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Myofascial pain syndromes and their evaluation

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Myofascial pain refers to a specific form of soft-tissue rheumatism that results from irritable foci (trigger points) within skeletal muscles and their ligamentous junctions. It must be distinguished from bursitis, tendonitis, hypermobility syndromes, fibromyalgia and fasciitis. On the other hand it often exists as part of a clinical complex that includes these other soft-tissue conditions, i.e., it is not a diagnosis of exclusion. The clinical science of trigger points can be traced to the pioneering work of Kellgren in the 1930s, with his mapping of myotomal referral patterns of pain resulting from the injection of hypertonic saline into muscle and ligaments. Most muscles have characteristic myotomal patterns of referred pain; this feature forms the basis of the clinical recognition of myofascial trigger points in the form of a tender locus within a taut band of muscle which restricts the full range of motion and refers pain centrifugally when stimulated. Although myofascial pain syndromes have been described in the medical literature for about the last 100 years, it is only recently that scientific studies have revealed objective abnormalities.

Key words: myofascial; pain; myotomal; trigger points; fibromyalgia; taut band; central sensitization.

Myofascial pain is a common form of pain arising from hyperirritable foci in muscle, usually referred to as myofascial trigger points. Few people go through life without experiencing a few episodes of muscle pain. It commonly develops as a result of acute muscle injury, overuse or repetitive strain. Fortunately, the discomfort usually resolves in a few weeks without the need for any medical intervention. When pain persists or worsens, necessitating a medical consultation, it is referred to as a myofascial pain syndrome.^{1–3} Myofascial pain, along with bursitis, tendonitis, hypermobility and fasciitis are the major diagnostic entities that need to be considered in patients presenting with soft-tissue pain problems. Rheumatologists are seldom as well trained in recognizing and managing myofascial pain as they are the other soft-tissue pain problems.

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Considering that muscle is the largest organ in the body, a familiarity with the clinical features of myofascial pain is a useful skill that should be developed by all physicians involved in the management of patients with musculoskeletal pain. Myofascial pain states are not synonymous with the widespread pain of fibromyalgia. However, pain arising from muscle is a potent stimulus for central sensitization, and myofascial pain foci may play a role in the initiation and maintenance of a sensitized nervous system in some fibromyalgia patients.⁴

DEFINITIONS

- *Myofascial pain*: pain arising from muscles or related fascia.
- *Active trigger point*: an active trigger point causes spontaneous pain at rest, with an increase in pain on contraction or stretching of the muscle involved. There is often a restriction of its range of motion. Pain on motion may cause 'pseudo-muscle weakness' due to reflex inhibition (Table I).
- *Latent trigger point*: a latent trigger point is a focal area of tenderness and tightness in a muscle that does not result in spontaneous pain. However, a latent trigger point may restrict range of movement and result in weakness of the muscle involved.

PREVALENCE

It has been estimated that some 44 million Americans have myofascial pain problems.⁵ A study from an internal medicine group practice found that 30% of patients with pain complaints had active myofascial trigger points.⁶ A report from a clinic specializing in head and neck pain reported a myofascial etiology in 55% of cases.⁷ Patients evaluated in one pain management center were found to have a myofascial component to their pain in 95% of cases.⁸ There is increasing awareness that active myofascial trigger points often play a role in the symptoms of patients with tension headaches⁹, low back pain^{10,11}, neck pain¹², temporomandibular pain¹³, forearm and hand pain¹⁴, postural pain¹⁵, and pelvic/urogenital pain syndromes.¹⁶⁻¹⁸

In interpreting the results of prevalence studies, it is important to distinguish between active myofascial trigger points and latent myofascial trigger points. Latent myofascial trigger points are defined as tender areas in muscle, in association with the other clinical features of the trigger point (see Table I), in the absence of associated pain syndrome. Active myofascial trigger points are associated with a pain syndrome that is reproduced by firm palpation of the trigger area. For instance, Sola found latent trigger points in the shoulder girdle muscles of 54% of female and 45% of male subjects who were completely asymptomatic.¹⁹

Table I. The characteristic features of a myofascial trigger point.

