptive input. The main difference between dry needling and manual trigger point release is its specificity. It is interesting that a meta-review concluded that there is insufficient evidence for dry needling.[228] This review included only a small portion of published papers.[3] A Cochrane review concluded that 'dry needling appears to be a useful adjunct to other therapies for chronic low back pain'.[229] Inactivation of latent trigger points with dry needling or with manual pressure techniques may prevent the development of active trigger points and reduce and in many cases remove their nociceptive input, normalize the synaptic efficacy, and reduce peripheral and central sensitization.[60] After eliciting a local twitch response with a needle, SP and CGRP were significantly reduced in active trigger points, which corresponds with the clinically observation of an immediate decrease in pain and local tenderness after the inactivation of a trigger point with dry needling.[10,11] We already mentioned that dry needling can restore range of motion and muscle activation patterns,[5,8,9] and reduce local, referred, and widespread pain.[4–7,36] Dry needling of trigger points can reduce the endplate noise associated with those trigger points[74] and with remote trigger points.[6] Dry needling of trigger points or acupuncture points in the forearm reduced the endplate noise in the upper trapezius muscle.[37,230] Patients with hemiparetic shoulder syndrome reported less severe and less frequent pain, required less analgesic medication, restored normal sleep patterns, and demonstrated increased compliance with the rehabilitation program after having been treated with dry needling.[231] Dry needling of trigger points resulted in a significant reduction of pain and showed significant improvements on the Geriatric Depression Scale in an elderly patient population.[35] Dry needling showed comparable effects to injections with lidocaine,[192,232] but dry needling was superior in its long-term reduction of pain.[232] There is even some evidence from animal studies that the anti-nociceptive effects of dry needling may at least partially be mediated through oxytocinergic mechanisms, which means that dry needling may trigger the central release of oxytocin.[233,234]

It is nearly impossible to develop double blind, placebo-controlled studies of dry needling or acupuncture, given the invasive nature of the stimulus.[235,236] In acupuncture, sham needling is often performed with superficial needling of non-acupuncture point locations, which is problematic as any needling is likely to have a physiological effect, such as a release of endorphins, a change in pain thresholds, or an expectancy of a positive outcome.[237–241] Therefore, studies comparing acupuncture or dry needling with sham needling may actually compare two treatment regimens.[242] In some studies, sham needling is attempted by tapping a von Frey monofilament on the skin;[243] however, both the actual needling and the tapping can induce specific brain responses, which means that tapping is not a suitable sham procedure either. The observation that both needling and sham acupuncture caused specific changes emphasizes the importance of including control groups in studies.

Others have used the so-called Streitberger needle, which gives subjects the impression of being needled, but the needle disappears into the needle shaft.[244–247] Placebo responses are processed in frontal cortical areas involved in generating and maintaining cognitive expectancies.[248] When comparing acupuncture, sham acupuncture using a Streitberger needle, and skin prick, Pariente and colleagues established that patients' expectations and belief regarding a positive outcome activated the dorsolateral prefrontal cortex and the anterior cingulate cortex.[240] Other functional magnetic resonance studies have confirmed that expectancy can significantly influence acupuncture analgesia.[249–251] A recent study concluded that patients with a high degree of dispositional optimism and low state anxiety were particularly receptive to placebo responses.[252] It is likely that similar issues must be considered when designing dry needling studies.

Considering the difficulties in designing placebo controlled research, Mayoral del Moral completed an interesting dry needling study of 40 subjects scheduled for knee replacement surgery.[253] All subjects were examined for the presence of trigger points and randomly assigned to one of two groups. Immediately following anesthesiology, but before the actual surgery, subjects in the intervention group received dry needling of their trigger points, while subjects in the control group were not treated. As all patients were anesthesized, they were truly blinded to the group allocation and intervention. Subjects who were treated with dry needling reported significantly lower pain levels and required fewer analgesics following the surgery.[253]

Dry Needling and Acupuncture

Although the focus of this article is on peripheral and central considerations related to dry needling, a few observations regarding acupuncture and dry needling are included here. Dry needling is often compared with and contrasted to acupuncture. Manual physical therapists must realize that dry needling is also within the scope of acupuncture practice. Statements that dry needling would not be in the scope of acupuncture are inaccurate and counterproductive and not based on accurate knowledge of contemporary acupuncture practice.[254] A formal complaint to the Maryland Board of Acupuncture by a Maryland-based physical therapist reporting that an acupuncturist would be practicing physical therapy without a license when using dry needling techniques spurred an investigation by the Maryland Attorney General and endangered the scope of physical therapy practice in that state.[257]

In the context of acupuncture treatments, dry needling would be considered a technique of acupuncture. Dry needling is, however, not in the exclusive scope of any discipline.[255–257] Dry needling is performed with the same solid filament needle acupuncturists employ, but dry needling does not require any knowledge of traditional acupuncture theory or Oriental health concepts.[1] Although many US state acupuncture statutes refer to acupuncture as a discipline based on Oriental medicine and the journal of the American Association of Acupuncture and Oriental Medicine (AAAOM) is targeted specifically to 'practitioners of Oriental Medicine', Hobbs emphasized that acupuncture is not necessarily 'limited to its historical roots and centuries' old theory, but is also a dynamic, evolving modern medical practice, which incorporates the use of neuroanatomical terminology'.[258] In other words, acupuncture is not necessarily always based on or limited to Oriental medicine concepts; contemporary schools of acupuncture usually include some education in Western medical principles.[259] Nevertheless, a 2008 report by the National Commission for the Certification of Acupuncture and Oriental Medicine (NCCAOM) showed that 80% of diplomates in acupuncture practiced Traditional Chinese Medicine (TCM) and less than 40% of practitioners practiced other approaches, such as 'auricular, laser, electroacupuncture, color puncture, and trigger point therapy', among others.[260]

Very few schools of acupuncture include the assessment, identification, and dry needling techniques of myofascial trigger points.[254] An online review of the curricula of US acupuncture school revealed only one school that mentioned trigger point dry needling (Dommerholt, 2011, unpublished data). In 2003, the NCCAOM reported that only 3.7% of acupuncturists used trigger point therapy as their primary practice tradition.[261] The 2002 NCCAOM acupuncture examination included only one question related to trigger points and motor points.[261] There are no inter-rater reliability studies of acupuncturists identifying trigger points. One study showed very poor inter-rater reliability of TCM diagnosis and treatment of persons with chronic low back pain. Six experienced TCM practitioners examining the same six patients on the same day made 20 different diagnoses and selected only one common acupuncture point. The researchers concluded that the differences among diagnoses and treatment recommendations depended more on the practitioner than on the patient.[262]

Some US state statutes define acupuncture in much broader terms. The Arizona statutes, for example, define acupuncture as 'puncturing the skin by thin, solid needles to reach subcutaneous structures, stimulating the needles to affect a positive therapeutic response at a distant site and the use of adjunctive therapies'.[263] The

statutes also include language that they do not apply to 'health care professionals […] practicing within the scope of their license' leaving the practice of dry needling available to other disciplines.[263] Generally speaking, statutes of one professional discipline should not restrict the scope of practice of another discipline. The Attorney General of Maryland determined that the Maryland Board of Physical Therapy Examiners is authorized to consider solid filament needles as 'mechanical devices' consistent with the state's physical therapy statutes. According to the Attorney General, 'the authority to use acupuncture needles for therapeutic purposes is not necessarily reserved exclusively to licensed acupuncturists […]. State law recognizes that the scope of practice of health care professions may overlap…'.[257] When Travell developed the concepts of myofascial pain and trigger points, she never considered the practice and concepts of acupuncture.[264] Later in life, she did interact with acupuncturists, but by that time the concept of trigger points was already well established.[254] In other words, the concept of trigger points and dry needling was developed independently of already existing acupuncture concepts.[265,266]

Within the acupuncture community, disagreement exists whether trigger point needling is similar to needling of so-called ah-shi points.[254,259,267–269] Ah-shi points belong to one of three major classes of acupuncture points. There are 361 primary acupuncture points referred to as 'channel' points and hundreds of secondary class acupuncture points, known as 'extra' or 'non-channel' points. The third class of acupuncture points is referred to as ah-shi points. By definition, ah-shi points must have pressure pain. Hong, Audette and Blinder suggested that acupuncturists may well be treating trigger points whenever they are treating ah-shi points.[270,271] While some believe that trigger points are nearly always acupuncture points especially in pain management,[272–275] well-known acupuncturist Birch maintains that at best there is only an 18%–19% overlap.[268,269]

