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Controversy Corner

Referred Pain of Peripheral Nerve Origin: An Alternative to the "Myofascial Pain" Construct

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Abstract: The theory of myofascial pain syndrome (MPS) has been constructed around the trigger point (TrP), a region within a muscle from which local and remote pain can be evoked by palpation. Although their pathophysiology is obscure, TrPs have been regarded as the cause of myofascial pain. Spread and chronicity of pain are attributed to the activation of latent, secondary, and satellite TrPs. Although it lacks internal validity, this tautological concept has given rise to a system of empirical treatment that has been uncritically accepted by many. However, not only does the anatomical distribution of pain referred from TrPs bear a close relationship to the course of peripheral nerves, but the pain of MPS is also similar to nerve trunk pain, which is an example of somatic referred pain. Pain of peripheral nerve origin can be present without neurological deficit and with normal findings on conventional electrodiagnostic examination. In contrast to the theory of MPS, which considers the TrPs to be sites of primary hyperalgesia, this article argues that all MPS phenomena are better explained as secondary hyperalgesia of peripheral neural origin.

Key Words: Myofascial pain syndrome—Neuropathic pain—Hyperalgesia—Epistemology.

Although painful conditions of varying degrees of severity involving the soft tissues (i.e., muscles, tendons, ligaments, and peripheral nerves) occur frequently, their underlying pathogenesis is poorly understood. During the 19th century, these conditions were called *muscular rheumatism* or *fibrositis* to distinguish them from conditions such as *articular rheumatism*, which primarily involve joints (1). Chronic forms of muscular rheumatism were attributed to inflammation of a "peculiar" kind affecting the fibrous tissues around joints; this inflammation was found in tendons, bursae, ligaments, fascia, nerve sheaths, muscles, and periosteum (2). Others only used the term *rheumatism* when they wished to denote the presence of nonspecific inflammation involving voluntary muscle fibers (1,3).

In the early part of this century, the English neurologist Gowers (4) championed the concept of "fibrositis" as a painful inflammatory disorder of the fibrous structure of muscle spindles (at that time the only known sensory structures in muscle). He taught that "fibrositic" inflammation could spread by direct fascial extension to involve nearby tendons, joints, and nerve sheaths (interstitial neuritis), thus unifying the two conceptual models of the

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19th century. By the 1930s his views had gained general acceptance. The committee on "arthritis and allied conditions" appointed by the British Medical Association in 1933 recognized the following subgroups of fibrositis: intramuscular and fascial; periarticular; bursal and tenosynovial; subcutaneous (panniculitis); and perineuritic (5). The clinical sequelae attributed to "perineuritis" included radiating pain, paresthesiae, cutaneous hyperesthesia, tenderness in muscles and joints in the sensory area served by the nerves involved, and tenderness over the site of the nerve itself, which was attributed to the involvement of the nervi nervorum. Motor and sensory impairments were uncommon (5).

Over the next 50 years, clinicians attempting to unravel the complex nature of muscular rheumatism focused their attention on palpatory findings in and around voluntary muscles, almost to the exclusion of a possible contribution to pain from peripheral nerves or other tissues. The concept of the "fibrositic nodule," described as "an area in the substance of a muscle or its tendinous sheath which gives rise to pain either in the same locality or referred to a distance when stimulated" (6), was discarded when it became clear that it lacked pathological support (7). In addition, as knowledge of spinal disc pathology increased, it was argued that so-called fibrositic lesions in muscles could be explained as secondary or referred phenomena (8).

However, those who still adhered to the belief that many localized or regional chronic pain syndromes are attributable to more subtle pathological changes occurring solely within voluntary muscle put forward the construct of the *myofascial pain syndrome* (9). More recently, another, competing, construct has been redefined to account for chronic and widespread musculoskeletal pain, namely *fibromyalgia syndrome* (10). The main diagnostic criterion for this syndrome is the presence of a defined number of "tender points" at predetermined anatomical sites. We have argued elsewhere that this construct conveys no pathophysiological insights, having been derived by a process of circular argument (11). Moreover, some authors have recognized that there may be considerable clinical overlap between the two syndromes (12,13). In view of the controversial and complex nature of these pain syndromes, a critical analysis of the prevailing hypotheses is justified to clarify the situation.

