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Myofascial trigger points: the current evidence

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Abstract

This paper provides an overview of the current state of knowledge regarding the history, pathophysiology, mechanisms of pain production, and proposed methods of treatment of myofascial trigger points. Despite the increasing body of published literature on this subject, many fundamental questions remain unanswered. This paper aims to give the therapist a greater understanding of the current knowledge of mechanisms of muscle pain, treatments that have been shown to be effective, and the ways in which these treatments may produce their effect. Most effective treatments have at their core a form of counter-stimulation or application of a second noxious stimulus. It remains unclear if it is this counter-stimulation or more specific elements of muscle stimulation that are the active ingredients, but it is possible that each contributes to effective outcomes.
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1. Introduction

Although myofascial trigger points are a widely recognised phenomenon in clinical practice, there remains much to be elucidated with regards to their pathophysiology, mechanisms of pain referral, and treatment of choice. From the outset, it must be noted that much of the early literature on trigger points, myofascial pain, and fibromyalgia was based on anecdotal reports and the clinical experience of those using this form of treatment. Most popular beliefs are based on theories generated on this basis, and it is only in recent times that a more scientific approach to defining and treating the phenomenon of myofascial trigger points has developed. Despite this increasing interest, much of the fundamental understanding remains based in the theories of the early clinicians and still requires experimental verification.

Trigger points are most often discussed in the setting of myofascial pain syndromes, in which widespread or regional muscular pain is associated with hyperalgesia, psychological disturbance, and significant restriction of daily functioning (Harden et al., 2000). Most patients with these syndromes recall an inciting factor for their pain, however, some may not. Inciting factors may often seem quite trivial when assessed. It is not clear whether the psychological disturbance seen in these patients is a part of the pathology or merely reactive to the chronic pain state. Importantly, psychological disturbance, from whatever cause, will impact on a patient's interpretation of pain (Yunus et al., 1989) and, potentially, their response to treatment.

As trigger points can also occur in the absence of pain syndromes, this paper aims to address the current state of knowledge with respect to trigger points as an isolated phenomenon, rather than addressing treatment approaches to more generalised pain syndromes.

Trigger points can be seen in the setting of occupational or athletic injury due to muscle imbalances, postural deficiencies, or secondary to another underlying pathological process. Examples of the latter include trigger points in quadratus lumborum in association with an irritated lumbar disc, or gluteal trigger points in the presence of hip joint pathology. Desk workers may present with headaches that are reproducible with pressure over trapezius trigger points due to the prolonged muscle contraction in inappropriate postures, or the development of thoracic spine stiffness. It is important to assess for and treat any precipitating or perpetuating factors in the presence of trigger points in order to maximise the chance of a long-term response to any treatment approaches.

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Early writings on trigger points and their treatment were sporadic and lacked uniformity of diagnostic criteria. The Trigger Point Manual (Simons et al., 1998; Travell and Simons, 1992) is the most popular and comprehensive reference for those involved in treating this condition. The theories of pathogenesis contained within this volume have subsequently been challenged, but never conclusively disproved and the definition of trigger points presented is still the most widely used in clinical practice.

2. Trigger points—definition

The classical and most commonly used description of trigger points is that by Simons et al. (1998) and Travell and Simons (1992). Travell and Simons define trigger points as the presence of exquisite tenderness at a nodule in a palpable taut band (of muscle). Trigger points are able to produce referred pain, either spontaneously or on digital compression. The clinical definition came to be that trigger points are localised areas of deep tenderness within a taut band of muscle. They exhibit a local twitch response (muscle fasciculation) or jump sign (whole body movement) in response to digital pressure or dry needling. In their text, Simons et al. (1999) also produced body maps of common trigger point locations and their referral zones. These locations have been noted to have significant similarities to acupuncture points used in traditional Chinese medicine for the relief of pain (Melzack et al., 1977).

Two main types of trigger points are described. Active trigger points are those that may be responsible for the presenting pain complaint. They may also be associated with less readily definable symptoms such as weakness, parasthesia, or temperature changes, and they may have associated referred pain. Latent trigger points present with muscle shortening, and pain occurs only on the application of external pressure. These trigger points may become activated by a variety of stimuli, including poor posture, overuse, or muscle imbalance.

3. Examination findings

On palpation of a muscle, a trigger point is recognised as a local tender spot within a taut muscle band. If a trigger point is active, the patient will recognise the symptoms produced when pressure is applied to it. Latent trigger points will be painful on palpation, but the sensations will not be recognisable. A local twitch response has been described in response to 'snapping palpation' of the taut band, and to the introduction of a needle (Simons et al., 1998). Snapping palpation is described as similar to plucking a guitar string. The fingers are placed over the trigger point and then quickly snapped back over the muscle at right angles to the direction of the muscle fibres (Simons et al., 1998).

