RHEUMATOLOGY

Rheumatology 2015;54:392–399
doi:10.1093/rheumatology/keu471
Advance Access publication 3 December 2014

Review

A critical evaluation of the trigger point phenomenon

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Abstract

The theory of myofascial pain syndrome (MPS) caused by trigger points (TrPs) seeks to explain the phenomena of muscle pain and tenderness in the absence of evidence for local nociception. Although it lacks external validity, many practitioners have uncritically accepted the diagnosis of MPS and its system of treatment. Furthermore, rheumatologists have implicated TrPs in the pathogenesis of chronic widespread pain (FM syndrome). We have critically examined the evidence for the existence of myofascial TrPs as putative pathological entities and for the vicious cycles that are said to maintain them. We find that both are inventions that have no scientific basis, whether from experimental approaches that interrogate the suspect tissue or empirical approaches that assess the outcome of treatments predicated on presumed pathology. Therefore, the theory of MPS caused by TrPs has been refuted. This is not to deny the existence of the clinical phenomena themselves, for which scientifically sound and logically plausible explanations based on known neurophysiological phenomena can be advanced.

Key words: nerve, muscle, referred pain, pain mechanisms, philosophy of science.

REVIEW

Rheumatology key messages

- The theory of myofascial pain based on trigger points is conjecture that has been put forward as established knowledge.
- The key phenomenon of muscle tenderness demands a robust plausible explanation based on neurobiology.
- Clinicians cannot ignore the important role of contextual factors when evaluating outcomes of their treatment for myofascial pain syndrome.

Introduction

The phenomena of muscle pain and tenderness in the absence of obvious disease are well recognized but poorly understood. Myofascial pain syndrome (MPS) is a popular explanatory model, which posits a local (muscle) origin of nociception called the trigger point (TrP) and advocates local treatment, primarily direct manipulation of TrPs using manual pressure or needles, the latter with and without injectate [1, 2]. These forms of treatment are being practised worldwide by physicians, physical therapists, chiropractors and various unlicensed and unregulated practitioners [3].

But does the evidence support these concepts? Are the hypotheses generated by MPS theory scientifically sound? And are treatments based on this theoretical model beneficial?

This article will show that the theory is flawed both in reasoning and in science. In seeking a resolution, two testable hypotheses are identified that point the way to neuroscientific explanations for the observed clinical phenomena.

Evolution of MPS theory

It has long been believed that muscle pain might originate from focal lesions within connective tissues [4, 5]. The initial description put forward by Stockman [6] was of fibrositic nodules, which were suggested to harbour low-grade inflammation that activated sensory fibres innervating muscle spindles and the interstitial tissues between muscle fibres. However, Stockman's claim that 'the essential lesion is a chronic inflammatory hyperplasia of white fibrous tissue in patches' [7] has never been confirmed [8].

An infective aetiology of such nodules was proposed, but other conjectures included microtrauma, exposure to environmental extremes, nerve root irritation and psychoneurosis [8, 9].

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Submitted 7 August 2014; revised version accepted 23 October 2014.

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Kraus (cited by Simons [2]) speculated that palpable muscle hardening of unknown cause could set up a reflex increase in muscle tension, resulting in a pain–reflex–pain self-perpetuating cycle that could be disrupted by ethyl chloride sprayed onto the overlying skin or by local injections of anaesthetic. Pain theorists William Livingstone [10] and John Bonica [11] favoured this vicious circle hypothesis, as did others [12, 13].

Speculation took a new turn when Travell and Rinzler [5] conceptualized that pain felt in voluntary muscles is myofascial in origin. Their claim, that 'trigger areas in myofascial structures can maintain pain cycles indefinitely' [5], was reminiscent of the vicious circle hypothesis.

Travell and Simons formalized the construct of 'myofascial pain arising from trigger points' [14]. Not only were TrPs described in exactly the same way as fibrositic nodules had been, but it was also asserted that they could potentially develop within every voluntary muscle and in multiple locations within a given muscle.