1. Focal point of tenderness to palpation of the muscle involved
2. Reproduction of pain complaint by trigger-point palpation (about 3 kg pressure)
3. Palpation reveals an induration of the adjacent muscle (the 'taut band')
4. Restricted range of movement in the muscle involved
5. Often pseudo-weakness of the muscle involved (no atrophy)
6. Often referred pain on continued (~5 sec) pressure over trigger point

There have been no prevalence studies of myofascial pain syndromes in the context of rheumatology practice, but it is the author's experience that myofascial pain problems are often undiagnosed/untreated components of pain in osteoarthritis, rheumatoid arthritis, systemic lupus and other common rheumatic disorders.

THE KEY ELEMENTS IN THE CASE HISTORY SUGGESTING A DIAGNOSIS OF MYOFASCIAL PAIN

The possibility that there may be myofascial syndrome should be considered in any patient in whom a well-defined etiology for their pain cannot be found. The clinical diagnosis of myofascial pain is dependent on the physician being aware of this diagnosis as a possible cause for the patient's pain complaint.³ In a patient with a recent muscle injury or symptoms of repetitive strain the possibility of a myofascial component is self-evident. In a patient with a focal pain complaint that cannot be readily explained by arthritis, bursitis, tendonitis, entrapment neuropathy, radiculopathy or local bone pathology, a careful clinical evaluation for myofascial pain may prove worthwhile. In the current medical climate, especially in the USA, a whole array of often expensive investigations have usually been undertaken before the possibility of a myofascial pain diagnosis is considered. Some patients, who already have a well-defined cause for their musculoskeletal pain (e.g. rheumatoid arthritis), may develop a myofascial pain syndrome that goes unrecognized, as it is assumed that all their pain emanates from their primary diagnosis. Myofascial pain has certain clinical characteristics that aid in considering this diagnosis. The pain is typically described as a deep aching sensation, often with a feeling of stiffness in the involved area; this is sometimes described in terms of joint stiffness. Myofascial pain is aggravated by use of the involved muscle(s), psychological stressors, anxiety, cold, and postural imbalance. Radiation from a trigger point may be described in terms of paresthesiae and thus mimic the symptoms of a cervical or lumbar radiculopathy. Over time muscle weakness, secondary to disuse, may present with the symptoms weakness, poor coordination, reduced work tolerance, fatigue and sleep disturbance. Patients with myofascial pain involving the neck and face muscles have been reported to have the symptoms of dizziness, tinnitus and poor balance.^{1,3}

THE KEY ELEMENTS IN THE PHYSICAL EXAMINATION OF SUSPECTED MYOFASCIAL PAIN

Rheumatologists are well trained in the diagnosis of arthritis, tendonitis and bursitis, but usually lack training in the diagnosis of myofascial pain syndromes. Myofascial pain syndromes may mimic a large number of other disorders²⁰; furthermore, the finding of myofascial trigger points does not rule out other conditions. Thus, a general history and physical examination is, as always, a prerequisite for a competent and informed diagnosis.

Examining for the presence of myofascial trigger points is not part of the standard physical examination, and appropriate training and experience is required for the confident delineation of myofascial pain problems. Considering that skeletal muscle accounts for approximately 40% of body weight and comprises some 400 individual muscles, it is advantageous to have some idea of which muscles could be the site of active trigger points. This search is aided and significantly shortened if the examiner is aware of the characteristic referred pain patterns of specific trigger points.^{21,22}

The defining characteristic of myofascial pain is the finding of a trigger point (Table 1). This is a well-defined point of focal tenderness within a muscle. It is not unusual for trigger point palpation to elicit an involuntary flinching that often seems to be disproportionate to the amount of pressure applied; this is sometimes referred to as the 'jump sign'. Sometimes firm palpation of a trigger point elicits pain in a referred distribution that reproduces the patient's symptoms. Importantly, referred pain from a trigger point does not follow a nerve root distribution (i.e. it is not dermatomal). Rather, it follows a more diffuse distribution referred to as 'myotomal'. In order to elicit referred pain it is often necessary to apply firm pressure over the trigger-point locus for at least 5 seconds. Palpation reveals a rope-like induration of the associated muscle fibers, often referred to as the 'taut band'. Snapping this band, by a sudden transverse pressure perpendicular to the orientation of the muscle fibers, often produces a localized twitch response of the involved muscle. This twitch response can only be reproducibly elicited in fairly superficial muscles. Both the phenomenon of referred pain and local twitch response can be elicited more reproducibly by needling the trigger point.²³ Importantly, trigger points produce functional consequences in terms of a restriction of range of movement and weakness (probably a reflex inhibition secondary to pain) which is usually associated with easy fatigability of the involved muscle.

