[REVIEW]

MYOFASCIAL TRIGGER POINTS: FROM THEORY TO PRACTICE

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Introduction: Travell defines the myofascial trigger point (MTrP) as a hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. The spot is painful on compression and can give rise to characteristic referred pain, referred tenderness, motor dysfunction and autonomic phenomena.

Objective: To assess the role that MTrPs can play in osteopathy as potential sources of pain, to describe their clinical characteristics in order to correctly diagnose them and to underline the importance of treating them within an osteopathic treatment protocol.

Materials and Methods: We carried out a literary review, followed by a discussion of the topic.

Results: MTrPs, despite having a high prevalence, are often forgotten or poorly treated due to the fact that the early training given to medical professionals rarely includes sufficient information to identify and treat MTrPs. There is good inter-rater concordance in detecting the presence or absence of either latent or active MTrPs. This makes diagnosis more reliable. The literature shows that the reliability of the identification of the clinical characteristics of MTrPs depends on the specific characteristic and muscle being studied. Clinical experience is essential for obtaining good results.

Conclusions: As MTrPs are potential sources of pain and noxious afferents, osteopaths should be able to correctly diagnose and deactivate them as soon as possible to avoid the central sensitisation of the nervous system. It is essential that osteopaths are trained to correctly identify MTrPs. A taut band (TB) and local sensitivity are the most reliable clinical signs for diagnosing MTrPs. The biggest obstacle preventing reliable diagnosis of MTrPs is the general lack of consensus surrounding the best diagnosis criteria to use.

KEY WORDS

› Trigger points.
› Diagnosis.
› Referred pain.

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INTRODUCTION

Travell defines the myofascial trigger point (MTrP) as a hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. The spot is painful on compression and can give rise to characteristic referred pain, referred tenderness, motor dysfunction and autonomic phenomena[1]. MTrPs are incredibly common[2]. In a study on the prevalence of MTrPs, it was found that all subjects with unilateral, chronic, non-traumatic pain in the shoulder presented with multiple MTrPs in the shoulder muscles.

MTrPs, despite having a high prevalence, are often forgotten or poorly treated due to the fact that the early training given to medical professionals rarely includes sufficient information to identify and treat them[2].

OBJECTIVES OF THE REVIEW

To assess the role that MTrPs can play in osteopathy as potential sources of pain, to describe their clinical characteristics in order to correctly diagnose them and to study the treatment of MTrPs within an osteopathic treatment protocol.

MATERIAL AND METHODS

Research Strategy

A literature search was carried out between July 2013 and January 2015. The following terms were used for the search: trigger point (MeSH 2012), diagnosis (MeSH 1966), reliability (word anywhere in text). The following databases were consulted: Medline, PubMed and Cochrane, as well as the Google search engine. A manual search was also carried out in the full text articles, to identify additional relevant studies. Any texts containing information that was deemed relevant were included.

Selection criteria

The inclusion criteria for the literature review consisted of articles that contained the following terms: Trigger Points, Diagnosis (included in MeSH), written in Spanish, English or Portuguese. Any articles that that were not related to either the topic or the objectives of this review were excluded as well as any not carried out on human beings.

RESULTS

Etiology

There have been several proposals as to the histopathological mechanisms involved in the development of MTrPs and their pain patterns, but the scientific evidence is insufficient. Many researchers agree that acute trauma or repeat microtraumas could lead to the development of MTrPs. Lack of exercise, prolonged poor posture, vitamin deficiencies, irregular sleep patterns and joint problems combined can lead to the development of a microtrauma[4].

The electromyographical study carried out by Hubbard revealed the existence of spontaneous electrical activity in MTrPs, while the non-sensitive muscle tissue (with no MTrPs) in the same muscle was electrically silent. The author realised that MTrPs are produced by intrafusal fibres contracted by sympathetic activation[5]. These interesting discoveries led Travell and Simons to point to dysfunctional motor end plates as being the underlying cause of MTrPs. The terms "motor end plates" and neuromuscular junction" are interchangable, although the former describes the structure, and the latter reflects the function. Both terms refer to the point where α-motor neurons contact their target muscle fibres[6]. Acute or chronic muscle strain triggers the excessive release of acetylcholine, which creates a local energy crisis, which appears to explain the clinical characteristics of MTrPs[2].