Unfortunately, in recent years US acupuncture associations have opposed dry needling by physical therapists.[258,259,276] This is a US phenomenon and has no correlates in other countries. The Council of Colleges of Acupuncture and Oriental Medicine suggested that 'professions such as physical therapy and others also recognize the efficacy of acupuncture […] and are attempting to use acupuncture and rename it as a physical therapy technique',[258] which is an inaccurate reflection of the history of trigger points and dry needling within the context of medicine and physical therapy. The AAAOM has also interpreted the integration of dry needling within the scope of physical therapy and other disciplines as a 'clear effort to redefine identical medical procedures and thereby circumvent or obscure established rules and regulations regarding practice',[276] which from a physical therapy perspective is once again an inaccurate interpretation of the history of trigger points, myofascial pain, and dry needling. Efforts to initiate a dialogue between physical therapists and acupuncturists have fallen on apparent deaf ears. On the brighter side, Western Medical Acupuncture (WMA) is a form of acupuncture, which does not consider the Oriental heritage and practice of TCM[277] and practitioners of WMA are usually not opposed to dry needling by physical therapists or chiropractors.[256]

Summary

Dry needling or trigger point inactivation rarely is a stand-alone kind of intervention and is just one aspect of a comprehensive manual physical therapy approach. Dry needling is usually combined with other manual therapies[116,278–280] and should be considered an instrument-assisted manual therapy technique, similarly to other instrument-assisted manual therapy techniques such as the Graston Technique.[281,282] Dry needling is not solely in the scope of any one particular discipline. Overlap in scope of practice is not only inevitable; it may even be desirable to best meet the needs of patients. Dry needling is an easy to learn technique in the hands of qualified health care providers.

In this review, we have postulated that dry needling is a potent therapeutic measure to remove a constant source of peripheral nociceptive input originating from myofascial trigger points. As such, dry needling does not replace other manual physical therapy technique, but may be useful in facilitating a rapid reduction of pain and a return to function. A thorough understanding of the role of trigger points in peripheral and central sensitization is important in manual physical therapy practice. Trigger points can be inactivated with manual techniques and joint manipulations,[119,283] but dry needling may be a more efficient and quicker method.[1]

References

1 Dommerholt J, Mayoral del Moral O, Gröbli C. Trigger point dry needling. J Man Manipulative Ther 2006;14:E70–87.
2 Dommerholt J. Dry needling in orthopedic physical therapy practice. Orthop Phys Ther Pract 2004;16:15–20.
3 Dommerholt J, Gerwin RD. Neurophysiological effects of trigger point needling therapies. In: Fernández de las Peñas C, Arendt-Nielsen L, Gerwin RD, editors. Diagnosis and management of tension type and cervicogenic headache. Boston, MA: Jones & Bartlett; 2010. p. 247–59.
4 Affaitati G, Costantini R, Fabrizio A, Lapenna D, Tafuri E, Giamberardino MA. Effects of treatment of peripheral pain generators in fibromyalgia patients. Eur J Pain 2011;15:61–9.
5 Fernandez-Carnero J, La Touche R, Ortega-Santiago R, Galan-del-Rio F, Pesquera J, Ge HY, *et al.* Short-term effects of dry needling of active myofascial trigger points in the masseter muscle in patients with temporomandibular disorders. J Orofac Pain 2010;24:106–12.
6 Hsieh YL, Kao MJ, Kuan TS, Chen SM, Chen JT, Hong CZ. Dry needling to a key myofascial trigger point may reduce the

irritability of satellite MTrPs. Am J Phys Med Rehabil 2007;86:397–403.
7 Lewit K. The needle effect in the relief of myofascial pain. Pain 1979;6:83–90.
8 Lucas KR, Polus BI, Rich PS. Latent myofascial trigger points: their effects on muscle activation and movement efficiency. J Bodyw Mov Ther 2004;8:160–6.
9 Lucas KR, Rich PA, Polus BI. Muscle activation patterns in the scapular positioning muscles during loaded scapular plane elevation: the effects of latent myofascial trigger points. Clin Biomech 2010;25:765–70.
10 Shah J, Phillips T, Danoff JV, Gerber LH. A novel microanalytical technique for assaying soft tissue demonstrates significant quantitative biomechanical differences in 3 clinically distinct groups: normal, latent and active. Arch Phys Med Rehabil 2003;84:A4.
11 Shah JP, Danoff JV, Desai MJ, Parikh S, Nakamura LY, Phillips TM, *et al.* Biochemicals associated with pain and inflammation are elevated in sites near to and remote from active myofascial trigger points. Arch Phys Med Rehabil 2008;89:16–23.
12 Mandel LM, Berlin SJ. Myofascial pain syndromes and their effect on the lower extremities. J Foot Surg 1982;21:74–9.
13 Mense S, Skeppar RF. Discharge behavior of feline gamma-motoneurons following induction of an artificial myositis. Pain 1991;46:201–10.
14 Simons DG, Mense S. Understanding and measurement of muscle tone as related to clinical muscle pain. Pain 1998;75:1–17.
15 Burke D. Critical examination of the case for or against fusimotor involvement in disorders of muscle tone. Adv Neurol 1983;39:133–50.
16 Kniffki KD, Schomburg ED, Steffens H. Synaptic effects from chemically activated fine muscle afferents upon alpha-motoneurones in decerebrate and spinal cats. Brain Res 1981;206:361–70.
17 Le Pera D, Graven-Nielsen T, Valeriani M, Oliviero A, Di Lazzaro V, Tonali PA, *et al.* Inhibition of motor system excitability at cortical and spinal level by tonic muscle pain. Clin Neurophysiol 2001;112:1633–41.
18 Masri R, Ro JY, Capra N. The effect of experimental muscle pain on the amplitude and velocity sensitivity of jaw closing muscle spindle afferents. Brain Res 2005;1050:138–47.