In summary, the diagnosis of trigger points relies on finding a local tender spot within a taut muscle band, reproduction of recognisable symptoms, and a local twitch response to snapping palpation or needle insertion.

There are, however, several caveats to bear in mind when establishing examination findings, not the least of which is the lack of a gold standard for assessment of trigger points. This lack of standardised assessment makes validity studies near impossible, although reliability trials have been performed. Wolfe et al. (1992) examined patients with chronic myofascial pain or fibromyalgia, and the most common finding in their subjects was local tenderness and taut muscle bands. Reliability of examination for taut bands, muscle twitch, and active trigger points, however, was problematic.

In a blinded trial of physiotherapists experienced in treating lower back pain (Nice et al., 1992), the reliability of assessment for the presence of three trigger points described by Travell and Simons was poor and it was noted that issues as simple as patient positioning, palpation technique, and the amount of force applied significantly influenced results. Of interest, reliability was not improved when the sample was reanalysed for only those therapists reporting the use of trigger point examination in their routine practice.

In a more recent study, Lew et al. (1997) found that both inter and intra-rater reliability, using two highly trained examiners for assessment of the presence and number of trigger points in asymptomatic patients, was poor. In a study by Gerwin et al. (1997), it was found that extensive training of four clinicians together resulted in improved reliability of identification of trigger points. In a study by Hsieh et al. (2000), it was reported that localisation of trigger points was unreliable in untrained examiners, and only marginally more reliable in trained examiners. Further, Hsieh et al. (2000) found that taut band and local twitch responses could not be reliably assessed, and examination for referred pain had low reliability when extensive training had been undertaken, but was not at all reliable without this. Another study has shown moderate reliability for the presence of local tenderness and production of recognised pain, but poor reliability for twitch responses and the production of referred pain (Njoo and Van der Does, 1994).

4. Pathology

Currently, there is no gold standard pathological test for the identification of trigger points. Therefore, much of the research into the pathophysiology of trigger points is directed towards indirectly verifying the common theories for their formation. Histological studies have been inconclusive, with either non-specific changes of fibrosis and absence of inflammatory cells, or negative findings (Yunus et al., 1986). Imaging of trigger points has not been shown to be reliable with thermography (Diakow, 1988; Swerdlow and Dieter, 1992), or ultrasound (Lewis and Tehan, 1999).

One study that used biopsy indicated that there may be altered levels of high energy phosphates in painful muscles of patients with fibromyalgia (Bengtsson et al., 1986), and this has led to some promising pilot trials on the use of 31P NMR which can assess the levels of different forms of phosphate within the muscle. 31P NMR is a form of nuclear magnetic resonance spectroscopy that can quantify relative amounts of different phosphate compounds in tissues, as each compound is at a different energy state. Phosphate compounds (e.g. ATP, phosphocreatine) in muscle are sources of energy, and quantification could assist in assessing the metabolic status of the muscle. Unfortunately, to date, there is no clear indication of the pathological changes to be expected in myofascial pain.

5. Theories of pathogenesis

The aetiology of trigger points is not clear, but the two most widely accepted theories (energy crisis theory and motor endplate hypothesis), when combined, provide a plausible explanation. There is a third, yet to be experimentally verified theory, which suggests the primary site of pathology to be the spinal nerve, with secondary muscle changes occurring (Gunn, 1997). The more widely accepted theory is centred on the muscle cell and motor endplate being the sites of primary pathology (Simons et al., 1998).

5.1. Energy crisis theory

The energy crisis theory is the earliest explanation of trigger point formation (Bengtsson et al., 1986; Hong, 1996; Simons et al., 1998). This theory postulates that increased demand on a muscle (increased neural input), macrotrauma, or recurrent microtrauma leads to increased calcium release from the sarcolemma and prolonged shortening of the sarcomeres. Prolonged shortening compromises the circulation, with the subsequently reduced oxygen supply leaving the cells unable to produce enough ATP to initiate the active process of relaxation. Ischaemic by-products of metabolism accumulate (Simons, 1996), being in part responsible for some of the pain produced, by sensitisation and direct stimulation of sensory nerves. Unfortunately, there are no studies to date that can confirm such muscle injury as the initiating factor.