The theory of MPS comprised two essential components: the TrP, a localized area of tenderness or hyperirritability deep within voluntary muscle; and a predictable discrete zone of deep aching pain, which could be located in the immediate region of or remote from the TrP, and which was worsened by palpation of the TrP [4, 14].

Travell and Simons [14] composed anatomical charts of TrPs and their characteristic pain referral patterns. However, it appears that their diagrams had 'sometimes been chosen arbitrarily, there being no accepted standard' [15].

Located within palpable taut bands, TrPs were said to represent shortened (contractured [16]) muscle fibres. On snapping palpation or insertion of a needle, a local twitch response could be elicited, which was accompanied by an irritable EMG response [14]. In contrast to a normal muscle, one containing a TrP was said to exhibit both antalgic inhibition when tested for its strength, and intolerance to passive stretch.

To explain the puzzling onset of pain in ostensibly lesion-free tissues, Travell and Simons [14] found it necessary to invent the latent TrP, a site of potential tenderness within a muscle unassociated with spontaneous pain but having the potential to be activated by a myriad of factors, within or outside the body. In an attempt to extend the theory to explain more widespread pain, they claimed that TrPs could self-propagate to become secondary TrPs in other muscles and even to metastasize throughout the bodily musculature.

The recent conjecture that peripheral pain generators can reside within muscles (i.e. myofascial tissues), and be responsible not only for spontaneous pain but also for the initiation and maintenance of profound changes within the CNS (known as central sensitization) rests upon these dubious premises [17, 18]. Similarly, prominent rheumatologists are among those who maintain that TrPs are responsible for the initiation and maintenance of the syndrome of chronic widespread pain (FM) [17–23].

Beliefs in TrP theory and the associated concept of MPS continue to be strongly held [24], despite the fact that such beliefs exemplify circular reasoning: TrPs cause myofascial pain because painful muscles contain them [25].

Review of the evidence

Clinical diagnosis

An extensive review identified at least 19 different sets of diagnostic criteria used for the MPS/TrP syndrome, and concluded there was a lack of consistency and consensus on case definition [26]. The authors suggested that until reliable diagnostic criteria had been established, 'there is a need for greater transparency in research papers on how a case of MTrP [*sic*] pain syndrome is defined, and claims for effective interventions in treating the condition should be viewed with caution' [26]. A similar study found that the diagnosis of MPS from putative TrPs was based on a clinical test of unknown reliability and validity with no accepted reference standard [27].

In studies of inter-examiner reliability, examiners were given the muscle to palpate with or without an accompanying diagnosis [28–31]. In one study, extensive training coupled with the use of an algometer resulted in examiner agreement that the phenomenon could be localized [29]. Another study reported that the assessments of an individual examiner were consistent from one test to another [31], and that more experience in assessment leads to better inter-examiner agreement [30]. These studies suggest that when shown where a problem may exist, examiners may agree. However, when blinded as to diagnosis, those who claimed expertise in the field were unable to detect putative TrPs in the majority of subjects with a MPS diagnosis [32]. In this study, there was virtually no inter-examiner reliability for either putative TrPs or taut bands. This finding questions the reliability of the diagnostic criteria used by these experts. More recent studies [33, 34] have also reported poor inter-examiner diagnostic reliability and poor methodological quality [35].

In summary, physical examination cannot be relied upon to diagnose a condition that is supposed to be defined by that physical examination. That is, the pathognomonic criterion for making the diagnosis of MPS is unreliable.

Pathology

The first histological analysis of fibrositic nodules reported diffuse inflammatory changes [9]. These findings were not confirmed, although tender muscles contained increased extracellular fluid [36]. The authors suggested that the resulting turgor might explain the observed finding of mechanical tenderness.

The term myogelosis describes a change in muscle structure analogous to TrPs [37]. Samples taken from unfixed cadavers following detection of such areas showed altered histology [37], but the clinical relevance to the findings on palpation is unknown.