As latent trigger points are very common in the normal population, an appreciation of the difference between active and latent trigger points is an essential prerequisite before ascribing a patient's pain to a tender area of muscle. Just 'prodding' a muscle and finding it to be tender does not constitute a skilled trigger-point examination.

In order to effectively treat myofascial pain syndromes, it is mandatory to include a thorough evaluation of potential contributory issues and triggers. Factors commonly cited as predisposing to trigger-point formation include deconditioning, poor posture, repetitive mechanical stress, psychological stressors, mechanical imbalance (e.g. leg length inequality), joint disorders, non-restorative sleep and vitamin deficiencies.^{1,2,24}

RELIABILITY OF DIAGNOSIS

There are no well-validated diagnostic criteria for the identification of trigger points. The usual recommendations for identifying a trigger point specify that gentle palpation should be performed across the direction of the muscle fibers in order for the examiner to identify a longitudinal region of nodularity (i.e. the taut band). If the taut band can be 'snapped', a local contraction of the muscle may be observed (i.e. 'the twitch response'). The patient's response to these maneuvers is a critical part of the current diagnostic recommendations for an active trigger point. Firm pressure over the taut band is usually exquisitely painful and reproduces the patient's pain complaint. Continued pressure for >5 seconds may reproduce the pattern of referred pain (see Table 1). In the absence of appropriate pain recognition by the patient, the finding of a tender area in muscle is more likely to be a latent trigger point; this is a very common finding in healthy individuals. As in any aspect of the clinical examination, skill and practice is required in order to perform a competent myofascial trigger-point evaluation. Unfortunately, this skill is not routinely taught in the training of most doctors. An accurate and competent diagnosis of an active trigger point requires both manual palpation skills and appropriate patient feedback. As such, identification of an active trigger point is a diagnosis based on mainly subjective information. Studies of diagnostic reliability between different observers have shown inter-rater reliability to be very dependent on skill and experience. For instance Gerwin et al reported a kappa value of 0.74

amongst four well-trained examiners in identifying specific trigger points in the upper torso.²⁵ On the other hand, Hsieh et al found poor reliability between trained and untrained examiners in the evaluation of trigger points in the lower torso.²⁶

There is little information in the rheumatologic literature on the identification of myofascial trigger points. In one study comparing four rheumatologists, experienced in fibromyalgia, with four physicians experienced in myofascial pain, there was a poor inter-rater reliability. In particular, rheumatologists tended to have more problems in reproducibly identifying the taut bands, muscle twitches and active trigger points (as opposed to latent trigger points).²⁷ In another rheumatology study, three blinded examiners evaluated tenderness (both palpation and algometry) in subjects with fibromyalgia or myofascial pain and healthy controls. There was moderately good reliability in the discrimination between healthy individuals and those with myofascial pain or fibromyalgia, using both palpation and algometry. However there was poor discrimination between diagnoses of fibromyalgia and myofascial pain.²⁸ This result may have been colored by a failure to differentiate active trigger points from latent trigger points.

The most rigorous study to look at myofascial trigger-point locations involved four trained clinicians who pinpointed the location of a latent trigger point in the trapezius muscle of 20 volunteers. Their precise anatomic location of the trigger point was recorded in a blinded fashion using a three-dimensional camera system. Algometry readings were also recorded to assess pain threshold at the trigger-point location. It was found that trained observers can reliably localize a trigger point with a precision that approaches the dimensions of the observer's own fingertips.²⁹