Diagnosis

Distinction between active and latent MTrPs

MTrPs are divided into active MTrPs (AMTrPs) and latent MTrPs (LMTrPs). Active TrPs produce a clinical complaint (usually pain) that the patient recognises when the TrP is digitally compressed[1,7] Compression should be maintained for 10-15 seconds, until you are able to identify whether there is any referred pain[8]. Although latent TrPs can produce the other effects characteristic of a TrP such as increased muscle tension and muscle shortening, they do not produce spontaneous pain, as in the case of AMTrPs. Both AMTrPs and LMTrPs can cause significant motor dysfunction[1].

Patients with active myofascial TrPs usually complain of poorly localised, regional, aching pain in subcutaneous tissues, including muscles and joints. They rarely complain of sharp, clearly-localised cutaneous-type pain. Application of digital pressure on a MTrP can elicit a referred pain pa-

ttern characteristic of that muscle. However, if the patient "recognises" the elicited sensation as a familiar experience, this establishes the TrP as being active and is one of the most important diagnostic criteria available when the palpable findings are also present. The myofascial pain is often referred to a distance from the TrP in a pattern that is characteristic for each muscle. Sometimes, the patient is aware of numbness or paresthesia rather than pain[1,7].

Shah et al found significantly higher concentrations of hydrogen ions [H+] (indicative of a more acidic pH level), bradykinin, neuropeptides (Substance P and calcitonin gene-related peptides), cytosine (tumour necrosis factor-α, interleukin-1β) and neurotransmitters (serotonin or 5-HT and norepinephrine in active MTrPs than latent MTrPs, which appears to justify the elevated hyperalgesia observed in active MTrPs[9]. In another study, the same author verified that subjects with active MTrPs in the upper trapezius muscle also presented with higher levels of these biochemical substances in a remote, unaffected muscle, which suggests that these conditions are not limited to local areas of active MTrPs[10].

As muscle stress decreases, TrPs can decrease in number and can diminish in activity. On the other hand, as the level of stress or activity increases, taut bands increase in number and "irritability," and become tender to palpation, or spontaneously painful with activity. A very active TrP will be painful at rest. Thus, the boundary between latent and active TrPs is actually very fluid and dynamic, and TrPs will increase and decrease the amount of pain they produce, depending on the demands placed on the muscle and its ability to meet those demands. The identification of all of the active MTrPs is mandatory, because if only one of them is overlooked the persistence of a certain amount of pain is inevitable. It is therefore necessary to locate MTrPs not only in the primarily affected muscles, but also in their synergists and antagonists (secondary MTrPs). Guidance as to where to look for these MTrPs may be obtained from carefully noting the distribution of pain and by observing which movements are restricted as a result of it[8].

Clinical characteristics of MTrPs

Sensory aspects may include local tenderness, referral of pain to a distant site, and peripheral and central sensitization. Peripheral sensitization can be described as a reduction in threshold and an increase in responsiveness of the peripheral ends of nociceptors, while central sensitization is an increase in the excitability of neurons within the central nervous system. Signs of peripheral and central sensitization are allodynia (pain due to a stimulus that does not normally provoke pain) and hyperalgesia (an increased response to a stimulus that is normally painful). Both active and latent MTrPs are painful on compression[12]. Analysing the local sensitivity and referred pain of a myofascial TrP produced increased inter-rater agreement, as these were the most reliable clinical signs for the diagnosis process for MTrPs[7]. An experienced physiotherapist can reliably identify MTrP locations using a palpation protocol that includes use of the pads of the fingers to locate local pain[13,14.] As well as being the simplest way to identifying MTrPs, clinically speaking, muscle palpation is also the most commonly used method. You can use flat palpation, pincer palpation or both, depending on the muscle you are examining. The best technique to use for the sternocleidomastoid and latissimus dorsi muscles is pincer palpation with the thumb and index finger. However, the infraspinatus and extensor digitorum muscles can only be examined using flat palpation. The trapezius muscle can easily be examined using either technique. The amount of pressure applied by the examiner should vary for each muscle and each different subject[1,7]. You have to be very firm (a pressure of approximately 4kg), otherwise the characteristic reactions of an active or latent MTrP such as a jump sign (withdrawal response) and pain (reported by the patient) will not occur. One of the most common reasons for MTrPs being overlooked is that palpation has been carried out too gently[8]. As of today, no study has managed to report the reliability of diagnosing MTrPs in symptomatic patients in accordance with the latest proposed criteria. Particularly prejudicial to MTrP theory, is the lack of any data on the reliability of pinpointing the exact location of active MTrPs[16]