In this article, the hypothesis that pain arising from trigger areas within muscles is of primary myofascial origin is critically examined. It will be shown on epistemological, clinical, and pathophysiological grounds that the myofascial pain syndrome (MPS) construct is invalid and that the phenomena it purports to explain are better understood as secondary hyperalgesia of peripheral neural origin.

THE "MYOFASCIAL PAIN" CONSTRUCT

The major sources for the synthesis provided herein are the principal writings of the proponents of MPS (9,14–19).

Definition and basic phenomenology

Myofascial pain has been described as "the most common cause of chronic pain" (17). Introduced in 1952 after a decade of research, and developed since by Travell and her co-workers (9,15), MPS has been defined as a regional pain syndrome with two major components: (a) the trigger point, a localized area of deep muscle tenderness or hyperirritability; and (b) a predictable, discrete reference zone of deep aching pain, which may be located in the immediate region of or remote from the trigger point (TrP), may be quite extensive, and is worsened by palpation of the TrP.

Trigger points have been described in skin, joint capsules, ligaments, and periosteum as well as in muscles and their fasciae. Myofascial TrPs are said to be located within palpable taut bands, purported to represent shortened muscle fibers. On "snapping" palpation or needling of a myofascial TrP, a local twitch response can be elicited. This clinical sign is accompanied by an irritable electromyographic response. A muscle containing a TrP exhibits antalgic inhibition when tested for strength and is also intolerant to stretch. In a seeming contradiction, muscle stretching is recommended as being efficacious treatment for myofascial pain. Relief of pain requires "inactivation" of the relevant trigger area, by physical (needling or stretch) or chemical (local anesthetic) means.

Travell and Simons (15) insist that the "specific muscle or muscle group that causes the symptoms should be identified." More recently, Simons (19) has defined MPS as "*primarily* a dysfunction of one or more specific muscles" (emphasis added). The constancy of distribution of pain referred from individual muscles is said to enable the clinician to "work backward" and thus locate the TrP(s) responsible for particular pain patterns.

One of our main criticisms of the construct of myofascial pain is that its major proponents have

incorporated their preferred hypothesis of causation within the definition. As will be shown elsewhere in this article, this error in reasoning has limited the discussion of other explanations for the various clinical phenomena observed in these syndromes.

Metaphysics of trigger points

The TrP is said to "cause" (16) or have "the propensity to cause" (18) or "the responsibility for causing" (17) local and referred pain. It has even been suggested that TrPs may at times "refer" hypoesthesia or anesthesia instead of pain (19). Trigger points may be "active," "latent," "satellite," or "secondary." Active TrPs are more likely to be found in musculature of the neck, shoulder, and pelvic girdles and in the muscles of mastication. They can occur in multiple locations in any one muscle; their site(s) can vary from person to person and their irritability is said to vary from hour to hour and from day to day.

A TrP is considered latent or dormant if it is not "causing" referred pain. Latent TrPs can be found in asymptomatic subjects, in whom the TrPs are nonetheless said to restrict movement and cause weakness in the affected muscle (20). Latent TrPs are said to accumulate with advancing age (14).

Satellite TrPs are those that can be found in muscles within the pain-reference zone of another TrP. Secondary TrPs develop in muscles that are either synergists or antagonists of the muscle that contains the primary TrP. Synergists are said to be overloaded when they substitute for the affected muscle and antagonists are said to be overloaded when they counter its tautness.

Initiation

It was originally proposed that myofascial TrPs may be initiated by "direct trauma to muscle or joint, chronic muscular strain, chilling of fatigued muscle, acute myositis, arthritis, nerve root injury, visceral ischemia or dykinesia, and hysteria" (9). These same factors, plus resumption of normal activity after periods of immobility, are also said to be capable of activating latent TrPs. A latent TrP may even be activated during therapy: as one set of muscles is being stretched, their antagonists, which presumably contain the latent TrP, are shortening.