EXPERIMENTAL MUSCLE PAIN

The pioneering studies of experimental myofascial pain were performed by a future rheumatologist, Jonas Henrik Kellgren, when he was a student of Sir Thomas Lewis at University College Hospital, London, UK. Lewis was interested in the precise localization of pain by a skin stimulus, whereas pain from deeper structures was not so accurately localized. In the 1930s it was assumed that most cutaneous pain resulted from a 'neuritis', and Lewis challenged this explanation by experiments using pain-evoking injections of a 6% saline into muscle and skin. Kellgren adopted this technique in the late 1930s to study the referral patterns of muscle and ligament pain throughout the body. To distinguish this type of pain from the better-established dermatomal pattern of referred pain, he used the terms 'myotomal' and 'sclerotomal' pain. His maps of myotomal and sclerotomal referral patterns formed the basis for much of the subsequent clinical research on myofascial pain.³⁰ Many subsequent investigators, in particular the group led by Arendt-Nielsen at Aalborg University, have used injections of hypertonic saline to study human muscle pain.³¹ This work has substantiated, in humans, the concept that pain of muscle origin often results in central neuroplastic changes that account in part for its referral patterns and an intensity that often appears disproportionate to the inciting stimulus.^{32,33} These studies have confirmed, in humans, the earlier animal experiments of Mendell and Wall showing that pain arising from muscle is a more potent stimulus for central sensitization than pain arising from skin.³⁴ Studies of experimentally induced muscle pain have provided a more enlightened understanding of pain in occupational disorders^{35,36}, delayed-onset muscle soreness³⁷, fibromyalgia³⁸, whiplash injury^{39,40}, referred pain and hypersensitivity in osteoarthritis⁴¹, pain and sleep disturbances⁴², and pain and mental stressors.^{43,44}

There is increasing appreciation that myofascial pain is of relevance to the initiation and maintenance of central sensitization in patients with fibromyalgia.^{24,45–47}

HISTOLOGICAL, NEUROPHYSIOLOGICAL AND BIOCHEMICAL FINDINGS

The precise pathophysiological basis for the trigger point phenomenon is still not fully understood, but there is emerging evidence for abnormal neurophysiology and a perturbed biochemical milieu being relevant to the histological finding of 'contracture knots'.

Histology

Myofascial trigger points are thought to arise from focal injury to muscle fibers. Thus the very act of biopsy of living muscle is likely to introduce preparation artifacts. Bearing in mind this caveat, there have been several light-microscopy studies that have reported 'bulging swellings' in focal muscle pain syndromes. In 1951, Glogowski and Wallraff reported finding numerous club-like swollen muscle fibers in patients with 'myogeloses' (a German word describing areas of focal muscle induration).⁴⁸ Miehle and Schulze reported similar findings in the fibrositis syndrome in 1960.⁴⁹ It should be noted¹ that fibrositis/fibromyalgia was not so precisely defined in 1960 and was often used to describe patients with both focal pain syndromes as well as widespread pain. In 1976 Simons and Trolow biopsied the muscles of dogs that had myofascial trigger points based on the clinical findings of a focal tender spot within a taut band. The tender spot was then marked and a widely biopsied under anesthesia.⁵⁰ They found that longitudinal sections revealed densely stained fibers with conspicuous bulges that they referred to as 'contraction knots'. They interpreted these knots as being due to contracted sarcomeres within an individual muscle fiber. In 1996 Windisch et al biopsied the still palpable muscle nodules from fresh cadavers and compared the histology to control areas from the same muscle.⁵¹ They found an overall increase in the average diameter of muscle fibers from the nodules compared to the control areas. On electron microscopy of the nodules there was an excess of A bands and a lack of the I band configuration (*note*: a predominance of A bands with an absence of I bands is the electron microscopic signature of contracted sarcomeres).

A recent study by Mense et al (2003) tested the hypothesis that contraction knots result from an increased acetylcholine (ACh) release from end plates.⁵² A small amount of an acetylcholinesterase inhibitor, diisopropylfluorophosphate (DFP), was injected into the distal half gastrocnemius muscle of rats, which was then electrically stimulated for 30–60 min for induction of muscle twitching. Sections of both the proximal and the distal halves the muscle were then evaluated for morphological changes. The DFP-injected half had significantly higher numbers of abnormally contracted fibers (local contractures) compared to the un-injected half (see [Figure 1](#)). The authors hypothesized that these findings support the notion that dysfunctional end plates, releasing increased quantities of ACh, are relevant to the development of the contraction knots which are thought to be involved in the formation of myofascial trigger points.

David Simons and others envision a myofascial trigger point to be 'a cluster of numerous microscopic foci of sarcomere contraction knots that are scattered throughout the tender nodule' (see [Figure 2](#)).^{2,53} It is thought that these foci result

from a local energy crisis (from injury or repetitive use) that results in contraction of focal sarcomeric units due to calcium release from the sarcoplasmic reticulum.