19 Birznieks I, Burton AR, Macefield VG. The effects of experimental muscle and skin pain on the static stretch sensitivity of human muscle spindles in relaxed leg muscles. J Physiol 2008;586:2713–23.
20 Partanen JV, Ojala TA, Arokoski JP. Myofascial syndrome and pain: a neurophysiological approach. Pathophysiology 2010;17:19–28.
21 Mense S, Masi AT. Increased muscle tone as a cause of muscle pain. In: Mense S, Gerwin RD, editors. Muscle pain: understanding the mechanisms. Heidelberg: Springer; 2011. p. 207–49.
22 Hodges P. Pain and motor control: From the laboratory to rehabilitation. J Electrom Kinesiol 2011;21:220–8.
23 Lund JP, Donga R, Widmer CG, Stohler CS. The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. Can J Physiol Pharmacol 1991;69:683–94.
24 Martin PG, Weerakkody N, Gandevia SC, Taylor JL. Group III and IV muscle afferents differentially affect the motor cortex and motoneurones in humans. J Physiol 2008;586:1277–89.
25 Farina D, Arendt-Nielsen L, Graven-Nielsen T. Spike-triggered average torque and muscle fiber conduction velocity of low-threshold motor units following submaximal endurance contractions. J Appl Physiol 2005;98:1495–502.
26 Farina D, Arendt-Nielsen L, Merletti R, Graven-Nielsen T. Effect of experimental muscle pain on motor unit firing rate and conduction velocity. J Neurophysiol 2004;91:1250–9.
27 Tucker K, Butler J, Graven-Nielsen T, Riek S, Hodges P. Motor unit recruitment strategies are altered during deep-tissue pain. J Neurosci 2009;29:10820–6.
28 Tucker KJ, Hodges PW. Motoneurone recruitment is altered with pain induced in non-muscular tissue. Pain 2009;141:151–5.
29 Sohn MK, Graven-Nielsen T, Arendt-Nielsen L, Svensson P. Inhibition of motor unit firing during experimental muscle pain in humans. Muscle Nerve 2000;23:1219–26.
30 Dommerholt J, Huijbregts PA. Myofascial trigger points: pathophysiology and evidence-informed diagnosis and management. Boston, MA: Jones & Bartlett; 2011.
31 Gunn CC. The Gunn approach to the treatment of chronic pain. 2nd ed. New York: Churchill Livingstone; 1997.
32 Ma YT, Ma M, Cho ZH. Biomedical acupuncture for pain management; an integrative approach. St Louis, MO: Elsevier; 2005.
33 Ma YT. Biomedical acupuncture for sports and trauma rehabilitation: dry needling techniques. St Louis, MO: Churchill Livingstone; 2011.
34 Gunn CC, Milbrandt WE, Little AS, Mason KE. Dry needling of muscle motor points for chronic low-back pain: a randomized clinical trial with long-term follow-up. Spine 1980; 5:279–91.
35 Ga H, Koh HJ, Choi JH, Kim CH. Intramuscular and nerve root stimulation vs lidocaine injection to trigger points in myofascial pain syndrome. J Rehabil Med 2007;39:374–8.
36 Srbely JZ, Dickey JP, Lee D, Lowerison M. Dry needle stimulation of myofascial trigger points evokes segmental antinociceptive effects. J Rehabil Med 2010;42:463–8.
37 Tsai CT, Hsieh LF, Kuan TS, Kao MJ, Chou LW, Hong CZ. Remote effects of dry needling on the irritability of the myofascial trigger point in the upper trapezius muscle. Am J Phys Med Rehabil 2010;89:133–40.
38 Baldry P. Superficial versus deep dry needling. Acupunct Med 2002;20:78–81.
39 Kuan TS, Hsieh YL, Chen SM, Chen JT, Yen WC, Hong CZ. The myofascial trigger point region: correlation between the degree of irritability and the prevalence of endplate noise. Am J Phys Med Rehabil 2007;86:183–9.
40 Simons DG, Travell JG, Simons LS. Travell and Simons' myofascial pain and dysfunction; the trigger point manual. 2nd ed. Baltimore, MD: Williams & Wilkins; 1999.
41 Travell JG, Simons DG. Myofascial pain and dysfunction: the trigger point manual. Baltimore, MD: Williams & Wilkins; 1992.
42 Gerwin RD, Shannon S, Hong CZ, Hubbard D, Gevirtz R. Interrater reliability in myofascial trigger point examination. Pain 1997;69:65–73.
43 Al-Shenqiti AM, Oldham JA. Test–retest reliability of myofascial trigger point detection in patients with rotator cuff tendonitis. Clin Rehabil 2005;19:482–7.
44 Bron C, Franssen J, Wensing M, Oostendorp RA. Interrater reliability of palpation of myofascial trigger points in three shoulder muscles. J Man Manipulative Ther 2007;15:203–15.
45 Hsieh CY, Hong CZ, Adams AH, Platt KJ, Danielson CD, Hoehler FK, *et al.* Interexaminer reliability of the palpation of trigger points in the trunk and lower limb muscles. Arch Phys Med Rehabil 2000;81:258–64.
46 Licht G, Müller-Ehrenberg H, Mathis J, Berg G, Greitemann G. Untersuchung myofaszialer Triggerpunkte ist zuverlässig. Manuelle Medizin 2007;45:402–8.
47 McEvoy J, Huijbregts PA. Reliability of myofascial trigger point palpation: a systematic review. In: Dommerholt J, Huijbregts PA, editors. Myofascial trigger points: pathophysiology and evidence-informed diagnosis and management. Boston, MA: Jones & Bartlett; 2011. P.65–88.
48 Myburgh C, Lauridsen HH, Larsen AH, Hartvigsen J. Standardized manual palpation of myofascial trigger points in relation to neck/shoulder pain; the influence of clinical experience on inter-examiner reproducibility. Man Ther 2011;16:136–40.
49 Sciotti VM, Mittak VL, DiMarco L, Ford LM, Plezbert J, Santipadri E, *et al.* Clinical precision of myofascial trigger point location in the trapezius muscle. Pain 2001;93:259–66.
50 Chen Q, Basford J, An KN. Ability of magnetic resonance elastography to assess taut bands. Clin Biomech 2008;23:623–9.
51 Chen Q, Bensamoun S, Basford JR, Thompson JM, An KN. Identification and quantification of myofascial taut bands with magnetic resonance elastography. Arch Phys Med Rehabil 2007;88:1658–61.
52 Sikdar S, Shah JP, Gilliams E, Gebreab T, Gerber LH. Assessment of myofascial trigger points (MTrPs): a new application of ultrasound imaging and vibration sonoelastography. Proceeding of the 30th Annual International IEEE EMBS Conference; 2008 Aug 20–24; Vancouver, BC, Canada. Piscataway, NJ: IEEE; 2008. p. 5585–8.

