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]

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