Neurophysiology

Routine surface electromyography (EMG) of muscles harboring active myofascial trigger points seldom records electrical activity at rest, but tends to show increased

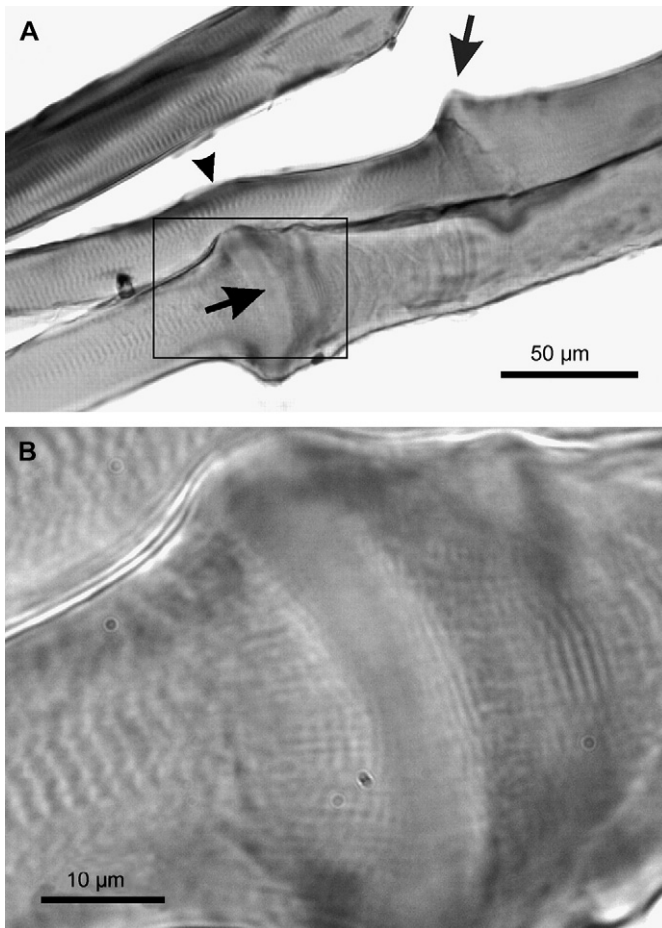


Figure 1. Contraction disks in an area of the muscle where end plates were blocked, as evidenced by a lack of cholinesterase stain. The blocked end plates were located outside the area shown. (A) Contraction disks (arrows) cause marked bulging of the sarcolemma that can impinge on adjacent muscle fibers and distort their sarcomere pattern (arrowhead). The widely spaced curved lines to the right of the lower contraction disk show that the arrangement of sarcomeres, which is normal to the left of the disk, is completely out of register. (B) Enlarged view of the boxed area in (A). Note the abnormally contracted regions flanking the hyaline center of the disk compared with the normal A band spacing seen in the uppermost fiber in (A). From Mense et al (2003, *Journal of Applied Physiology* **94**: 2494–2501) with permission.

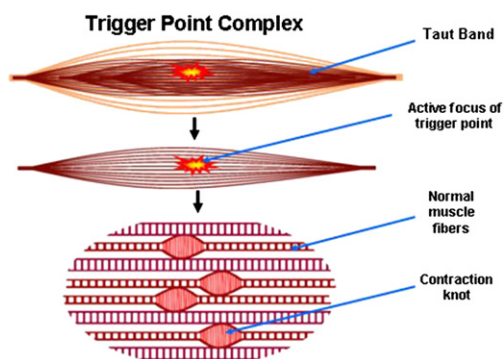


Figure 2. A cartoon of a trigger-point complex seen in a longitudinal section of muscle. The top component represents a muscle with a taut band. The middle component represents a magnified view of the taut band containing an active trigger-point focus. The lower component represents further magnification of the taut band and trigger-point focus showing contraction knots (contracted sarcomere units). It is envisaged that these contraction knots are responsible for the nodularity of the taut band. Adapted from Simons and Travell (1999, *The Trigger Point Manual* Volume 1. Baltimore: Williams and Wilkins) with permission.