53 Hong CZ, Yu J. Spontaneous electrical activity of rabbit trigger spot after transection of spinal cord and peripheral nerve. J Musculoskelet Pain 1998;6:45–58.
54 Hubbard DR, Berkoff GM. Myofascial trigger points show spontaneous needle EMG activity. Spine 1993;18:1803–7.
55 Simons DG, Hong CZ, Simons LS. Endplate potentials are common to midfiber myofascial trigger points. Am J Phys Med Rehabil 2002;81:212–22.
56 Mense S. Muscle pain: mechanisms and clinical significance. Dtsch Arztebl Int 2008;105:214–9.
57 Mense S. How do muscle lesions such as latent and active trigger points influence central nociceptive neurons? J Musculokelet Pain 2010;18:348–53.
58 Vecchiet L, Giamberardino MA, Dragani L. Latent myofascial trigger points: changes in muscular and subcutaneous pain thresholds at trigger point and target level. J Manual Med 1990;5:151–4.
59 Vecchiet L, Pizzigallo E, Iezzi S, Affaitati G, Vecchiet J, Giamberardino MA. Differentiation of sensitivity in different tissues and its clinical significance. J Musculoskeletal Pain 1998;6:33–45.
60 Ge HY, Arendt-Nielsen L. Latent myofascial trigger points. Curr Pain Headache Rep 2011;to be published.
61 Ge HY, Serrao M, Andersen OK, Graven-Nielsen T, Arendt-Nielsen L. Increased H-reflex response induced by intramuscular electrical stimulation of latent myofascial trigger points. Acupunct Med 2009;27:150–4.
62 Ge HY, Zhang Y, Boudreau S, Yue SW, Arendt-Nielsen L. Induction of muscle cramps by nociceptive stimulation of latent myofascial trigger points. Exp Brain Res 2008;187:623–9.
63 Li LT, Ge HY, Yue SW, Arendt-Nielsen L. Nociceptive and non-nociceptive hypersensitivity at latent myofascial trigger points. Clin J Pain 2009;25:132–7.
64 Wang YH, Ding XL, Zhang Y, Chen J, Ge HY, Arendt-Nielsen L, *et al.* Ischemic compression block attenuates mechanical hyperalgesia evoked from latent myofascial trigger points. Exp Brain Res 2010;202:265–70.
65 Xu YM, Ge HY, Arendt-Nielsen L. Sustained nociceptive mechanical stimulation of latent myofascial trigger point induces central sensitization in healthy subjects. J Pain 2010;11:1348–55.
66 Zhang Y, Ge HY, Yue SW, Kimura Y, Arendt-Nielsen L. Attenuated skin blood flow response to nociceptive stimulation of latent myofascial trigger points. Arch Phys Med Rehabil 2009;90:325–32.
67 Gerwin RD. Myofascial pain syndrome: unresolved issues and future directions. In: Dommerholt J, Huijbregts PA, editors. Myofascial trigger points: pathophysiology and evidence-informed diagnosis and management. Boston, MA: Jones & Bartlett; 2011. p. 263–83.
68 Gerwin RD, Dommerholt J, Shah JP. An expansion of Simons' integrated hypothesis of trigger point formation. Current Pain Headache Reports 2004;8:468–75.
69 Simons DG. New views of myofascial trigger points: etiology and diagnosis. Arch Phys Med Rehabil 2008;89:157–9.
70 McPartland JM, Simons DG. Myofascial trigger points: translating molecular theory into manual therapy. J Man Manipulative Ther 2006;14:232–9.
71 Bukharaeva EA, Salakhutdinov RI, Vyskocil F, Nikolsky EE. Spontaneous quantal and non-quantal release of acetylcholine at mouse endplate during onset of hypoxia. Physiol Res 2005;54:251–5.
72 Chang CW, Chen YR, Chang KF. Evidence of neuroaxonal degeneration in myofascial pain syndrome: a study of neuromuscular jitter by axonal microstimulation. Eur J Pain 2008;12:1026–30.
73 Chen JT, Chen SM, Kuan TS, Chung KC, Hong CZ. Phentolamine effect on the spontaneous electrical activity of active loci in a myofascial trigger spot of rabbit skeletal muscle. Arch Phys Med Rehabil 1998;79:790–4.
74 Chen JT, Chung KC, Hou CR, Kuan TS, Chen SM, Hong CZ. Inhibitory effect of dry needling on the spontaneous electrical activity recorded from myofascial trigger spots of rabbit skeletal muscle. Am J Phys Med Rehabil 2001;80:729–35.
75 Couppé C, Midttun A, Hilden J, Jørgensen U, Oxholm P, Fuglsang-Frederiksen A. Spontaneous needle electromyographic activity in myofascial trigger points in the infraspinatus muscle: a blinded assessment. J Musculoskeletal Pain 2001;9:7–17.
76 Macgregor J, Graf von Schweinitz D. Needle electromyographic activity of myofascial trigger points and control sites in equine cleidobrachialis muscle — an observational study. Acupunct Med 2006;24:61–70.
77 Qerama E, Fuglsang-Frederiksen A, Kasch H, Bach FW, Jensen TS. Evoked pain in the motor endplate region of the brachial biceps muscle: an experimental study. Muscle Nerve 2004;29:393–400.
78 Simons DG. Do endplate noise and spikes arise from normal motor endplates? Am J Phys Med Rehabil 2001;80:134–40.
79 Simons DG. Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. J Electromyogr Kinesiol 2004;14:95–107.
80 Kuan TS, Chen JT, Chen SM, Chien CH, Hong CZ. Effect of botulinum toxin on endplate noise in myofascial trigger spots of rabbit skeletal muscle. Am J Phys Med Rehabil 2002;81:512–20.
81 Cheshire WP, Abashian SW, Mann JD. Botulinum toxin in the treatment of myofascial pain syndrome. Pain 1994;59:65–9.
82 de Andrés J, Cerda-Olmedo G, Valía JC, Monsalve V, Lopez-Alarcón MD, Minguez A. Use of botulinum toxin in the treatment of chronic myofascial pain. Clin J Pain 2003;19:269–75.
83 Gobel H, Heinze A, Reichel G, Hefter H, Benecke R. Efficacy and safety of a single botulinum type A toxin complex treatment (Dysport) for the relief of upper back myofascial pain syndrome: results from a randomized double-blind placebo-controlled multicentre study. Pain 2006;125:82–8.
84 Graboski CL, Shaun Gray D, Burnham RS. Botulinum toxin A versus bupivacaine trigger point injections for the treatment of myofascial pain syndrome: a randomised double blind crossover study. Pain 2005;118:170–5.
85 Kamanli A, Kaya A, Ardicoglu O, Ozgocmen S, Zengin FO, Bayik Y. Comparison of lidocaine injection, botulinum toxin injection, and dry needling to trigger points in myofascial pain syndrome. Rheumatol Int 2005;25:604–11.
86 Kern U, Martin C, Scheicher S, Muller H. Botulinum-Toxin-A in der Behandlung von Phantomschmerzen. Eine Pilotstudie. Schmerz 2003;17:117–24.
87 Reilich P, Fheodoroff K, Kern U, Mense S, Seddigh S, Wissel J, *et al.* Consensus statement: botulinum toxin in myofascial pain. J Neurol 2004;251:I36–8.
88 Bodine-Fowler S, Garfinkel A, Roy RR, Edgerton VR. Spatial distribution of muscle fibers within the territory of a motor unit. Muscle Nerve 1990;13:1133–45.
89 Edström L, Kugelberg E. Histochemical composition, distribution of fibres and fatiguability of single motor units. Anterior tibial muscle of the rat. J Neurol Neurosurg Psychiatry 1968;31:424–33.
90 Monti RJ, Roy RR, Edgerton VR. Role of motor unit structure in defining function. Muscle Nerve 2001;24:848–66.
91 Kuan LC, Li YT, Chen FM, Tseng CJ, Wu SF, Kuo TC. Efficacy of treating abdominal wall pain by local injection. Taiwan J Obstet Gynecol 2006;45:239–43.
92 Chang CW, Chang KY, Chen YR, Kuo PL. Electrophysiologic evidence of spinal accessory neuropathy in patients with cervical myofascial pain syndrome. Arch Phys Med Rehabil 2011;92:935–40.
93 Kuan TS, Lin TS, Chen JT, Chen SM, Hong CZ. No increased neuromuscular jitter at rabbit skeletal muscle trigger spot spontaneous electrical activity sites. J Musculoskeletal Pain 2000;8:69–82.
94 Hoyle JA, Marras WS, Sheedy JE, Hart DE. Effects of postural and visual stressors on myofascial trigger point development and motor unit rotation during computer work. J Electromyogr Kinesiol 2011;21:41–8.
95 Treaster D, Marras WS, Burr D, Sheedy JE, Hart D. Myofascial trigger point development from visual and postural stressors during computer work. J Electromyogr Kinesiol 2006;16:115–24.
96 Chaitow L. Breathing pattern disorders, motor control, and low back pain. J Osteop Med 2004;7:33–40.
97 Jammes Y, Zattara-Hartmann M, Badier M. Functional consequences of acute and chronic hypoxia on respiratory and skeletal muscles in mammals. Comp Biochem Physiol 1997;118:15–22.
98 FitzGerald MP, Anderson RU, Potts J, Payne CK, Peters KM, Clemens JQ, *et al.* Randomized multicenter feasibility trial of myofascial physical therapy for the treatment of