Tissue biochemistry

Shah *et al*. [38, 39] employed microdialysis to sample tissue fluid within and near to a palpated trigger zone in trapezius muscles in patients with a diagnosis of TrPs and also in normal pain-free subjects. Samples were taken from the following regions: normal (no pain, no TrP), active (pain and TrP detected) and latent (no pain, TrP detected). Samples were also taken from asymptomatic gastrocnemius muscles. Elevated levels of calcitonin gene-related peptide (CGRP), substance P (SP), norepinephrine, TNF-α, IL-1, IL-6 and low pH were reported in fluid from all sampled regions of symptomatic patients. However, elevated levels were also found in uninvolved, control muscle areas.

These reported alterations in biochemical milieu are consistent with inflammation due either to tissue damage or to altered peripheral nerve function, in contrast to pathology necessarily being in the tissue sampled [40, 41].

EMG studies

In one study, EMG examination of TrPs failed to provide evidence of ongoing denervation or focal muscle spasm [42]. But another study did report spontaneous electrical activity (i.e. endplate noise and spikes) in regions considered to be TrPs in patients with chronic tension headache and pericranial muscle tenderness [43].

Simons *et al*. [44] addressed the question of whether endplate noise and spikes arise from normal endplates by performing EMG on 25 patients who met the ACR 1990 criteria for FM and 8 pain-free subjects in whom latent TrPs had been identified by manual palpation of taut bands and characteristic referral of pain [*sic*] [45, 46]. Unfortunately, the researchers conflated the TrPs of MPS and the tender points of FM, another issue yet to be resolved [47]. They concluded that endplate noise is characteristic of but not restricted to TrPs, and that the finding could not be considered a reliable diagnostic criterion [45, 46].

An alternative interpretation of these EMG findings is that insertional and spontaneous activity (i.e. endplate noise) from single muscle fibres generated by the activation of i.m. nerve termini irritated by the needle was being recorded [48]. Nonetheless, it is still asserted that spontaneous electrical activity is one of the characteristics of myofascial TrPs [49].

Imaging studies

Seven patients with a 3-year history of myofascial pain associated with the presence of a taut band in the upper trapezius muscle were examined using magnetic resonance elastography [50]. A signature chevron-like pattern was reported, with its leading edge coincident with the physician-identified taut band. The authors did not offer diagnostic criteria nor make any comment on the relationship of a taut band to a TrP. A subsequent study of eight subjects, four of whom were said to have MPS and four of whom did not, is open to the same criticism [51].

Attempts were made to visualize TrPs using diagnostic US of the anterior abdominal wall of 10 patients [52]. The points in question appeared as a mixed echoic area in the rectus abdominis muscle that became prominent on injection of local anaesthetic solution [52]. They conceded that the findings could have been coincidental. Also, the image presented is consistent with the normal sonographic appearance of abdominal muscles [53].

In another study, 44 patients with acute cervical pain and at least one putative TrP identified by palpation in the upper trapezius were evaluated using sonoelastography and Doppler imaging [54]. The authors claimed to have measured TrP size and to have distinguished normal muscle from active and latent TrPs. Although the data on which these assertions were made were not presented, the authors found no correlation between claimed TrP area and pain pressure threshold. The absence of pain-free control subjects is yet another flaw. These methodological concerns do not lend credibility to the findings.

Animal models

Animal models are often informative about pathophysiology in ways that are impossible to demonstrate in humans. To be considered relevant, models must have symptomatic and/or pathological similarities to the condition being studied. For TrP research, no such model exists.

Simons and Stolov [55] biopsied ostensibly normal canine muscles, seeking to correlate palpated taut bands with morphological and histological changes. The findings were negative, given that there was no indication of pain or a pathological condition present prior to these studies. The researchers observed 'rubbing palpation produced a transient contraction which could be primarily responsible for the sensation of a hardness palpated in the dog muscles' [55]. This is the myotatic reflex, which correlates with the twitch response also evocable on palpation of normal human muscle [56].

Based upon the conjecture that '... latent TrPs can be identified in almost all skeletal muscles of normal adults' [14], a rabbit model of TrPs was proposed [57, 58]. Rabbit leg muscles were palpated until they exhibited a myotatic reflex. Such muscles were considered to contain taut bands and, by assumption, TrPs. A number of papers have since been published using this model [58–65], but have not offered evidence of clinical relevance.