motor activity during contraction.¹ However, when a needle examination of an unstimulated trigger point is performed under conditions of high amplification, more spontaneous activity is observed in trigger-point locations than in sites that are outside this location.^{53,54} In recording this spontaneous electrical activity (SEA), the needle has to be very carefully inserted in order to evoke no insertion potentials.² It is currently unclear as to whether SEA represents a specific trigger-point signal, normal end-plate potentials⁵³, muscle spindle activity, or a manifestation of focal dystonia.^{1,55}

When a myofascial trigger point is stimulated there is a burst of electrical activity in the associated muscle fibers of the taut band.⁵⁶ Importantly, there is no generalized contraction of adjacent muscle fibers. The twitch response is a spinal reflex that can be abolished by transection of the spinal nerve that innervates the trigger point.⁵⁷ It can also be abrogated by the infusion of lidocaine into the trigger-point area. Transecting the spinal cord above the level of the segmental innervation of the trigger point has no long-lasting effects on the electromyographic activity elicited by the twitch response.

A particularly revealing study reported on the generation of a mirror image electromyographic activity from the unilateral stimulation of either active trigger points or latent trigger points (trapezius or levator scapulae).⁵⁸ In this study the recording electrodes were inserted ipsilaterally into both the muscle with the trigger point and into the same muscle on the contralateral side. In subjects with *active* trigger points, *bilateral* motor unit activation was observed. In contrast, stimulation of *latent* trigger points was only associated with *ipsilateral* motor unit activation. It was hypothesized that this supports the notion that the perpetuation of pain and muscle dysfunction in active trigger points may be related to abnormal sensory processing at the level of the spinal cord. Furthermore, it provides a possible diagnostic test to distinguish between latent and active myofascial trigger points. However, these results need to be confirmed in a larger cohort of patients with an analysis of sensitivity and specificity.

Biochemical milieu

Over the last few years microanalytical techniques have evolved for measuring the local biochemical milieu of human skeletal muscle.⁵⁹ The basic theory behind this technique is to achieve an equilibrium with muscle tissue by the use of a microdialysis needle perfused with sterile normal saline (see Figure 3). A ground-breaking study performed microdialysis of myofascial trigger points in the trapezius muscle after their location had been verified by eliciting an EMG twitch response.⁶⁰ Compared to normal muscle and latent trigger points, active trigger points showed elevated levels of several biologically relevant molecules: namely tumor necrosis factor α (TNF α), interleukin 1 β (IL-1 β), calcitonin-gene-related polypeptide (CGRP), substance P, bradykinin, serotonin and norepinephrine. Furthermore, the active trigger points had an acidic milieu compared to normal muscle and latent trigger points (see Figure 4).

Several other microdialysis studies have reported on the intramuscular biochemical milieu without localization of active trigger points. In a study of patients with trapezius myalgia, 20 minutes of repetitive low-force exercise resulted in increased concentrations of interstitial potassium ions, but no change in lactate dehydrogenase (LDH) or IL-6.⁶¹

Another study in 19 women with trapezius myalgia and 20 healthy controls reported microdialysis findings after 20 minutes of repetitive low-force exercise.⁶² Resting levels of glutamate and 5HT were increased in the patients with trapezius myalgia. Levels of pyruvate and lactate increased significantly in the muscle pain group with exercise. It was hypothesized that trapezius myalgia is associated with increased anaerobic metabolism and that pain is related to peripheral nociceptive processes. One