urological chronic pelvic pain syndromes. J Urol 2009;182:570–80.
99 Giamberardino MA, Affaitati G, Iezzi S, Vecchiet L. Referred muscle pain and hyperalgesia from viscera. J Musculoskeletal Pain 1999;7:61–9.
100 Jarrell JF, Vilos GA, Allaire C, Burgess S, Fortin C, Gerwin R, *et al.* Consensus guidelines for the management of chronic pelvic pain. J Obstet Gynaecol Can 2005;27:781–826.
101 Vecchiet L, Vecchiet J, Giamberardino MA. Referred muscle pain: clinical and pathophysiologic aspects. Curr Rev Pain 1999;3:489–98.
102 Falla D, Farina D. Neuromuscular adaptation in experimental and clinical neck pain. J Electromyogr Kinesiol 2008;18:255–61.
103 Falla D, Farina D. Neural and muscular factors associated with motor impairment in neck pain. Curr Rheumatol Rep 2007;9:497–502.
104 Henriksen M, Aaboe J, Simonsen EB, Alkjaer T, Bliddal H. Experimentally reduced hip abductor function during walking: implications for knee joint loads. J Biomech 2009;42:1236–40.
105 Henriksen M, Alkjaer T, Lund H, Simonsen EB, Graven-Nielsen T, Danneskiold-Samsoe B, *et al.* Experimental quadriceps muscle pain impairs knee joint control during walking. J Appl Physiol 2007;103:132–9.
106 Henriksen M, Alkjaer T, Simonsen EB, Bliddal H. Experimental muscle pain during a forward lunge — the effects on knee joint dynamics and electromyographic activity. Br J Sports Med 2009;43:503–7.
107 Bajaj P, Graven-Nielsen T, Arendt-Nielsen L. Osteoarthritis and its association with muscle hyperalgesia: an experimental controlled study. Pain 2001;93:107–14.
108 Bajaj P, Bajaj P, Graven-Nielsen T, Arendt-Nielsen L. Trigger points in patients with lower limb osteoarthritis. J Musculoskeletal Pain 2001;9:17–33.
109 Itoh K, Hirota S, Katsumi Y, Ochi H, Kitakoji H. Trigger point acupuncture for treatment of knee osteoarthritis–a preliminary RCT for a pragmatic trial. Acupunct Med 2008;26:17–26.
110 Hodges PW, Tucker K. Moving differently in pain: a new theory to explain the adaptation to pain. Pain 2011;152:S90–8.
111 Otten E. Concepts and models of functional architecture in skeletal muscle. Exerc Sport Sci Rev 1988;16:89–137.
112 Fernandez-Carnero J, Ge HY, Kimura Y, Fernandez-de-Las-Penas C, Arendt-Nielsen L. Increased spontaneous electrical activity at a latent myofascial trigger point after nociceptive stimulation of another latent trigger point. Clin J Pain 2010;26:138–43.
113 Carlson CR, Okeson JP, Falace DA, Nitz AJ, Lindroth JE. Reduction of pain and EMG activity in the masseter region by trapezius trigger point injection. Pain 1993;55:397–400.
114 Bretischwerdt C, Rivas-Cano L, Palomeque-del-Cerro L, Fernandez-de-las-Penas C, Alburquerque-Sendin F. Immediate effects of hamstring muscle stretching on pressure pain sensitivity and active mouth opening in healthy subjects. J Manipulative Physiol Ther 2010;33:42–7.
115 Srbely JZ, Dickey JP, Lowerison M, Edwards AM, Nolet PS, Wong LL. Stimulation of myofascial trigger points with ultrasound induces segmental antinociceptive effects: a randomized controlled study. Pain 2008;139:260–6.
116 Gerwin RD, Dommerholt J. Treatment of myofascial pain syndromes. In: Boswell MV, Cole BE, editors. Weiner's pain management; a practical guide for clinicians. Boca Raton, FL: CRC Press; 2006. p. 477–92.
117 Majlesi J, Unalan H. High-power pain threshold ultrasound technique in the treatment of active myofascial trigger points: a randomized, double-blind, case-control study. Arch Phys Med Rehabil 2004;85:833–6.
118 Fernández de las Peñas C, Cuadrado ML, Pareja JA. Myofascial trigger points, neck mobility, and forward head posture in episodic tension-type headache. Headache 2007;47:662–72.
119 Grieve R, Clark J, Pearson E, Bullock S, Boyer C, Jarrett A. The immediate effect of soleus trigger point pressure release on restricted ankle joint dorsiflexion: a pilot randomised controlled trial. J Bodyw Mov Ther 2011;15:42–9.
120 Chaitow L, DeLany J. Neuromuscular techniques in orthopedics. Tech Orthop 2003;18:74–86.
121 Anderson RU, Wise D, Sawyer T, Chan CA. Sexual dysfunction in men with chronic prostatitis/chronic pelvic pain syndrome: improvement after trigger point release and paradoxical relaxation training. J Urol 2006;176:1534–8.
122 Anderson RU, Wise D, Sawyer T, Glowe P, Orenberg EK. 6-day intensive treatment protocol for refractory chronic prostatitis/chronic pelvic pain syndrome using myofascial release and paradoxical relaxation training. J Urol 2011;185:1294–9.
123 Bogduk N. Evidence-informed management of chronic low back pain with facet injections and radiofrequency neurotomy. Spine J 2008;8:56–64.
124 Cooper G, Bailey B, Bogduk N. Cervical zygapophysial joint pain maps. Pain Med 2007;8:344–53.
125 Frost HM. The frozen shoulder syndrome plus other evidence and the Utah Paradigm suggest the syndrome's pathogenesis and new targets for collagenous tissue research. J Musculoskelet Neuronal Interact 2000;1:113–9.
126 Schofferman J, Bogduk N, Slosar P. Chronic whiplash and whiplash-associated disorders: an evidence-based approach. J Am Acad Orthop Surg 2007;15:596–606.
127 Balla J, Karnaghan J. Whiplash headache. Clin Exp Neurol 1987;23:179–82.
128 Bener A, Rahman YS, Mitra B. Incidence and severity of head and neck injuries in victims of road traffic crashes: in an economically developed country. Int Emerg Nurs 2009;17:52–9.
129 Quinlan KP, Annest JL, Myers B, Ryan G, Hill H. Neck strains and sprains among motor vehicle occupants — United States, 2000. Accid Anal Prev 2004;36:21–7.
130 Nadler SF. Myofascial pain after whiplash injury. In: Malanga GA, Nadler SF, editors. Whiplash. Philadephia, PA: Hanley & Belfus; 2002. p. 219–39.
131 Dommerholt J, Bron C, Franssen JL. Myofascial trigger points; an evidence-informed review. J Manual Manipulative Ther 2006;14:203–21.
132 International Association for the Study of Pain. Classification of chronic pain: descriptions of chronic pain syndromes and definitions of pain terms. Pain 1986;3:S1–225.
133 Fields HL, Basbaum AI. Central nervous system mechanisms of pain modulation. In: Melzack R, Wall PD, editors. Textbook of pain. 4th ed. Edinburgh: Churchill Livingstone; 1999. p. 309–29.
134 Fleckenstein J, Zaps D, Rüger LJ, Lehmeyer L, Freiberg F, Lang PM, *et al.* Discrepancy between prevalence and perceived effectiveness of treatment methods in myofascial pain syndrome: results of a cross-sectional, nationwide survey. BMC Musculoskeletal Dis 2010;11:32.
135 Harden RN, Bruehl SP, Gass S, Niemiec C, Barbick B. Signs and symptoms of the myofascial pain syndrome: a national survey of pain management providers. Clin J Pain 2000;16:64–72.
136 Fernandez-Lao C, Cantarero-Villanueva I, Fernandez-de-Las-Penas C, Del-Moral-Avila R, Arendt-Nielsen L, Arroyo-Morales M. Myofascial trigger points in neck and shoulder muscles and widespread pressure pain hypersensitivtiy in patients with postmastectomy pain: evidence of peripheral and central sensitization. Clin J Pain 2010;26:798–806.
137 Ge HY, Nie H, Madeleine P, Danneskiold-Samsoe B, Graven-Nielsen T, Arendt-Nielsen L. Contribution of the local and referred pain from active myofascial trigger points in fibromyalgia syndrome. Pain 2009;147:233–40.
138 Ge HY, Wang Y, Fernandez-de-Las-Penas C, Graven-Nielsen T, Danneskiold-Samsoe B, Arendt-Nielsen L. Reproduction of overall spontaneous pain pattern by manual stimulation of active myofascial trigger points in fibromyalgia patients. Arthritis Res Ther 2011;13:R48.
139 Fernández de las Peñas C, Cuadrado M, Arendt-Nielsen L, Simons D, Pareja J. Myofascial trigger points and sensitization: an updated pain model for tension-type headache. Cephalalgia 2007;27:383–93.
140 Wall PD, Woolf CJ. Muscle but not cutaneous C-afferent input produces prolonged increases in the excitability of the flexion reflex in the rat. J Physiol 1984;356:443–58.
141 Sessle BJ, Hu JW, Cairns BE. Brainstem mechanisms underlying temporomandibular joint and masticatory muscle pain. J Musculoskeletal Pain 1999;7:161–9.
142 Sessle BJ, Hu JW, Amano N, Zhong G. Convergence of cutaneous, tooth pulp, visceral, neck and muscle afferents onto nociceptive and non-nociceptive neurones in trigeminal subnucleus caudalis (medullary dorsal horn) and its implication for referred pain. Pain 1986;27:219–35.
143 Brückle W, Sückfull M, Fleckenstein W, Weiss C, Müller W. Gewebe-pO_2-Messung in der verspannten Rückenmuskulatur (m. erector spinae). Z Rheumatol 1990;49:208–16.