The concept of altered muscle metabolism underlying the changes at trigger point sites was investigated by Bengtsson (1986). Muscle energy stores can be measured by the levels of various phosphate containing compounds. Adenosine triphosphate, phosphocreatine, and adenosine diphosphate are compounds capable of donating their phosphate moiety and releasing energy for muscle activity. Adenosine monophosphate and free creatine are the remaining compounds after this process and are, therefore, low energy molecules. In a biopsy study of patients with fibromyalgia, it was found that the levels of high-energy phosphates were reduced and low energy phosphates increased at trigger point sites in patients when compared to non-tender muscle points in both patients and controls (Bengtsson et al., 1986). This supports the idea of a metabolic derangement at trigger point sites. However, lactate and pyruvate are the products of anaerobic muscle metabolism, and their levels were not increased. Therefore, although a pure ischaemic cause is unlikely, there is some evidence to suggest a metabolic abnormality at trigger point sites.

5.2. Motor end plate hypothesis

The energy crisis theory could well co-exist with the motor end plate hypothesis. The motor nerve synapses with a muscle cell at the motor endplate. Needle EMG studies have found that each trigger point contains minute loci that produce characteristic electrical activity (Hubbard and Berkhoff, 1993). These loci are predominantly located at the motor endplate zone (Simons, 2001; Simons et al., 2002). The endplate noise seen on EMG is thought to represent an increased rate of release of acetylcholine (ACh) from the nerve terminal. A small amount of activity at the motor endplate is not enough to cause muscle contraction, but can result in action potentials being propagated a small distance along the muscle cell membrane. This small amount of propagation may be enough to cause activation of a few contractile elements and be responsible for some degree of muscle shortening (Simons, 1996).

5.3. Radiculopathic model for muscular pain

Not all researchers agree with the theories of Travell and Simons. Most opposing theorists postulate a neurological cause as the primary stimulus and trigger points as a secondary phenomenon (Gunn, 1997; Quintner and Cohen, 1994). Gunn (1997) suggested a radiculopathic model for muscular pain and states that ‘myofascial pain describes neuropathic pain that presents predominantly in the musculoskeletal system’ (p. 121). The radiculopathic model is based on all denervated structures exhibiting super sensitivity. From clinical observations, Gunn (1997) states that neuropathic nerves are most commonly found at the rami of segmental nerves, and therefore represent a radiculopathy. If neural injury or compression and partial denervation are the site of origin of this pathology, he believes that it helps to explain the lack of pathology seen in muscle and the sensory, motor, and autonomic changes seen in myofascial pain syndromes.

Gunn (1997) suggests that myofascial pain most often relates to intervertebral disc degeneration with nerve root compression or angulation due to reduced intervertebral space and resultant paraspinal muscle spasm. This is described as a form of neuropathy. This neuropathy then sensitises structures in the distribution of the nerve root, causes distal muscle spasm, and contributes to other

degenerative changes in tendons and ligaments within its distribution that are then perpetuated by the ongoing muscle shortening. Therefore, this theory is not only used to explain trigger point formation, but also conditions such as tendinopathy and enthesopathy.

Based on his theories, Gunn (1997) proposes that long lasting pain relief requires needle treatment to the shortened paraspinal muscles in order to reduce nerve root compression, as well as to trigger points more local to the site of perceived pain.

Quintner and Cohen (1994) argued that the reasoning behind traditional trigger point teaching is circular and excludes the possibility of a non-muscular origin of the pathology. They suggest that the characteristics of the pain from trigger points are not distinguishable from neural pain, and that a primary neurological cause is a much more likely explanation for the local and referred sensations of myofascial pain. To date, no neurophysiological studies have confirmed or denied these claims. Routine nerve conduction testing has not identified any abnormalities, but may be lacking the sensitivity to do so.

6. EMG findings

A pattern of EMG activity said to be characteristic of trigger points was first described in human subjects by Hubbard and Berkhoff (1993) and subsequently investigated by several other researchers (Hong et al., 1995; McNulty et al., 1994; Simons et al., 1995). The pattern has been termed spontaneous electrical activity (SEA), and has subsequently also been confirmed in rabbit studies (Simons et al., 1995). SEA is seen as low amplitude background noise (50 μV), with superimposed high amplitude spike activity (100–700 μV), in a resting muscle. Initially, this pattern of activity was postulated to represent stimulation of intrafusal muscle spindle fibres, which are innervated by the sympathetic nervous system (Hubbard and Berkhoff, 1993). Other researchers have since claimed that it is more likely to represent motor endplate noise (Simons, 2001; Simons et al., 2002). The motor end plate theory states that the background noise (motor endplate noise) represents excessive release of packets of ACh by the motor nerve terminal next to the muscle cell. ACh is a neurotransmitter, which causes mini depolarisations of the post-synaptic muscle cell membrane (Simons, 2001). A muscle contraction requires a large amount of ACh release to initiate adequate depolarisation of the muscle membrane for propagation of an impulse. The spontaneous activity of normal motor endplates shows more discrete, random, and non-overlapping electrical activity (known as mini endplate potentials), rather than the haphazard discharge that has been attributed to trigger points. Within this theory, the origin of the spikes seen in trigger point EMG is unclear. One suggestion is that they may represent propagated single muscle fibre action potentials occurring as a result of summation of background noise with extra ACh released in response to the needle contact.