By gently rubbing across the direction of the muscle fibres in a superficial muscle, the examiner can feel a nodule at the MTrP and a rope-like induration that extends from this nodule to the attachment of the taut muscle fibres at each end of the muscle, no doubt very similar to myalgic cords in osteopathy, corresponding to myotome disorders. The increased tension of the palpable taut band is the result of regional shortening of the sarcomeres of numerous involved muscle fibres in the taut band[2.] Identifying the taut band led to good inter-rater agreement, as it is very high in muscles such as the trapezius, sternocleidomastoid and extensor digitorum[7]. Chen verified that taut bands are detectable and quantifiable with MRE imaging. The findings in the subjects suggest that the stiffness of the taut bands in patients with myofascial pain may be 50% greater than that of the surrounding muscle tissue[17]. When

an MTrP is stimulated mechanically, either through needle penetration or snapping palpation, the taut band will contract, producing a local twitch response. Mechanical stimulation of an MTrP can also trigger referred pain, which occurs at a distance from the point of stimulation. The area in which the pain is felt can be local, in the same muscle or adjacent to it or remote (referred pain). Palpation of the MTrP reproduces or increases the spontaneous pain of an active MTrP. Range of motion is reduced because of the taut band and the pain. Muscle weakness occurs, but no atrophy and autonomic phenomena can occur with stimulation of the MTrP in certain muscles, such as the sternocleidomastoid muscle[1,7]. Identification of a local twitch response can be visual or tactile; it is barely palpable by the examiner. Both forms of identification are valid, although of all the clinical characteristics of MTrPs, local twitch response is the least reliable in the majority of muscles. In muscles such as the sternocleidomastoid and infraspinatus, local twitch response is very unreliable[7]. A local twitch response is accompanied by a local chemical alteration. This can be seen when a needle is inserted into the MTrP, after a local twitch response, and the concentration of substance P calcitonin gene-related peptides is reduced[9]. These findings support the reduction of pain and sensitivity observed clinically when the MTrPs were released, after penetration. Changes in analyte levels after a local twitch response might result from increasing local blood flow to the MTrP region, leading to a "wash out" of the pain and inflammatory mediators[10].

Muscles with MTrPs present with a limited passive range of motion (stretching) because of the pain as well as reduced force / resistance. Although muscle weakness is a common characteristic of a muscle with an active MTrP, the level of weakness varies from one muscle and subject to another[1]. Limited range of motion in a muscle while stretching and increased palpable muscle tension (reduced distensibility) are most accentuated in the most active MTrPs[7,18]. Trying to passively stretch the muscle beyond this limit will trigger severe pain as the muscle fibres involved will be tense, even while resting[1].

Diagnostic criteria

A lack of consensus surrounding the most appropriate diagnosis criteria to use to examine MTrPs has greatly impeded the development of a general assessment protocol for MTrPs and prevents us from being able to compare studies on the effectiveness of treating them[1,8].

Simons and Travell established the following essential criteria for diagnosing MTrPs: the presence of a palpable taut band (if the muscle is accessible), the presence of a hypersensitive nodule in the muscle, the patient recognising the pain when the sensitive nodule is palpated (which identifies an active MTrP) and pain and limited range of motion of the muscle when stretched. A local twitch response or pain in the distribution expected from a trigger point in the muscle in question when compressed are considered to be confirmatory observations[1].