144 Sikdar S, Ortiz R, Gebreab T, Gerber LH, Shah JP. Understanding the vascular environment of myofascial trigger points using ultrasonic imaging and computational modeling. Conf Proc IEEE Eng Med Biol Soc 2010;1:5302–5.
145 Chen BM, Grinnell AD. Kinetics, Ca^{2+} dependence, and biophysical properties of integrin-mediated mechanical modulation of transmitter release from frog motor nerve terminals. J Neurosci 1997;17:904–16.
146 Grinnell AD, Chen BM, Kashani A, Lin J, Suzuki K, Kidokoro Y. The role of integrins in the modulation of neurotransmitter release from motor nerve terminals by stretch and hypertonicity. J Neurocytol 2003;32:489–503.
147 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;99:1977–84.
148 Sahlin K, Harris RC, Nylind B, Hultman E. Lactate content and pH in muscle obtained after dynamic exercise. Pflugers Arch: Eur J Physiol 1976;367:143–9.
149 Gautam M, Benson CJ, Sluka KA. Increased response of muscle sensory neurons to decreases in pH after muscle inflammation. Neuroscience 2010;170:893–900.
150 Sluka KA, Kalra A, Moore SA. Unilateral intramuscular injections of acidic saline produce a bilateral, long-lasting hyperalgesia. Muscle Nerve 2001;24:37–46.
151 Sluka KA, Price MP, Breese NM, Stucky CL, Wemmie JA, Welsh MJ. Chronic hyperalgesia induced by repeated acid injections in muscle is abolished by the loss of ASIC3, but not ASIC1. Pain 2003;106:229–39.
152 Sluka KA, Rohlwing JJ, Bussey RA, Eikenberry SA, Wilken JM. Chronic muscle pain induced by repeated acid Injection is reversed by spinally administered mu- and delta-, but not kappa-, opioid receptor agonists. J Pharmacol Exp Ther 2002;302:1146–50.
153 Deval E, Gasull X, Noel J, Salinas M, Baron A, Diochot S, *et al.* Acid-sensing ion channels (ASICs): pharmacology and implication in pain. Pharmacol Ther 2010;128:549–58.
154 Walder RY, Rasmussen LA, Rainier JD, Light AR, Wemmie JA, Sluka KA. ASIC1 and ASIC3 play different roles in the development of Hyperalgesia after inflammatory muscle injury. J Pain 2010;11:210–8.
155 da Silva LF, Desantana JM, Sluka KA. Activation of NMDA receptors in the brainstem, rostral ventromedial medulla, and nucleus reticularis gigantocellularis mediates mechanical hyperalgesia produced by repeated intramuscular injections of acidic saline in rats. J Pain 2010;11:378–87.
156 Reinohl J, Hoheisel U, Unger T, Mense S. Adenosine triphosphate as a stimulant for nociceptive and non-nociceptive muscle group IV receptors in the rat. Neurosci Lett 2003;338:25–8.
157 Birdsong WT, Fierro L, Williams FG, Spelta V, Naves LA, Knowles M, *et al.* Sensing muscle ischemia: coincident detection of acid and ATP via interplay of two ion channels. Neuron 2010;68:739–49.
158 Jensen K, Tuxen C, Pedersen-Bjergaard U, Jansen I, Edvinsson L, Olesen J. Pain and tenderness in human temporal muscle induced by bradykinin and 5-hydroxytryptamine. Peptides 1990;11:1127–32.
159 Babenko V, Graven-Nielsen T, Svensson P, Drewes AM, Jensen TS, Arendt-Nielsen L. Experimental human muscle pain induced by intramuscular injections of bradykinin, serotonin, and substance P. Eur J Pain 1999;3:93–102.
160 Marchettini P, Simone DA, Caputi G, Ochoa JL. Pain from excitation of identified muscle nociceptors in humans. Brain Res 1996;740:109–16.
161 Simone DA, Marchettini P, Caputi G, Ochoa JL. Identification of muscle afferents subserving sensation of deep pain in humans. J Neurophysiol 1994;72:883–9.
162 Curatolo M, Arendt-Nielsen L, Petersen-Felix S. Central hypersensitivity in chronic pain: mechanisms and clinical implications. Phys Med Rehabil Clin N Am 2006;17:287–302.
163 Bajaj P, Madsen H, Arendt-Nielsen L. Endometriosis is associated with central sensitization: a psychophysical controlled study. J Pain 2003;4:372–80.
164 O'Neill S, Manniche C, Graven-Nielsen T, Arendt-Nielsen L. Generalized deep-tissue hyperalgesia in patients with chronic low-back pain. Eur J Pain 2007;11:415–20.
165 Rossel P, Drewes AM, Petersen P, Nielsen J, Arendt-Nielsen L. Pain produced by electric stimulation of the rectum in patients with irritable bowel syndrome: further evidence of visceral hyperalgesia. Scand J Gastroenterol 1999;34:1001–6.
166 Wilder-Smith OH, Tassonyi E, Senly C, Otten P, Arendt-Nielsen L. Surgical pain is followed not only by spinal sensitization but also by supraspinal antinociception. Br J Anaesth 1996;76:816–21.
167 Banic B, Petersen-Felix S, Andersen OK, Radanov BP, Villiger PM, Arendt-Nielsen L, *et al.* Evidence for spinal cord hypersensitivity in chronic pain after whiplash injury and in fibromyalgia. Pain 2004;107:7–15.
168 Curatolo M, Arendt-Nielsen L, Petersen-Felix S. Evidence, mechanisms, and clinical implications of central hypersensitivity in chronic pain after whiplash injury. Clin J Pain 2004;20:469–76.
169 Gwilym SE, Oag HC, Tracey I, Carr AJ. Evidence that central sensitisation is present in patients with shoulder impingement syndrome and influences the outcome after surgery. J Bone Joint Surgery (Br) 2011;93:498–502.
170 Clauw DJ. Fibromyalgia: a label for chronic widespread pain. Medscape 2008. Available from: http://www.Medscape.com. [accessed September 29, 2011].
171 Yunus MB. Central sensitivity syndromes: a new paradigm and group nosology for fibromyalgia and overlapping conditions, and the related issue of disease versus illness. Semin Arthritis Rheum 2008;37:339–52.
172 Freeman MD, Nystrom A, Centeno C. Chronic whiplash and central sensitization; an evaluation of the role of a myofascial trigger points in pain modulation. J Brachial Plex Peripher Nerve Inj 2009;4:2.
173 Fernández de las Peñas C, Ge HY, Arendt-Nielsen L, Cuadrado ML, Pareja JA. Referred pain from trapezius muscle trigger points shares similar characteristics with chronic tension type headache. Eur J Pain 2007;11:475–82.
174 Fernández de las Peñas C, Ge HY, Arendt-Nielsen L, Cuadrado ML, Pareja JA. The local and referred pain from myofascial trigger points in the temporalis muscle contributes to pain profile in chronic tension-type headache. Clin J Pain 2007;23:786–92.
175 Fernández-de-las-Peñas C, Caminero AB, Madeleine P, Guillem-Mesado A, Ge HY, Arendt-Nielsen L, *et al.* Multiple active myofascial trigger points and pressure pain sensitivity maps in the temporalis muscle are related in women with chronic tension type headache. Clin J Pain 2009;25:506–12.
176 Fernández de las Peñas C, Cuadrado ML, Barriga FJ, Pareja JA. Active muscle trigger points as sign of sensitization in chronic primary headaches. J Musculoskeletal Pain 2009; 17:155–61.
177 Calandre EP, Hidalgo J, Garcia-Leiva JM, Rico-Villademoros F. Trigger point evaluation in migraine patients: an indication of peripheral sensitization linked to migraine predisposition? Eur J Neurol 2006;13:244–9.
178 Giamberardino MA, Tafuri E, Savini A, Fabrizio A, Affaitati G, Lerza R, *et al.* Contribution of myofascial trigger points to migraine symptoms. J Pain 2007;8:869–78.
179 Fernández-Carnero J, Fernández-de-las-Peñas C, de la Llave-Rincón AI, Ge HY, Arendt-Nielsen L. Bilateral myofascial trigger points in the forearm muscles in patients with chronic unilateral lateral epicondylalgia: a blinded, controlled study. Clin J Pain 2008;24:802–7.
180 Fernández-Carnero J, Fernández de las Peñas CF, de la Llave-Rincón AI, Ge HY, Arendt-Nielsen L. Prevalence of and referred pain from myofascial trigger points in the forearm muscles in patients with lateral epicondylalgia. Clin J Pain 2007;23:353–60.
181 Fernandez-Lao C, Cantarero-Villanueva I, Fernandez-de-las-Penas C, Del-Moral-Avila R, Menjon-Beltran S, Arroyo-Morales M. Widespread mechanical pain hypersensitivity as a sign of central sensitization after breast cancer surgery: comparison between mastectomy and lumpectomy. Pain Med 2011;12:72–8.
182 Fernandez-Lao C, Cantarero-Villanueva I, Fernandez-de-las-Penas C, Del-Moral-Avila R, Menjon-Beltran S, Arroyo-Morales M. Development of active myofascial trigger points in neck and shoulder musculature is similar after lumpectomy or mastectomy surgery for breast cancer. J Bodyw Mov Ther 2011;to be published.
183 Torres Lacomba M, Mayoral del Moral O, Coperias Zazo JL, Gerwin RD, Goni AZ. Incidence of myofascial pain syndrome in breast cancer surgery: a prospective study. Clin J Pain 2010;26:320–5.
184 Torres-Lacomba M, Mayoral del Moral O. Les thromboses lymphatiques superficielles a l'origine du syndrome douloureux