Although Hubbard and Berkhoff (1993) found no abnormal activity in normal subjects or in non-tender muscle points 1 cm from the trigger points, there remains controversy about the significance of this ‘characteristic’ SEA. In rabbit muscle, this pattern of activity has been identified at trigger point sites, but not control sites, even when the control sites were also located close to motor endplate zones (Simons et al., 1995). The current data on humans is a little more confusing. In an unblinded study on ten human subjects, endplate noise occurred in all muscles tested at clinically determined trigger points (Simons et al., 2002). Such endplate noise, however, also occurred in endplate zones outside of trigger points in four muscles. Taut bands outside of the endplate zone did not exhibit endplate activity. The authors concluded that these potentials are characteristic of, but not restricted to, trigger points.

EMG has been found to exhibit SEA in both patients with long-term myofascial symptoms and asymptomatic patients with identified trigger points. In the study by Hubbard and Berkhoff (1993), the onset of recorded EMG activity corresponded well to the subjects’ report of the onset of pain with needle advancement.

SEA in trigger spots in rabbit muscle has been shown to reduce after dry needling treatment when compared to a control side (Chen et al., 2001). This reduction, however, occurred more reliably if local twitch responses were observed in response to needle insertion. This finding suggests that any effect of dry needling may be due to a more complex mechanism than muscle trauma alone.

7. Muscle pain

As early as 1938, the production of characteristic patterns of local and referred muscle pain was described in response to injection of hypertonic saline (Kellgren, 1938). Muscle pain is likely to be transmitted by Group III (A delta, thin myelinated) and Group IV (c, non myelinated) afferent nerve fibres, as for cutaneous pain (Franz and Mense, 1975; Simone et al., 1994). Neurotransmitters implicated in this pain response include bradykinin (Franz and Mense, 1975), serotonin (Ernberg et al., 2000), and prostaglandins (Hedenberg-Magnusson et al., 2001). Interestingly, however, serotonin has been shown to increase muscle pain in healthy volunteers, but not subjects with myofascial pain (Ernberg et al., 2000). As well, injection of a serotonin antagonist has not been shown to influence muscle pain (Ernberg et al., 2003), and serotonin has also been shown to be important in the increase in pain threshold after electrical muscle stimulation in rats (Hoffmann et al., 1990). Muscle pain has been positively correlated with muscle levels of prostaglandin E2 (Hedenberg-Magnusson et al., 2001). Although substance P and calcitonin gene related peptide

(CGRP) are important transmitters in the nerve endings in muscle, and also at the spinal cord, they are not directly algesic compounds in muscle (Graven-Nielsen and Mense, 2001).

Muscle pain from the injection of capsaicin (a substance used for experimental pain production) has been shown to be of lower intensity, but more likely to produce referral, either to skin or deeper structures when compared to intradermal injection of the same quantity and concentration (Witting et al., 2000). Thus, it would seem that pain from muscle is more likely to be referred, and although the exact mechanism of this remains to be investigated, it is likely to relate to central (spinal) mechanisms. Mechanoreceptors in muscle have a lowered stimulation threshold in the presence of excitatory compounds such as bradykinin (Graven-Nielsen and Mense, 2001).

8. Trigger point pain

Although, as previously stated, there is no evidence of inflammation or increased levels of nociceptive transmitters in the region of trigger points, the most popular theories proposed to date assume that injury and mediator release is the precipitant of trigger point related muscle pain, with these sensitised nociceptors then having increased responses to normal mechanical stimuli. Theories implicating a primary neurogenic cause do not share this weakness, although lack any confirmatory data. Further clarification of this area is required.

Once a painful stimulus is established, by whatever means, dorsal horn neurons may be sensitised and new receptive fields opened due to the flux of substance P and other transmitters at the spinal cord in response to the initial pain (Mense, 1996). This neural plasticity may help to explain referred muscle pain and possibly be responsible for the misinterpretation of signals previously recognised as innocuous. Neural plasticity may be important in the progression from acute to chronic pain (Bendtsen et al., 1996).

9. Pain referral from trigger points

The traditional theory used to explain the phenomenon of referred pain is the convergence projection theory. This states that each dorsal horn neuron has connections from more than one body part. Noxious stimuli are only expected to arise in one of those body parts. When a noxious stimulus is received from another area, it is misinterpreted as coming from the usual recognised site of pain (Gerwin, 1994; Mense, 1993).