Delayed onset muscle soreness

Studies of delayed onset muscle soreness (DOMS) have been undertaken using eccentric exercise to cause symptoms, in both humans and animals. Although DOMS has been related to TrPs in only one study [66], this model was proposed for MPS [67]. The relevant experiment was performed in humans and used eccentric exercise of the extensor digitorum of the middle finger [66]. Following the development of DOMS, the muscle was palpated, revealing a tender band judged to be taut. However, since the muscle itself is a band, relating the description to TrPs seems meaningless. It should be noted that DOMS is

self-limiting, whereas whatever phenomenon is occurring with chronic muscle-related pain is not. The relevance of DOMS to TrPs remains unclear.

Integrated hypothesis

Dommerholt *et al*. [68, 69] postulated that low-level isometric muscle contraction or eccentric or submaximal concentric contractions could result in muscle dysfunction or damage, and that the formation of TrPs would follow. According to Gerwin *et al*. [70], excessive release of acetylcholine from dysfunctional neuromuscular endplates might be responsible for the taut band phenomenon (i.e. focal muscle contraction modulated by muscle spindle afferents) and that these bands could in turn produce muscle ischaemia, apparently by compressing adjacent capillaries supplying the muscle. This physiological process could precipitate an energy crisis in the relevant working muscle, which would respond by releasing proinflammatory molecules, thereby activating nociceptive neurons. Although there is no experimental evidence in support of this hypothesis, others [71, 72] have accepted the motor endplate and the energy crisis theories of tonic muscle hyperactivity and TrP formation.

Recent studies of induced muscle pain in humans has not provided evidence for a reflex increase in fusimotor drive and spindle discharge [73, 74]. In fact, persistent musculoskeletal pain is associated with decreased agonist muscle tone [75]; in other words, digital pressure or other stimuli that evoke pain will decrease the tone of the muscle stimulated. The validity of the paradigm that correlates endplate activity or noise with pain arising from the TrP became further suspect when it was reported that injection of botulinum toxin A in the region of a TrP had no effect on pain intensity or mechanical pain thresholds, but did significantly reduce motor endplate activity and the EMG interference pattern [76]. Finally, the vicious circle hypothesis has now been laid to rest by microneurographic recordings in humans performed during sustained muscle pain [73, 74]. The integrated hypothesis remains conjecture in the face of conflicting data.

Treatment

Non-invasive interventions that have been advocated include compression of the TrP, spray and stretch, transcutaneous electrical stimulation and, more recently, high-intensity focused US [77]. Invasive treatments have included injection of local anaesthetic agents, injection of CSs, injection of botulinum toxin, needle acupuncture and dry needling [78].

In their systematic review, Cummings and White [79] were unable to find evidence that needling therapies have any specific effect. Their later review of 1517 studies found only seven that were of high enough quality for meaningful analysis [80]. Rickards [81] also found limited strength of evidence for any treatment of TrPs.

Another review remarked upon the heterogeneity of the populations being treated, and the lack of widely accepted standard diagnostic criteria for MPS [82]. This review also concluded that there was insufficient evidence to support the use of most interventions.

A systematic review of botulinum toxin A for TrP treatment located 21 randomized controlled trials, with 12 eligible for consideration but only five suitable for inclusion, and concluded that the current evidence does not support any therapeutic value [83]. Again, these authors reported that the data were limited and that the patient populations were heterogeneous.

These studies provide little evidence that dry needling of TrPs is associated with a treatment effect compared with standard care [3]. They are based on small sample sizes, uncertainty as to whether TrPs were the sole cause of pain, as well as neglect of technical issues such as the variability in the location of TrPs and the depth of needle insertion.

With these results in mind, why do many clinicians insist that their treatments work? One explanation is that the treatments are rarely performed in an isolated fashion; that is, treatment is accompanied by manual therapy, home exercises and stretching.