myofascial apres curage axillaire pour cancer du sein. Kinesitherapie Scientifique 2008;494:25–8.
185 Ge HY. Prevalence of myofascial trigger points in fibromyalgia: the overlap of two common problems. Curr Pain Headache Rep 2010;14:339–45.
186 Fernández de las Peñas C, Galán del Rio F, Fernández Carnero J, Pesquera J, Arendt-Nielsen L, Svensson P. Bilateral widespread mechanical pain sensitivity in women with myofascial temporomandibular disorder: evidence of impairment in central nociceptive processing. J Pain 2009;10:1170–8.
187 Nijs J, van Houdenhove B, Oostendorp RA. Recognition of central sensitization in patients with musculoskeletal pain: application of pain neurophysiology in manual therapy practice. Man Ther 2010;15:135–41.
188 Lim EC, Sterling M, Stone A, Vicenzino B. Central hyperexcitability as measured with nociceptive flexor reflex threshold in chronic musculoskeletal pain: a systematic review. Pain 2011;152:1811–20.
189 Escobar PL, Ballesteros J. Teres minor. Source of symptoms resembling ulnar neuropathy or C8 radiculopathy. Am J Phys Med Rehabil 1988;67:120–2.
190 Rocha CA, Sanchez TG. Myofascial trigger points: another way of modulating tinnitus. Prog Brain Res 2007;166:209–14.
191 Hong CZ, Kuan TS, Chen JT, Chen SM. Referred pain elicited by palpation and by needling of myofascial trigger points: a comparison. Arch Phys Med Rehabil 1997;78:957–60.
192 Cummings TM, White AR. Needling therapies in the management of myofascial trigger point pain: a systematic review. Arch Phys Med Rehabil 2001;82:986–92.
193 Bron C. Het subacromiaal-impingementsyndroom. Tijdschr Man Ther 2006;3:20–6.
194 Bron C, de Gast A, Dommerholt J, Stegenga B, Wensing M, Oostendorp RA. Treatment of myofascial trigger points in patients with chronic shoulder pain; a randomized controlled trial. BMC Med 2011;9:8.
195 Hidalgo-Lozano A, Fernández-de-las-Peñas C, Alonso-Blanco C, Ge HY, Arendt-Nielsen L, Arroyo-Morales M. Muscle trigger points and pressure pain hyperalgesia in the shoulder muscles in patients with unilateral shoulder impingement: a blinded, controlled study. Exp Brain Res 2010;202:915–25.
196 Perez-Palomares S, Olivan-Blazquez B, Arnal-Burro AM, Mayoral del Moral O, Gaspar-Calvo E, de-la-Torre-Beldarrain ML, *et al.* Contributions of myofascial pain in diagnosis and treatment of shoulder pain. A randomized control trial. BMC Musculoskeletal Dis 2009;10:92.
197 Torres Lacomba M, Mayoral del Moral O, Coperias Zazo JL, Yuste Sanchez MJ, Ferrandez JC, Zapico Goni A. Axillary web syndrome after axillary dissection in breast cancer: a prospective study. Breast Cancer Res Treat 2009;17:625–30
198 Niddam DM, Chan RC, Lee SH, Yeh TC, Hsieh JC. Central representation of hyperalgesia from myofascial trigger point. Neuroimage 2008;39:1299–306.
199 Niddam DM, Chan RC, Lee SH, Yeh TC, Hsieh JC. Central modulation of pain evoked from myofascial trigger point. Clin J Pain 2007;23:440–8.
200 Travell J, Rinzler SH. The myofascial genesis of pain. Postgrad Med 1952;11:452–34.
201 Arendt-Nielsen L, Graven-Nielsen T. Deep tissue hyperalgesia. J Musculoskeletal Pain 2002;10:97–119.
202 Arendt-Nielsen L, Svensson P. Referred muscle pain: basic and clinical findings. Clin J Pain 2001;17:11–9.
203 Graven-Nielsen T, Arendt-Nielsen L. Induction and assessment of muscle pain, referred pain, and muscular hyperalgesia. Curr Pain Headache Rep 2003;7:443–51.
204 Hoheisel U, Koch K, Mense S. Functional reorganization in the rat dorsal horn during an experimental myositis. Pain 1994;59:111–8.
205 Hoheisel U, Mense S, Simons D, Yu XM. Appearance of new receptive fields in rat dorsal horn neurons following noxious stimulation of skeletal muscle: a model for referral of muscle pain? Neurosci Lett 1993;153:9–12.
206 Mense S. Referral of muscle pain: new aspects. Amer Pain Soc J 1994;3:1–9.
207 Mense S. Algesic agents exciting muscle nociceptors. Exp Brain Res 2009;196:89–100.
208 Shah JP, Gilliams EA. Uncovering the biochemical milieu of myofascial trigger points using *in vivo* microdialysis: an application of muscle pain concepts to myofascial pain syndrome. J Bodyw Mov Ther 2008;12:371–84.
209 Chacur M, Lambertz D, Hoheisel U, Mense S. Role of spinal microglia in myositis-induced central sensitisation: an immunohistochemical and behavioural study in rats. Eur Pain 2009;13:915–23.
210 Zhang J, Hoffert C, Vu HK, Groblewski T, Ahmad S, O'Donnell D. Induction of CB2 receptor expression in the rat spinal cord of neuropathic but not inflammatory chronic pain models. Eur J Neurosci 2003;17:2750–4.
211 Clark AK, Gentry C, Bradbury EJ, McMahon SB, Malcangio M. Role of spinal microglia in rat models of peripheral nerve injury and inflammation. Eur J Pain 2007;11:223–30.
212 Thacker MA, Clark AK, Bishop T, Grist J, Yip PK, Moon LD, *et al.* CCL2 is a key mediator of microglia activation in neuropathic pain states. Eur J Pain 2009;13:263–72.
213 Gosselin RD, Suter MR, Ji RR, Decosterd I. Glial cells and chronic pain. Neuroscientist 2010;16:519–31.
214 Cairns BE, Gambarota G, Svensson P, Arendt-Nielsen L, Berde CB. Glutamate-induced sensitization of rat masseter muscle fibers. Neuroscience 2002;109:389–99.
215 Castrillon EE, Cairns BE, Ernberg M, Wang K, Sessle B, Arendt-Nielsen L, *et al.* Glutamate-evoked jaw muscle pain as a model of persistent myofascial TMD pain? Arch Oral Biol 2008;53:666–76.
216 Castrillon EE, Ernberg M, Cairns BE, Wang K, Sessle BJ, Arendt-Nielsen L, *et al.* Interstitial glutamate concentration is elevated in the masseter muscle of myofascial temporomandibular disorder patients. J Orofacial Pain 2010;24:350–60.
217 Dong XD, Mann MK, Sessle BJ, Arendt-Nielsen L, Svensson P, Cairns BE. Sensitivity of rat temporalis muscle afferent fibers to peripheral N-methyl-D-aspartate receptor activation. Neuroscience 2006;141:939–45.
218 Gerdle B, Lemming D, Kristiansen J, Larsson B, Peolsson M, Rosendal L. Biochemical alterations in the trapezius muscle of patients with chronic whiplash associated disorders (WAD) — a microdialysis study. Eur J Pain 2008;12:82–93.
219 Sarchielli P, Di Filippo M, Nardi K, Calabresi P. Sensitization, glutamate, and the link between migraine and fibromyalgia. Curr Pain Headache Rep 2007;11:343–51.
220 Miller KE, Hoffman EM, Sutharshan M, Schechter R. Glutamate pharmacology and metabolism in peripheral primary afferents: physiological and pathophysiological mechanisms. Pharmacol Ther 2011;130:283–309.
221 Müller W, Stratz T. Local treatment of tendinopathies and myofascial pain syndromes with the 5-HT3 receptor antagonist tropisetron. Scand J Rheumatol Suppl 2004;119:44–8.
222 Gerber RK, Nie H, Arendt-Nielsen L, Curatolo M, Graven-Nielsen T. Local pain and spreading hyperalgesia induced by intramuscular injection of nerve growth factor are not reduced by local anesthesia of the muscle. Clin J Pain 2011;27:240–7.
223 Shinoda M, Asano M, Omagari D, Honda K, Hitomi S, Katagiri A, *et al.* Nerve growth factor contribution via transient receptor potential vanilloid 1 to ectopic orofacial pain. J Neurosci 2011;31:7145–55.
224 Cuppini R, Sartini S, Agostini D, Guescini M, Ambrogini P, Betti M, *et al.* BDNF expression in rat skeletal muscle after acute or repeated exercise. Arch Ital Biol 2007;145:99–110.
225 Matthews VB, Astrom MB, Chan MH, Bruce CR, Krabbe KS, Prelovsek O, *et al.* Brain-derived neurotrophic factor is produced by skeletal muscle cells in response to contraction and enhances fat oxidation via activation of AMP-activated protein kinase. Diabetologia 2009;52:1409–18.
226 Hoheisel U, Unger T, Mense S. The possible role of the NO–cGMP pathway in nociception: different spinal and supraspinal action of enzyme blockers on rat dorsal horn neurones. Pain 2005;117:358–67.
227 Tidball JG. Inflammatory processes in muscle injury and repair. Am J Physiol Regul Integr Comp Physiol 2005; 288:R345–53.
228 Tough EA, White AR, Cummings TM, Richards SH, Campbell JL. Acupuncture and dry needling in the management of myofascial trigger point pain: a systematic review and meta-analysis of randomised controlled trials. Eur J Pain 2009;13:3–10.
229 Furlan A, Tulder M, Cherkin D, Tsukayama H, Lao L, Koes B, *et al.* Acupuncture and dry-needling for low back pain: an updated systematic review within the framework of the Cochrane collaboration. Spine 2005;30:944–63.
230 Chou LW, Hsieh YL, Kao MJ, Hong CZ. Remote influences of acupuncture on the pain intensity and the amplitude changes of endplate noise in the myofascial trigger point of the