Myofascial pain is now mainly ascribed to an initial insult to muscle fibers, either from macrotrauma or repetitive microtrauma (16). The consequences of such an insult may include release of such substances as histamine, serotonin, kinins, and prostaglandins which may then activate nociceptors and cause reflex muscle contraction.

However, this proposition of muscle injury lacks empirical support. Muscle pain and damage following eccentric contractions have been extensively studied (21). In normal subjects, complete recovery is the rule and no long-lasting effects have been noted. Unless muscle strains are severe (e.g., complete tears) or associated with deep hematoma formation, recovery is complete. Severe distraction or contusion injuries are common in sport but no evidence has been presented that such well-defined acute injuries are antecedents of MPS. Furthermore, electromyography of painful muscles (22) and thermographic studies of the tissues overlying them (23) have not demonstrated abnormalities in TrPs. Muscle biopsy studies of TrPs have also been largely unrewarding in terms of muscle inflammation or damage (18).

Perpetuation

The chronicity of pain that follows the activation of a myofascial TrP has been explained by a feedback cycle maintained by bombardment of the central nervous system (CNS) by impulses from TrPs themselves: that is, they become self-perpetuating. However, remote lesions in joints or chronic visceral disease and dysfunction may also provide noxious input into this cycle, as may emotional stimuli, chronic infection, various metabolic disturbances, and even dietary deficiencies (14).

As the painful muscles in MPS are electrically silent, the presence of muscle spasm that may reflect ectopic impulse formation seems most unlikely (22). Furthermore, the efferent arm of the proposed vicious cycle has been tested. Mense (24) found that γ-motoneuron activity was diminished rather than increased in muscles with carrageenan-induced injury and concluded that the proposed vicious-circle models "have to be considered as working hypotheses rather than explanations of known mechanisms."

Spread

Spread of pain is attributed to latent TrPs being activated or to active myofascial TrPs "metastasizing" to sites within or outside of the pain-reference zone of the original TrP(s) (18). Travell (14) postulated a chain reaction whereby an ever-increasing number of satellite TrPs come into being, causing complex overlapping patterns of pain.

Reliability of TrP phenomena

When blinded as to diagnosis, those expert in the field of MPS were able to detect active TrPs in only 18% of examinations of subjects with a MPS diagnosis (25). In the same study, expert assessments for taut bands and muscle twitch responses were also found to be unreliable. These findings call into question the internal validity of the construct.

Treatment

Inactivation of the TrP by physical and chemical means would be predicted if the TrP is indeed a site of primary hyperalgesia. However, reports of the efficacy of this approach are only anecdotal; inactivation has not been subjected to formal trial. Furthermore, the persistence of using the recommended approach in the face of clinical inefficacy, along with the continuing failure over time to reveal a reasonable anatomical or pathophysiological basis for so doing, is not only irrational but also fails to acknowledge powerful placebo effects (26) and the wider psychosocial context of chronic pain (27).

Objections to MPS construct

The definition of MPS incorporates a preferred hypothesis of causation. This logical error has resulted in a system of diagnosis and treatment that has become popular but remains entirely anecdotal. Moreover, the proposition that myofascial pain and TrPs are intimately related constitutes circular reasoning: that is, by virtue of its form this proposition must always be true (Table 1).

In their efforts to preserve the centrality of the myofascial TrP, myofascial pain theorists have allowed the number and nature of predisposing, precipitating, and perpetuating factors to be open-ended and to encompass the full spectrum of etiology, including the untestable psychogenic level. (16,17). This serves only to perpetuate the circularity of the reasoning.

Perhaps in an attempt to provide external validity, researchers have said that TrPs arise from muscle damage, despite electrical silence and the lack of histological or biochemical evidence. Furthermore, there is neither support from an animal experimental model (24) nor from studies of human muscle injury (21). Trigger points are nonetheless said to be maintained via the CNS, not only by their own activity but also by a legion of processes associated with afferent neural input. Spread of pain is attributed to the activation of latent TrPs or to the metastasis of TrPs. This teleological argument is physiologically unsound.

Taken together, the tenets of the MPS construct arise out of circular reasoning, which should condemn MPS as epistemologically unacceptable.