A modification of convergence projection theory postulates that not all convergent connections are active all the time, but previously dormant spinal cord connections are unmasked in response to a painful stimulus (Hoheisal et al., 1993; Hong, 1996; Mense, 1996). There is strong agreement that the phenomenon of referred muscle pain must have a central basis. It is thought that in a resting state, each dorsal horn neurone has a receptive field in the body from which it receives noxious input. Mense (1996) and Hoheisal et al. (1993) have applied stimuli to known receptive fields of specific dorsal horn neurons in rats and found that new receptive fields have opened for these neurons. That is, the neurons now perceive noxious input as coming from more than one source and a pain referral is experienced. Hong (1996) showed that this referral can cross to different spinal cord levels in association with increased spinal cord levels of substance P and CGRP. It is, therefore, theorised that the increased release of substance P and CGRP in the dorsal horn in response to a noxious stimulus diffuses around several levels of the spinal cord and increases the sensitivity of those areas to noxious input. The levels of these two transmitters have been found to be independent, and it is not known if their source is local neurons or release from higher centres (Vaeroy et al., 1989). What was previously an innocuous stimulus may now be perceived as pain, and pain may be perceived at a seemingly unrelated anatomical site. This may also apply to the newly opened receptive field, such that a pressure or tightness sensation in the muscle is interpreted as pain (Bendtsen et al., 1996).

Muscle spasm, as seen in many conditions of muscle pain may relate to a connection between dorsal horn neurons in the spinal cord and gamma afferent neurons. Gamma afferent neurons supply muscle spindles and are responsible for reflex muscle shortening, such as that seen with tendon reflexes. Inhibition of dorsal horn neurons indirectly inhibits discharge from gamma afferents (Xian-Min et al., 1992).

10. Clinical precipitants for trigger point formation

Trigger points are thought to form in response to increased or altered muscle demands. Muscle overload, as often seen in the pre-season conditioning phase of sport training, is one such example. Other mechanisms of increased or altered muscle demands include prolonged muscle contraction, such as in workplace postural errors, proximal nerve compression and resultant muscle spasm, and post-trauma (Simons et al., 1998). Latent trigger points are thought to become activated in response to the same conditions that cause trigger point formation, that is, muscle overload, prolonged muscle contraction, or nerve compression.

Trigger points can also be influenced by descending factors such as stress or constitutional illnesses. The sympathetic ‘flight or fight’ response to stress is related to increases in the amount of circulating catecholamines. It has been shown that the EMG activity in trigger points can be reduced by the use of sympathetic antagonists (Chen et al., 1998), and that it increases at times of stress (McNulty et al., 1994).

Trigger points should, therefore, be considered when assessing for the sources of pain in many different clinical scenarios. For example, sedentary workers presenting with head or neck pain may exhibit thoracic spine tightness or signs of early degenerative change in their cervical spine and may have a history of poor posture at work. They may also have some elements of their pain reproduced with palpation of trigger points in the trapezius, long neck extensors, or sternocleidomastoid muscle. Similarly, a lumbar disc injury is often responsible for significant pain, but elements of this pain may be reproduced with pressure on trigger points in the paraspinal, quadratus lumborum, or gluteal muscles. Osteitis pubis may be accompanied by trigger points in the adductor muscles or the gluteals. Importantly, trigger points can form in either of these regions purely in response to an increase in load, without any specific underlying injury.

Of interest, is that clinical syndromes seen may not reflect the locality of the trigger points, but rather, their referral zones. Examples include posterior thigh pain reproduced from gluteal trigger points or achilles tendon pain reproduced from calf trigger points. When assigning causality for pain to trigger points, it is essential to assess carefully for the amount of contribution to the overall syndrome. To illustrate this, consider the following; the commonest source of pain in the Achilles region is still pathology at or around the Achilles tendon, but when this diagnosis is not clear cut on clinical assessment, trigger points at a proximal location may enter into the therapist's consideration. Likewise, when the pain complaint is out of proportion to the evident local pathology, or when treatment of a local area fails to completely relieve symptoms, if trigger points are a possible contributing factor, they should be considered and carefully assessed. It is important not to assign significance to trigger points if they do not produce reproduction of a *recognisable* pain. Non-specific pain does not identify a contributing trigger point according to the classical definition (Simons et al., 1998). Recognisable pain is one of the essential criteria for diagnosis of an active trigger point.

11. Trigger point therapy

Trigger point therapy is essentially divided into invasive and non-invasive techniques. Non-invasive techniques are those that have been traditionally employed by physical and manual therapists. In recent years, there has been marked increase in the use of invasive therapies, in particular, dry needling to manage trigger points. Anecdotally, all therapies have their supporters. Scientifically, however, very few of them stand up to scrutiny. Of those that do produce a result, a clear mechanism for this improvement has not been found, but all share the feature of application of a noxious stimulus. This section will describe the evidence for a variety of non-invasive and invasive methods of managing trigger points.