upper trapezius muscle. Arch Phys Med Rehabil 2009;90:905–12.
231 Dilorenzo L, Traballesi M, Morelli D, Pompa A, Brunelli S, Buzzi MG, *et al.* Hemiparetic shoulder pain syndrome treated with deep dry needling during early rehabilitation: a prospective, open-label, randomized investigation. J Musculoskeletal Pain 2004;12:25–34.
232 Ga H, Choi JH, Park CH, Yoon HJ. Acupuncture needling versus lidocaine injection of trigger points in myofascial pain syndrome in elderly patients — a randomised trial. Acupunct Med 2007;25:130–6.
233 Lundeberg T, Uvnas-Moberg K, Agren G, Bruzelius G. Antinociceptive effects of oxytocin in rats and mice. Neurosci Lett 1994;170:153–7.
234 Uvnas-Moberg K, Bruzelius G, Alster P, Lundeberg T. The antinociceptive effect of non-noxious sensory stimulation is mediated partly through oxytocinergic mechanisms. Acta Physiol Scand 1993;149:199–204.
235 Dincer F, Linde K. Sham interventions in randomized clinical trials of acupuncture–a review. Complement Ther Med 2003;11:235–42.
236 White P, Lewith G, Hopwood V, Prescott P. The placebo needle, is it a valid and convincing placebo for use in acupuncture trials? A randomised, single-blind, cross-over pilot trial. Pain 2003;106:401–9.
237 Birch S. A review and analysis of placebo treatments, placebo effects, and placebo controls in trials of medical procedures when sham is not inert. J Altern Complement Med 2006;12:303–10.
238 Lund I, Lundeberg T. Are minimal, superficial or sham acupuncture procedures acceptable as inert placebo controls? Acupunct Med 2006;24:13–5.
239 Lund I, Naslund J, Lundeberg T. Minimal acupuncture is not a valid placebo control in randomised controlled trials of acupuncture: a physiologist's perspective. Chin Med 2009;4:1.
240 Pariente J, White P, Frackowiak RS, Lewith G. Expectancy and belief modulate the neuronal substrates of pain treated by acupuncture. Neuroimage 2005;25:1161–7.
241 Wang SM, Kain ZN, White PF. Acupuncture analgesia: II. Clinical considerations. Anesth Analg 2008;106:611–21.
242 White A, Cummings M. Does acupuncture relieve pain? BMJ 2009;338:a2760.
243 Napadow V, Dhond RP, Kim J, LaCount L, Vangel M, Harris RE, *et al.* Brain encoding of acupuncture sensation — coupling on-line rating with fMRI. Neuroimage 2009;47:1055–65.
244 Ernst E. Acupuncture–a critical analysis. J Intern Med 2006;259:125–37.
245 Kleinhenz J, Streitberger K, Windeler J, Gussbacher A, Mavridis G, Martin E. Randomised clinical trial comparing the effects of acupuncture and a newly designed placebo needle in rotator cuff tendinitis. Pain 1999;83:235–41.
246 McManus CA, Schnyer RN, Kong J, Nguyen LT, Hyun Nam B, Goldman R, *et al.* Sham acupuncture devices — practical advice for researchers. Acupunct Med 2007;25:36–40.
247 Streitberger K, Kleinhenz J. Introducing a placebo needle into acupuncture research. Lancet 1998;352:364–5.
248 Faria V, Fredrikson M, Furmark T. Imaging the placebo response: a neurofunctional review. Eur Neuropsychopharmacol 2008;18:473–85.
249 Bausell RB, Lao L, Bergman S, Lee WL, Berman BM. Is acupuncture analgesia an expectancy effect? Preliminary evidence based on participants' perceived assignments in two placebo-controlled trials. Eval Health Prof 2005;28:9–26.
250 Kong J, Kaptchuk TJ, Polich G, Kirsch I, Vangel M, Zyloney C, *et al.* An fMRI study on the interaction and dissociation between expectation of pain relief and acupuncture treatment. Neuroimage 2009;47:1066–76.
251 Wager TD, Rilling JK, Smith EE, Sokolik A, Casey KL, Davidson RJ, *et al.* Placebo-induced changes in FMRI in the anticipation and experience of pain. Science 2004;303:1162–7.
252 Morton DL, Watson A, El-Deredy W, Jones AK. Reproducibility of placebo analgesia: Effect of dispositional optimism. Pain 2009;146:194–8.
253 Mayoral del Moral O. Dry needling treatments for myofascial trigger points. J Musculoskeletal Pain 2010;18:411–6.
254 Seem M. A new American acupuncture; acupuncture osteopathy. Boulder, CO: Blue Poppy Press; 2007.
255 Association of Social Work Boards, Federation of State Boards of Physical Therapy, Federation of State Medical Boards of the United States Inc., National Association of Boards of Pharmacy, National Board for Certification in Occupational Therapy Inc., The National Council of State Boards of Nursing Inc. Changes in healthcare professions scope of practice: legislative considerations. Chicago, IL: National Council of State Boards of Nursing; 2009.
256 Dommerholt J. Dry Needling und Akupunkturtechniken. In: Reilich P, Gröbli C, Dommerholt J, editors. Myofasziale Schmerzen und Triggerpunkte Die klinische Essenz. Munich: Urban & Fischer; 2011. P.58–75.
257 Gansler DF, McDonald RN. Opinions of the attorney general. Baltimore, MD: Office of the Attorney General; 2010.
258 Hobbs V. Council of colleges of acupuncture and oriental medicine position paper on dry needling. Baltimore, MD: Council of Colleges of Acupuncture and Oriental Medicine; 2011.
259 Hobbs V. Dry needling and acupuncture emerging professional issues. Qi Unity Report; September/October 2007.
260 Ward-Cook K, Hahn T. NCCAOM® 2008 job task analysis: a report to the acupuncture and Oriental medicine (AOM) community. Jacksonville, FL: National Certification Commission of Acupuncture and Oriental Medicine; 2010.
261 Fabrey LJ, Cogdill KS, Kelley JA. A national job analysis: acupuncture and oriental medicine profession. Jacksonville, FL: National Certification Commission for Acupuncture and Oriental Medicine; 2003.
262 Hogeboom CJ, Sherman KJ, Cherkin DC. Variation in diagnosis and treatment of chronic low back pain by traditional Chinese medicine acupuncturists. Complement Ther Med 2001;9:154–66.
263 Arizona Revised Statutes, Stat. 32-3901.1 — Definitions; 2011.
264 Arizona Revised Statutes, Stat. 32-3921.B1 — Licensure; acts and persons not affected; 2011.
265 Cardinal S. Points détente et acupuncture: approche neurophysiologique. Montreal, Que: Centre collégial de développement de matériel didactique; 2004.
266 Cardinal S. Points-détente et acupuncture: techniques de puncture. Montréal, Que.: Centre collégial de développement de matériel didactique; 2007.
267 Amaro JA. When acupuncture becomes 'dry needling'. Acupunct Today 2007;33:43.
268 Birch S. Trigger point: acupuncture point correlations revisited. J Altern Complement Med 2003;9:91–103.
269 Birch S. On the impossibility of trigger point-acupoint equivalence: a commentary on Peter Dorsher's analysis. J Altern Complement Med 2008;14:343–5.
270 Audette JF, Blinder RA. Acupuncture in the management of myofascial pain and headache. Curr Pain Headache Rep 2003;7:395–401.
271 Hong CZ. Myofascial trigger points: pathophysiology and correlation with acupuncture points. Acupunct Med 2000;18:41–7.
272 Dorsher P. Trigger points and acupuncture points: anatomic and clinical correlations. Med Acupunct 2006;17:21–5.
273 Dorsher PT, Fleckenstein J. Trigger points and classical acupuncture points. Part 1: Qualitative and quantitative anatomic correspondences. Dt Ztschr f Akup 2008;51:15–24.
274 Dorsher PT, Fleckenstein J. Trigger points and classical acupuncture points. Part 2: Clinical correspondences in treating pain and somatovisceral disorders. Dt Ztschr f Akup 2008;51:6–11.
275 Dorsher PT, Fleckenstein J. Trigger points and classical acupuncture points. Part 3: Relationships of myofascial referred pain patterns to acupuncture meridians. Dt Ztschr f Akup 2009;52:10–4.
276 American Association of Acupuncture and Oriental Medicine (AAAOM) Position Statement on Trigger Point Dry Needling (TDN) and Intramuscular Manual Therapy (IMT); 2011.
277 White A. Western medical acupuncture: a definition. Acupunct Med 2009;27:33–5.
278 Pérez-Palomares S, Oliván-Blázquez B, Magallón-Botaya R, de-la-Torre-Beldarraín M, Gaspar-Calvo E, Romo-Calvo L, *et al.* Percutaneous electrical nerve stimulation versus dry needling: effectiveness in the treatment of chronic low back pain. J Musculoskeletal Pain 2010;18:23–30.
279 Fernandez-Carnero J, Fernández-de-las-Peñas C, Cleland JA. Mulligan's mobilization with movement and muscle trigger point dry needling for the management of chronic lateral epicondylalgia: a case report. J Musculoskeletal Pain 2009;17:409–15.
280 Edwards J. The importance of postural habits in perpetuating myofascial trigger point pain. Acupunct Med 2005;23:77–82.

281 Hammer WI. The effect of mechanical load on degenerated soft tissue. J Bodyw Mov Ther 2008;12:246–56.
282 Looney B, Srokose T, Fernandez-de-las-Penas C, Cleland JA. Graston instrument soft tissue mobilization and home stretching for the management of plantar heel pain: a case series. J Manipulative Physiol Ther 2011;34:138–42.
283 Ruiz-Saez M, Fernandez-de-las-Penas C, Blanco CR, Martinez-Segura R, Garcia-Leon R. Changes in pressure pain sensitivity in latent myofascial trigger points in the upper trapezius muscle after a cervical spine manipulation in pain-free subjects. J Manipulative Physiol Ther 2007;30:578–83.