TABLE 1. *Problems with the MPS hypothesis*

Definition of syndrome incorporates hypothesis of causation.
TrPs lack clinical reliability and validity.
Predisposing, precipitating, and perpetuating factors are legion.
Histological, biochemical, and electrical evidence of primary muscle pathology is lacking.
There is no support for the MPS hypothesis from animal experimental models or human muscle injuries.
Trigger points are an operational concept elevated to the status of theory by circular reasoning.

"MYOFASCIAL PAIN" VERSUS PERIPHERAL NEURAL PAIN

The argument that follows explores a putative relationship between "myofascial pain" and pain of peripheral neural origin. We show that the explanation for peripheral neural involvement in MPS, which depends on nerve compression by "taut bands," is speculative and unconvincing. Application of current concepts of the physiology of nociception can lead to an alternate construct.

Differential diagnosis of MPS

The differential diagnosis of myofascial pain, as proposed (14,16), includes a variety of painful and somewhat loosely defined neurological conditions such as thoracic outlet syndrome (28), radiculopathies, and polyneuropathies. Their differentiation from myofascial pain is said to be facilitated by the presence of accompanying neurological deficits (particularly those matching a peripheral nerve or root distribution) and electrodiagnostic abnormalities (15). Although a fundamental distinction has been made between TrP pain (deep and aching) and pain of peripheral neural origin (prickling, tingling, and numbing), Dalton and Jull (29) were not able to distinguish between somatogenic and neurogenic cervicobrachial pain when they relied solely on the characteristics of pain. Moreover, peripheral neural pain can occur without neurological deficit (30) and without conventional electrodiagnostic abnormality (31).

By contrast, when neurological deficit (often accompanied by electrodiagnostic abnormality) accompanies MPS, it has been ascribed to peripheral nerve entrapment by the taut band containing the

TrP (16,19). The taut band is said to cause an overall shortening of the involved muscle, which then, in turn, can lead to a "secondary" nerve entrapment syndrome (32). The dual propositions that neurogenic mechanisms can activate myofascial TrPs and that myofascial TrPs can cause neurogenic pain add up to a circular argument. Furthermore, the neurological literature does not include the TrP taut band as a recognized anatomical cause of entrapment neuropathy (33,34).

However, on clinical grounds alone, there appears to be an intimate relationship between MPS and defined neuropathology. This relationship is worth exploring in terms of current understanding of nociceptive mechanisms.

Characteristics of myofascial pain

The pain attributed to myofascial TrPs is described as deep, dull, and aching, varying in intensity from mild to severe and occurring either at rest or only on motion (Table 2). These are the characteristics of deep somatic pain. By the 1930s, it had been long known that pain arising in deeply situated joints was often referred to anatomically distant structures. The seminal clinical experiments carried out by Lewis (35) and Kellgren (36) convincingly demonstrated the same phenomenon for pain arising in other deep musculoskeletal tissues, such as muscles, ligaments, and periosteum.

According to Kellgren (36), "The diffuse pain from a given muscle is always distributed within certain regions, though the distribution within these limits varies from individual to individual, and according to the part of the muscle stimulated" and "pain from muscle may be confused with pain arising from other deep structures such as joints and testis."

Some caution is therefore necessary before a mechanically provoked pain response is attributed to a particular structure or structures. Afferents from muscles that are the sites of referred pain and tenderness are the very ones that converge centrally onto spinal neurones that could be involved in processing information from a region of deep damage, thus leading to central summation effects (37).

Vasoconstriction, hypoesthesia, dermographia, and hyperhidrosis have been observed in the skin overlying a region of deep pain. These phenomena appear to be reflexly induced concomitants of somatic referred pain (38).

Peripheral neural pain

The connective tissues of human peripheral nerves are well-innervated. They derive their nerve supply from axons within the nerve and from fibers accompanying the extrinsic vessels that provide its nutrition (39). As well as regulating intraneural microcirculation, this intrinsic nerve system, the nervi nervorum, is thought to have a nociceptive function (40).