Contextual effects could explain the plethora of anecdotal responses to treatment [84, 85]. This is not unexpected when a medical treatment with high face validity is based solely on practical experience rather than reflecting a rational approach based on pathogenesis. Apparent effectiveness of any treatment may be attributed to the natural history of the particular problem being treated, regression to the mean, and the expectation of something being done to the area in question. This can lead to the fallacy known as *post hoc ergo propter hoc* (after this, therefore because of this), when the treatment offered in fact had nothing to do with the pathogenesis of the condition towards which it was directed. A recent study comparing dry needling with manual compression, in which there was no control group, exemplifies this critical methodological issue [86].

One common factor shared by most therapies is that they elicit pain at the site of their application; that is, they are noxious stimuli. If they do work, this similarity suggests a common mechanism of action. One possible mechanism is counterirritation, or application of a competing noxious stimulus [87, 88]. It is not surprising that a noxious stimulus applied in the region where pain is experienced, whether or not there is local pathology present at that site, would elicit a transient reduction in pain intensity by recruiting those higher order brain regions responsible for anti-nociception [89, 90]. In conclusion, the vast majority of studies and meta-analyses do not support the prediction from MPS theory that focal treatment of TrPs is effective.

An impasse

In 1976, Simons hoped that: ‘It would now appear possible to resolve much of the conflicting data of the past by carefully distinguishing trigger from reference zones, and acute from chronic lesions using modern electrodiagnostic, biochemical, histochemical, and ultramicroscopic techniques’ [1]. Some three decades later, he conceded

that acceptance of the concept of TrPs had been hampered by two outstanding considerations: the lack of a diagnostic gold standard and the lack of generally recognized pathogenesis [91].

We propose that sufficient research has been performed to allow TrP theories to be discarded. The scientific literature shows not only that diagnosis of the pathognomonic feature of MPS (the TrP) is unreliable, but also that treatment directed to the putative TrP elicits a response that is indistinguishable from the placebo effect. As these conclusions refute MPS, formulating a plausible scientific explanation for pain perceived by patients as coming from their muscles remains a challenge.

Towards explaining the clinical phenomena

In our opinion, current neuroscientific hypotheses can form the basis for collaborative scientific investigation to explain the clinical phenomena. We offer two for consideration, neither of which relies on local pathophysiology.

Neuritis model

Nerve inflammation as a source of pain was discussed in the 19th century [92–97], but focused research on nerve inflammation as a primary disease aetiology has been limited.

Quintner and Cohen [25] hypothesized that the TrP was an area of what was then called secondary hyperalgesia occurring in muscles that are structurally and physiologically unimpaired. Noting the remarkable proximity of TrPs to known peripheral nerves, these authors argued that sensitization of the axons within the nerves, possibly by inflammation, may inform the underlying mechanism. Subsequent research has emerged in support of this hypothesis.

Focal inflammation of peripheral nerves leads to ectopic axonal mechanical sensitivity and spontaneous discharge of some but not all of the nociceptors within the inflamed nerve [98–101]. These changes can be expected to lead to focal areas of neurogenic inflammation and possibly to sensitization in the muscle innervated. If confirmed, they can inform further investigation that might be highly relevant to explaining the phenomenon of chronic muscle pain.

Referred pain and tenderness (allodynia)

Kellgren [102–104] reported the critical observation that, in addition to referred pain, referred tenderness could be induced by targeted injections of hypertonic saline into tissues such as interspinous ligaments, periosteum, cancellous bone, or voluntary muscle. His studies and those of others [105, 106] showed that nociception in deep tissues can induce the phenomena of remote localized pain and tenderness. This relegates the TrP to being a site of secondary allodynia reflecting altered central nociceptive mechanisms [107].

Conclusion

The construct of MPS caused by TrPs remains conjecture. All working hypotheses derived from this conjecture have been refuted and therefore the theory can be discarded. In contrast, evolving insights into the neurobiology of nociception and pain suggest plausible hypotheses that form a basis for advancing knowledge and therapeutics in this challenging area.

Disclosure statement: The authors have declared no conflicts of interest.

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