Treatment

Clinicians have recognized for more than a century that effective treatment of painful, tense, tender muscles includes stretching the involved muscle fibres, either locally in the region of tenderness (massage) or by lengthening the muscle as a whole. Frequently MTrPs were the cause of the symptoms and were what was being treated[2]. For the successful management of myofascial trigger point (MTrP) pain it is essential to first identify all of the MTrPs from which the pain is emanating, and to deactivate them by one or other of several methods currently employed. Following this, measures should be adopted as necessary to prevent reactivation of the MTrPs. In addition, treatment should be started as early as possible, before pain-perpetuating changes take place, in particular spinal cord neuroplasticity (central sensitisation)[8]. Any treatment that reduces the sarcomere shortening in the region of the MTrP reduces the energy consumption, which in turn reduces the release of sensitizing substances. The degree to which the released sensitizing substances get to actually sensitize nociceptors depends strongly on the closeness of the nociceptors to an affected motor end plate and that depends on variations in local anatomical structure. Generally the end plate and nociceptors are near each other, but not always. This is why MTrPs are primarily a motor dysfunction disease and only secondarily a pain phenomenon. The pain results secondarily from the end plate motor dysfunction. This helps to explain why there is only a general correlation between the motor expression of a MTrP (the taut band) and its degree of painfulness and why latent MTrPs are so much more common than active ones[2].

More than twenty years ago, Travell and Simons described the technique "ischemic compression", used to treat MTrPs, applying firm pressure to the MTrP with the thumb[1]. In the 1999 edition of their work, Travell and Simons recommend applying gentle digital pressure on the MTrPs as firm pressure could create additional ischaemia, which could be harmful. This new myofascial trigger point pressure release

technique seeks to restore the shortened sarcomere to its normal length in the contracted nodule. Other techniques associated with osteopathy such as the muscle energy technique, counterstrain and fascial release are also mentioned[1]. Other methods of deactivating MTrPs found in the literature include: ischemic compression, trigger point pressure release[19,] transverse friction massage[20], a lidocaine 1% injection[21] and superficial or deep dry needling[22,23]. After MTrP inactivation, muscle stretching exercises are instructed as appropriate. This therapy is accompanied with a gradual increase in daily activities[3].

The osteopath must take all necessary measures should to prevent reactivation of the MTrPs, by correcting posture disorders and imbalanced leg length. It is also essential to underline the importance of teaching post-deactivation muscle stretching exercises. The patient should be taught to identify the activities that increase the pain in order to avoid those activities[22,24].

DISCUSSION AND CONCLUSIONS

The primary objective of this review was to show the importance of MTrPs in osteopathy as potential sources of pain, to describe their clinical characteristics in order to correctly diagnose them and to underline the importance of treating them within an osteopathic treatment protocol. Palpation continues to be the simplest method of diagnosing MTrPs and so increased training for osteopaths is recommended. Palpation allows the osteopath to assess local sensitivity and referred pain from an MTrP, as well as to identify the taut band and clinical signs with increased inter-rater agreement as these signs are deemed reliable for the diagnosis process. The taut band and local sensitivity are the most reliable clinical signs for MTrPs and constitute the minimum criteria for diagnosis. A latent MTrP should have a sensitive point and a taut band, and is differentiated from active MTrPs by the apparition of pain. The presence of a local twitch response or referred pain increases the reliability of a diagnosis of MTrPs and so are useful as confirmatory signs of MTrPs[7].

Palpation is still the main tool used to identify MTrPs and so osteopathic training is essential for a correct diagnosis.

As MTrPs are potential sources of pain and noxious afferents, osteopaths should be able to correctly diagnose and deactivate them as soon as possible to avoid the central sensitisation of the nervous system. A lack of consensus surrounding the most appropriate diagnosis criteria to use to examine MTrPs has greatly impeded the development of a general assessment protocol for MTrPs and prevents us from being able to compare studies on the effectiveness of treating them.

BIBLIOGRAPHICAL REFERENCES

1. Simons DG, Travell JG, Simons LS. *Myofascial pain and dysfunction. The trigger point manual. Upper half of the body. Volume 1*. 2nd ed. Baltimore: Williams & Wilkins; 1999.