11.1. Non-invasive therapies

11.1.1. Stretching

Stretching after the application of a vapocoolant spray is reported by Travell and Simons (1983) to be the 'single most effective treatment' for trigger point pain. An attempt was made to clarify this, using pain scales and pressure threshold as outcome measures for response to spray and stretch techniques in patients with chronic neck and head pain (Jaeger and Reeves, 1986). Although this study used contralateral sides as controls in symptomatic patients and was not blinded, it did show a significant reduction in both reported pain and pain pressure threshold after the intervention. There was no correlation between the improvements in these two parameters. It was postulated that the coolant spray acted by a counter-irritation mechanism. From these results, it is impossible to say if spray or stretch in isolation might have the same effects.

Hanten et al. (2000) compared the results of a home program of ischaemic pressure and stretching to a program of stretching alone in subjects with trigger points in the trapezius region. Ischaemic pressure combined with stretching resulted in a greater improvement in pain scores and pain pressure threshold. The pressure was applied prior to the stretching and, therefore, could well have been acting in a counter-stimulatory fashion. Importantly, this research shows a benefit of massage techniques above the effect of stretching alone.

11.1.2. Transcutaneous electrical nerve stimulation

Transcutaneous electrical nerve stimulation (TENS) is often postulated for use in chronic pain and can be used at different frequencies and intensities to attempt to achieve relief. Graff-Redford et al. (1989) investigated the use of 100 Hz, 2 Hz and control TENS on subjects with trigger point related chronic pain in the thoracic, neck, or head region. Low frequency and control TENS had no effect on pain, whereas the high frequency resulted in significant pain relief. None of the modalities resulted in any change in pain pressure threshold. Graff-Redford et al. (1989) postulated the results to be due to modulation of central pain sensitivity. Hsueh et al. (1997) also found a reduction in pain with the use of 60 Hz TENS when compared to placebo, but interestingly also found a significantly greater improvement in pain threshold in the active group. A third arm of their trial used electrical muscle stimulation or interferential. Although this group did not report the same benefits on pain, they did exhibit a marked improvement in range of cervical lateral flexion. Again, a central mechanism of pain relief was postulated, but it was also suggested that both modalities be tried together to maximise effects on range of motion and pain. There is no data to date on the expected duration of any improvement from these therapies.

11.1.3. Ultrasound

A search of the literature only identified one well-designed trail on the use of ultrasound for the treatment of trigger points in patients with neck and shoulder myofascial pain. Therapeutic ultrasound was used in a three-armed trial (Gam et al., 1998) with subjects randomised to receive ultrasound with home exercise and massage, sham ultrasound with home exercise and massage or a non-intervention control group. Both intervention groups showed significant improvements in the number and sensitivity of trigger points, but the ultrasound had no additional advantage. Despite the use of massage and stretching in this trial, there was no reduction in pain scores or analgesic use in the subjects of any group. This contradicts the findings of the other studies on stretching discussed above, but no explanation is offered.

11.1.4. Laser

Although some studies indicated potential benefits for laser on trigger points (Ceccherelli et al., 1989; Olavi et al., 1989; Snyder-Mackler et al., 1986), this has not been borne out in all research (Thorsen et al., 1992; Waylonis et al., 1988). Olavi et al. (1989) investigated infrared laser in a double blinded trial on 18 patients with upper limb active trigger points and found a significant reduction in pain threshold immediately, and a larger reduction at 15 min after therapy in the active laser group. Ceccherelli et al. (1989) used pulsed infrared laser in neck pain and found an improvement that was higher in the active group, but only at day 24 after 12 treatments and after 3 months of follow up. Snyder-Mackler et al. (1986) used helium–neon laser in conjunction with routine physiotherapy in a double blinded fashion. They found a significant increase in skin resistance over trigger point sites with laser therapy that was postulated to accompany the resolution of the pathology, however, there is no data to date to corroborate this proposed association. In contrast, Waylonis et al. (1988) found no difference between five treatments of helium–neon laser and placebo on pain at any time point in a double blinded cross over trial. Thorsen et al. (1992) used low level laser on neck and shoulder pain in a double blind trial, and found that subjective reports of improvement were higher in the placebo group. There is, therefore, no reproducible evidence to date of the benefit of using laser therapy in the treatment of muscle pain.