Two types of pain, present singly or in combination, have been described in patients with peripheral neuropathy: "nerve trunk pain" and "dysesthetic pain" (41). The former pain has been described as aching, knifelike, or tender, whereas the latter has been described as burning, tingling, searing, crawling, drawing, or electric. Nerve trunk pain is therefore indistinguishable from pain described as myofascial (see Table 2). Nerve trunk pain has been attributed to increased activity in mechanically or chemically sensitized nociceptors within the nerve sheath, while dysesthetic pain has been attributed to damaged nociceptive afferent axons themselves.

TABLE 2. *Comparison of peripheral neural pain with myofascial pain*

Clinical feature	Myofascial pain syndrome	Peripheral neural pain: nerve trunk variety
Pain descriptors	Deep, dull, aching	Deep, aching; can be accompanied by dysesthetic pain
Tenderness		
Local	Trigger points (in muscle): active or latent	Nerve trunk (localized); e.g., site of entrapment
Remote	Trigger points: satellite or secondary	Somatic referred
Associated phenomena	Sympathetic dysfunction; neurological deficit (if nerve is entrapped by taut band)	Sympathetic dysfunction; neurological deficit; neuropathic phenomena, including allodynia and hyperalgesia
Electrodiagnostic abnormality	Usually absent	Usually absent
Therapeutic implications	Desensitization (inactivation) of trigger points	Nerve decompression; treatment of neuropathic pain

Nerve trunk pain characteristically follows the course of the involved nerve, which is found to be tender, whereas dysesthetic pain is felt in its peripheral sensory distribution (41). However, when pain of nerve origin is severe, it can be felt in regions outside the sensory distribution of the particular nerve (33,34).

Peripheral neural pain may be associated with neurological deficit, but it can be accompanied by a hyperesthetic syndrome, which includes both allodynia (pain due to a normally nonpainful stimulus) and hyperalgesia (an increased response to a normally painful stimulus) (42–44). The term *peripheral neuropathic pain* has recently been suggested to embrace the combination of positive and negative symptoms in patients in whom pain is due to pathological changes or dysfunction in peripheral nerves or nerve roots (45).

Pain with the characteristics of "nerve trunk pain" has been described by patients with irritative cervical (46) and lumbar (47) radicular lesions, with brachial neuropathy (40), and following peripheral nerve injury (48).

Most nerve pain syndromes commence with symptoms more in keeping with an irritative than a destructive process (49,50). Local tenderness is commonly found over nerve trunks at sites of entrapment or metabolic insult; this tenderness has been attributed to sensitization of free nerve endings within neural connective tissue (nervi nervorum) (40). Such specific tender points over peripheral nerves, palpation of which could cause distant pain, was reported over a century ago (51). It has recently been suggested that radiating pain and other sensory phenomena could originate from ectopic neural pacemaker nodules formed at a site of entrapment (52). Tenderness has also been noted over motor bands (zone of innervation) and muscles in association with cervical and lumbar radicular pain without gross physical signs of denervation (53). Neuropathic pain states are frequently accompanied by abnormalities in functioning of the sympathetic nervous system (54).

Referred neural pain

Intraneural stimulation of muscle fascicles within the median and ulnar nerve trunks of normal volunteers has been shown to refer pain both distally to muscles within the innervation territories of each nerve, and proximally to deep structures (muscle and bone) in segmentally related regions outside the innervation territory of each nerve (55,56).

Recounting his personal and clinical experience, Ochoa (57) described both local elbow pain and referral of pain into the ipsilateral scapular region following mechanical stimulation of an entrapped ulnar nerve at the elbow. In his own and the other cases, none of the distal symptomatology typical of ulnar neuropathy was present.

Thus, peripheral neural tissue is a rich source of local and potential referred pain.

Anatomical concordance of myofascial TrPs and peripheral nerves

Some TrPs said to be myofascial could be situated in an adjacent hyperalgesic nerve trunk. For example, the discrete upper-limb pain syndromes attributed to TrPs in the middle finger extensor, the extensor carpi radialis, and the supinator muscles can equally be attributed to TrPs in the radial or posterior interosseous nerve trunks. The TrP said to be situated in the pronator teres muscle coincides with the median nerve, and the pain projected therefrom into the thenar muscles follows the course of the median nerve in the forearm. TrPs in the flexores digitorum referring pain into the hand may represent a tender compressed median nerve in the proximal forearm. MPSs in the shoulder girdle region may represent entrapment of the suprascapular nerve, the long thoracic nerve, the axillary nerve, and the dorsal scapular nerve, as the pain-reference zone of the TrPs follow the course of these nerves. In the lower limb, MPSs have been attributed to TrPs close to the sciatic, tibial, and superficial and deep peroneal nerves.