2. Simons DG. *Understanding effective treatments of myofascial trigger points*. J Bodyw Mov Ther. 2002;6(2):81-88.

3. Bron C, Dommerholt J, Stegenga B, Wensing M, Oostendorp R. *High prevalence of shoulder girdle muscles with myofascial trigger points in patients with shoulder pain*. BMC Musculoskelet Disord. 2011; 12:139.

4. Han SC, Harrison P. *Myofascial pain syndrome and trigger-point management*. Reg Anesth. 1997;22(1):89-101.

5. Hubbard DR, Berkoff GM. *Myofascial trigger points show spontaneous needle EMG activity*. Spine. 1993;18(13):1803-7.

6. McPartland JM. *Travell trigger points–molecular and osteopathic perspectives*. J Am Osteopathic Assoc. 2004; 104(6):244-249.

7. Gerwin RD, Shannon S, Hong CZ, Hubbard D, Gevirtz R. *Interrater reliability in myofascial trigger point examination*. Pain. 1997;69:65-73.

8. Baldry P. *Management of myofascial trigger point pain*. Acupunct Med. 2002;20(1):2-10.

9. Shah JP, Phillips TM, Danoff JV, Gerber LH. *An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle*. J Appl Physiol. 2005;99(5):1977-1984.

10. Shah JP, Gilliams EA. *Uncovering the biochemical milieu of myofascial trigger points using in vivo microdialysis: an application of muscle pain concepts to myofascial pain syndrome*. J Bodyw Mov Ther. 2008;12:371-384.

11. Gerwin RD. *Classification, epidemiology, and natural history of myofascial pain syndrome*. Curr Pain Headache Rep. 2001;5:412-420.

12. Dommerholt J, Bron C, Franssen J. *Myofascial trigger points: an evidence-informed review*. J Man Manip Ther. 2006:14(4): 203-221.

13. Barbero M, Bertoli P, Cescon C, Macmillan F, Coutts F, Gatti R. *Intra-rater reliability of an experienced physiotherapist in locating myofascial trigger points in upper trapezius muscle*. J Man Manip Ther. 2012; 20(4):171-177.

14. Bron C, Franssen J, Wensing M, Oostendorp R. *Interrater reliability of palpation of myofascial trigger points in three shoulder muscles*. J Man Manip Ther. 2007; 15(4):203-215.

15. Sciotti VM, Mittak VL, DiMarco L, Ford LM, Plezbert J, Santipadri E et al. *Clinical precision of myofascial trigger point location in the trapezius muscle*. Pain. 2001;93(3):259-66.

16. Lucas N, Macaskill P, Irwig L, Moran R, Bogduk N. *Reliability of physical examination for diagnosis of myofascial trigger points: A systematic review of the literature*. Clin J Pain. 2009;25(1):80-89.

17. Chen Q, Bensamoun S, Basford JR, Thompson JM, An KN. *Identification and quantification of myofascial taut bands with magnetic resonance elastography*. Arch Phys Med Rehabil. 2007;88(12):1658-61.

18. Macdonald AJ. *Abnormally tender muscle regions and associated painful movements*. Pain. 1980;8(2):197-205.

19. Gemmell H, Miller P, Nordstrom H. *Immediate effect of ischaemic compression and trigger point pressure release on neck pain and upper trapezius trigger points: A randomised controlled trial*. Clinical Chiropractic. 2008;11:30-36.

20. Fernández de las Peñas C, Carnero JF. *Masaje de fricción transversal. Una alternativa terapéutica para el tratamiento del síndrome de dolor miofascial*. Fisioterapia. 2004;26(3):126-33.

21. Balbino LF, Vieira LR. *Avaliação objetiva da síndrome dolorosa miofascial: uso da termografia antes e após tratamento associando mesoterapia a bloqueio anestésico*. ACTA FISIATR. 2005;12(3):115-117.

22. Baldry P. *Superficial versus deep dry needling*. Acupuncture Med. 2002;20(2-3):78-81.

23. Dommerholt J, Moral OM, Gröbli C. *Trigger Point Dry Needling*. J Man Manip Ther. 2006;14(4):70-87.

24. Gerwin RD. *The management of myofascial pain syndromes*. J Musculoskel Pain. 1993;1(3-4):83-94.

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