11.2. Invasive therapies

Literature exists from the first half of last century detailing the use of injection therapies for the treatment of muscle pain, described as ‘fibrositis’. Most of these involved the injection of local anaesthetic formulations, but hypotonic glucose, urea, and quinine were also proposed (Button, 1940; Howard, 1941; Ray, 1941; Souttar, 1923; Steinbrocker, 1944). All of these reports on the treatment of muscle pain with injection were case reports, case series or letters to journals, however, and it wasn’t until much later that the therapies were more formally investigated. Most of the substances listed are irritants to muscle and are not considered in modern practice, however, local anaesthetics are still widely used. Interestingly, very early in the discussion, Steinbrocker (1944) suggested that the mere insertion of a needle somewhere in the region of the pain without introducing analgesic solutions has been reported to give frequent lasting relief. This is the basis on which investigation of dry needling began, and, to date, there is no evidence to convincingly refute his statement.

11.2.1. Local anaesthetic

Local anaesthetic is certainly the substance most investigated for injection into trigger points. Many different agents have been used, and most of them have equivalent results to the injection of normal saline (Frost et al., 1980; Garvey et al., 1989; Hameroff et al., 1981). One consistent finding is that the pain relief, when seen, well outlasts the expected half-life of the injected solution, suggesting mechanisms of pain relief above the pure pharmacological one of the anaesthetic. It must be noted, that in these studies it is extremely difficult to have a true placebo, particularly if Steinbrocker’s early comments are taken into account.

Overall, local anaesthetic injection is most likely to improve subjective outcome measures (e.g. pain scores) (Frost et al., 1980; Garvey et al., 1989; Hameroff et al., 1981; McMillan, 1997), although improvements in range of motion and pressure threshold have been reported (Hong, 1994). This may well be explained by central modulation of pain as the dominant factor in relief. This is supported by the fact that in all of the above research reports pain relief far outlasts the half-life of the local anaesthetics used, making it unlikely to be a purely local phenomenon.

Local anaesthetic use can result in reversible myotoxicity, seen as ischaemia and necrosis of muscle fibres in the injected region (Benoit, 1978; Foster and Carlson, 1980). These changes are much worse in the presence of vasoconstrictors (adrenaline). Perhaps this is simply the optimum form of counter-irritation, where a true inflammatory response produces mediators sufficient to reset the spinal transmission of pain, and hence, result in prolonged pain relief.

Injection of the skin over the trigger point with sterile water has been postulated as an appropriate treatment if counter-stimulation is the active mechanism (Byrn et al., 1993). Although there was evidence for prolonged pain relief in long standing neck pain patients in the study of Byrn et al. (1993), the protocol tested involved a large number of injections at multiple points over three separate treatments. Sterile water injection (more painful) resulted in greater improvements than saline injection (less painful). Subcutaneous injection is a very painful procedure that is generally not well tolerated by patients, and a large number of injections over multiple treatments are likely to influence patient compliance, despite

the existence of some evidence to say that it can reduce long standing pain.

Of interest to those treating mainly acute or athletic pain is that, injection therapy has been shown to have a more rapid onset of pain relief in those with isolated regional pain than in those with multiple regions involved and longer standing pain (Hong and Hsueh, 1996).

11.2.2. Botulinum toxin

Botulinum toxin injection has been proposed for use in trigger point therapy, based on the assumption that there is excessive ACh release from motor nerve terminals. Botulinum toxin is produced by the bacteria *Clostridium botulinum* and acts by blocking the release of ACh at the neuromuscular junction. Whilst one early pilot trial suggested a potential benefit over saline at 2–3 weeks (Cheshire et al., 1994), this was not borne out in subsequent research. In a double blind design of patients with unilateral neck pain, injection of saline or 50 or 100 units of botulinum toxin produced equivalent results. There was no dry needling or placebo groups. All three treatments resulted in similarly improved pain scores and pain pressure thresholds (Wheeler et al., 1998). An interesting but unexplained finding is that subsequent treatment of the non-responders from all groups (saline, 50 and 100 units Botox) with 100 units of Botox resulted in a significantly better response in those who received 100 units of Botox as their initial treatment.

11.2.3. Dry needling

Dry needling involves multiple advances of an acupuncture-type needle into the muscle in the region of a trigger point, aiming to reproduce the patient's symptoms, visualise local twitch responses, and achieve relief of muscle tension and pain. Unfortunately, there are few well-designed published studies of this technique. In an early study, dry needling was found to be equivalent to local anaesthetic, corticosteroid, and coolant spray in the treatment of lower back pain (Garvey et al., 1989). In a more recent study, Karakurum et al. (2001) investigated subjects with tension headache and found that muscle dry needling resulted in equivalent improvements in pain and neck range of motion compared to placebo (subcutaneous) dry needling. The methods of range of motion measurement in this study, however, were not clearly stated.