ALTERNATIVE EXPLANATIONS FOR MPS PHENOMENA

Alternative explanations for MPS phenomena are summarized in Table 3.

TrPs as sites of secondary hyperalgesia

The weight of evidence does not support myofascial TrPs as the anatomical sites of pain origin. By contrast, the presence of hyperalgesia in muscles that are structurally and electrically normal suggests that it must be secondary (referred) hyperalgesia (58). This hyperalgesia could be due to peripheral mechanisms such as antidromic activation or sensitization of nociceptive afferents (59) or, more likely, to a state of central sensitization, including spontaneous firing and expansion of the receptive fields of nociceptive dorsal horn neurones (60).

TABLE 3. *Pathophysiological explanations for the phenomena of myofascial pain syndrome*

Phenomenon	MPS theory explanation	Preferred explanation
Hyperalgesia	TrPs (primary hyperalgesia)	Secondary (referred) hyperalgesia
Spread of pain	Activation of latent, satellite, or secondary TrPs; nerve entrapment by taut bands	Sensitization of nervi nervorum; altered central nociception; enlarged receptive fields
Intolerance of muscles to stretch	Contracture of taut band	Reflex spasm secondary to nociception elsewhere, e.g., peripheral nerve
Chronicity	Self-perpetuation; many other factors	Maintenance by nociception elsewhere; central sensitization
Cutaneous correlates		
Hypoesthesia	Nerve entrapment by taut bands	Nerve compression itself
Vasomotor and sudomotor	Reflex efferent phenomena	Reflex efferent phenomena

Spread of pain

Latent, metastasizing, and secondary TrPs lack supporting evidence, as does nerve entrapment by taut bands. The spread of pain is more likely to be the consequence of altered central nociceptive processes and enlarged receptive fields in response to ongoing nociception or ectopic impulse generation (60).

Intolerance of muscle to stretch

The taut bands described in muscles containing TrPs may represent reflex spasm secondary to nociception in structures innervated by the same spinal segment (8). The intolerance to stretch could also be explained as a reflex response to the stretching of adjacent hyperalgesic neural tissue.

Chronicity of pain

MPS theorists attribute chronicity of pain to the self-perpetuating propensity of TrPs, usually in the presence of an assortment of other factors such as a short leg, poor posture, somatoform pain disorder, chronic infection, and secondary gain—all of which are teleological arguments. Alternatively, it has been shown that the altered central processing held responsible for secondary hyperalgesia may be maintained by nociception elsewhere possibly including, of course, peripheral neural structures (61).

Hypoesthesia

There are two explanations for hypoesthesia in MPS theory: compressive neuropathy by a taut band or a referred phenomenon reflecting the downward modulation of receptive fields in the pain-reference zone of the TrP (19). Irrespective of the particular entrapping mechanism, it is accepted that hypoesthesia results from the loss of sensory afferents due to nerve compression at the site of an entrapment (33). However, hypoaesthesia has also been attributed to a functional block occurring at spinal or higher levels associated with a peripheral neural pain state (43).

Vasomotor and sudomotor disturbances

Disturbances of sympathetic efferent function that have been described in association with MPS have also been recognised as reflexly induced accompaniments of neuropathic pain states.

CONCLUSION

The construct of MPS, as proposed to explain chronic, deep, aching, poorly localized pain, not only lacks internal and external validity but also is epistemologically unsound. The emphasis on the primacy of the TrP phenomenon has directed attention away from other possible explanations. By contrast, there are anatomical and physiological grounds to suggest that the phenomenon of the TrP, on which depends the theory of MPS, is better understood as a region of secondary hyperalgesia of peripheral nerve origin. This proposition is testable to achieve external validity for the described clinical phenomena.

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