In another study, a four armed blinded trial in dental patients found no improvement in pain pressure threshold, but equivalent improvements in pain intensity and unpleasantness, regardless of group allocation (McMillan et al., 1997). Groups randomly received placebo needling, local anaesthetic plus placebo needling, dry needling plus placebo local anaesthetic, or placebo dry needling plus placebo local anaesthetic over two sessions. Interestingly, the placebo dry needle used did penetrate to the subcutaneous, but not the muscular level.

In a double blind randomised controlled trial, dry needling versus a non-penetrating placebo needling of the gluteal muscles for trigger point related referred hamstring pain in athletes found no change in the chosen range of motion measures, but an equivalent improvement in activity related pain scores in both groups (Huguenin et al., submitted for publication).

A recent trial evaluated the use of acupuncture at distal points, local dry needling, and sham laser acupuncture on patients with chronic neck pain (Irnich et al., 2002). Although none of the groups exhibited a large reduction in pain, it is interesting to note that the acupuncture group improved by 30%, whereas the other two groups did not. In the dry needling protocol, the needle was moved within trigger points until a twitch response was seen. Twitch responses are painful and do correlate with post-treatment soreness, and this may therefore have clouded the results seen. The most important point to be taken from this trial is the lack of response when a non-noxious placebo (sham laser) is used.

The major drawback to the use of dry needling over local anaesthetic injection is the higher incidence of post-treatment soreness (Hong, 1994). This would appear to be maximal in the 24 h after therapy and is usually manageable with heat packs and stretching, but may be intolerable to some patients, and therefore care with patient selection is important.

12. How do these treatments work?

Given that dry needling appears to be as effective as local anaesthetic, but has no convincing benefit over placebo treatment, the mechanism of action of all of these therapies remains to be clarified. The placebo treatments used have varied, but all have still involved the application of a penetrative or non-penetrative but nonetheless noxious stimulus to the skin. Central opioid release is thought to produce a global reduction in pain perception by gating spinal cord pain impulse transmission. This is known as diffuse noxious inhibitory control. Reversal of local anaesthetic-induced analgesia has been observed with the administration of an opioid antagonist (Fine et al., 1988). This implicates the endogenous opioid system, which acts to produce hypoalgesia at a spinal cord level, to at least a partial extent in the reduction of pain seen with this therapy. This is the system implicated in the production of a runners' high (Koltyn, 2002), and it has been suggested to be important in production of the placebo effect (Grevert et al., 1983).

Beyond these suppositions, there is little hard evidence to date on the mechanisms of action of any of the therapies discussed. It is a notoriously hard area to research due to the interactions of so many systems on both a regional and whole-body level. Stress and the sympathetic nervous

system have been shown to increase pain perception, but the effect of these treatments on this system has not been conclusively evaluated.

13. Conclusions

Although trigger point related pain is widely recognised by health professionals, reliable clinical evaluation and imaging for diagnosis still eludes us. Many treatments in widespread use are poorly validated and not necessarily more effective than placebo. The application of a noxious stimulus may be the key to obtaining improvements in pain perception. Less stimulatory interventions, such as laser and ultrasound, have not convincingly been shown to be beneficial. Most stimulatory interventions are able to induce subjective improvements in pain scores, if not objectively measurable improvement. Stretch, TENS, injection therapies, and dry needling have all shown benefit. Unfortunately, we have extremely limited data comparing results between different therapeutic approaches, in particular, invasive versus non-invasive from which to draw clinical conclusions.

Studies of invasive treatment utilising a placebo intervention have not found the active treatments to be any more effective. Importantly, the placebo interventions used are themselves, stimulatory. The amount of stimulation required to induce analgesia is currently unknown. Despite EMG evidence of changes in the regions of trigger points, muscle penetration does not seem to be necessary to produce an analgesic effect. The evidence is trending towards the magnitude of the effect being consistent regardless of the therapy chosen, or the depth of needle penetration, as long as some counter-stimulation is involved. The relative contributions of local tissue effects and central pain modulation to these clinical improvements require further investigation.

The choice of therapy can, therefore, be guided by patient specific criteria, the therapist's experience and qualifications, and patient preference. The discomfort induced by the therapy, the likelihood of post-treatment soreness, and the current functional level of the patient are important to consider. Dry needling may not be appropriate for someone with long standing chronic pain that is known to flare after deep massage treatment, but it may be the treatment of choice for an athlete with a regional pain that has not responded to previous soft tissue work. Needle phobias or other known adverse reactions will limit therapeutic choices.

Regardless of the treatment chosen, it is imperative to remember that trigger points are rarely an isolated phenomenon, and the key to successful long-term outcomes of any treatment regime is addressing the precipitating and predisposing factors for each particular patient.

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