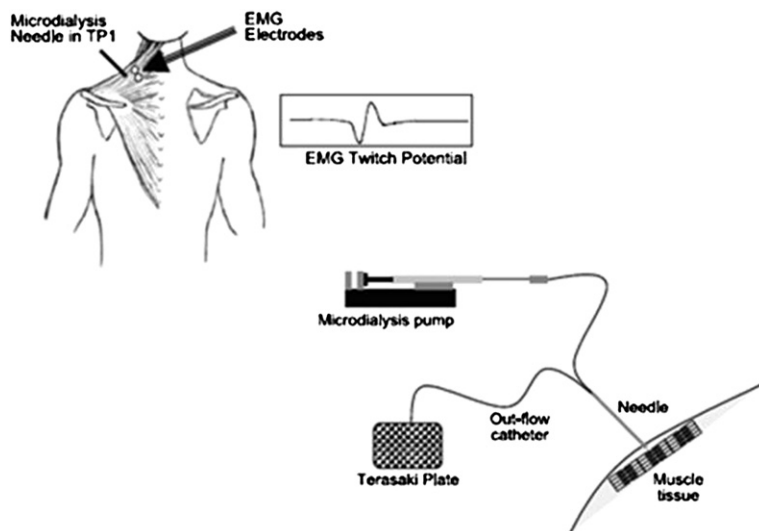


Figure 3. Schematic representation of the microdialysis technique for evaluation of the interstitial milieu of human muscle. Upper cartoon shows needle location in upper trapezius. TPI, trigger point I (TPI). Electromyograph (EMG) potential during local twitch response. Lower cartoon shows microdialysis needle within muscle diffusion pump and collection directly into a Terasaki plate. From Shah et al (2005, *Journal of Applied Physiology* 99: 1977–1984) with permission.

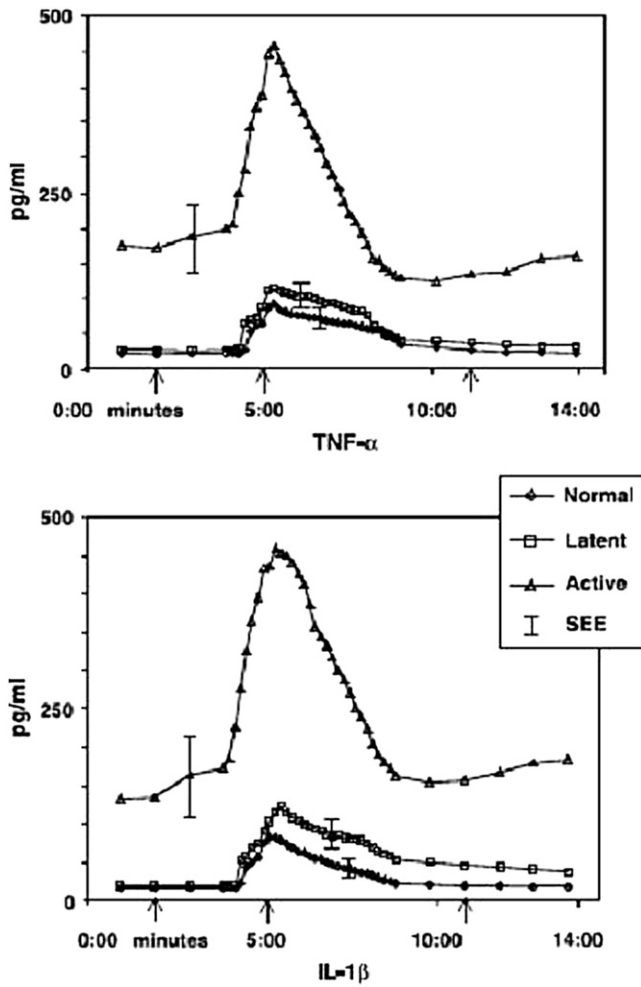


Figure 4. Concentrations of tumor necrosis factor (TNF α) and interleukin (IL-1 β) in the muscle dialysate over time. Comparisons of an active trigger point, a latent trigger point and normal muscle. From Shah et al (2005, *Journal of Applied Physiology* **99**: 1977–1984) with permission.

study reported elevation of CRGP to muscle levels after eccentric exercise⁶³; it was hypothesized that this molecule could be associated with the regulation of delayed-onset muscle soreness.

COMMON CLINICAL SYNDROMES OF MYOFASCIAL PAIN

A myofascial pain syndrome may be due to just one trigger point, but more commonly there are several trigger points responsible for any given regional pain problem. It is not uncommon for the problem to be initiated with a single trigger point, with the subsequent development of satellite trigger points that evolve over time due to the

mechanical imbalance resulting from the reduced range of movement and pseudo-weakness. The persistence of a trigger point may lead to neuroplastic changes at the level of the dorsal horn which results in amplification of the pain sensation (i.e. central sensitization) with a tendency to spread beyond its original boundaries (i.e. expansion of receptive fields).⁶⁴ In some instances segmental central sensitization leads to the phenomenon of mirror image pain (i.e. pain on the opposite side of the body in the same segmental distribution), and in other instances a progressive spread of segmental central sensitization gives rise to the widespread pain that characterizes fibromyalgia.^{38,65,66}

Head and jaw pain

Active myofascial trigger points in the muscles of the shoulder neck and face are a common source of headaches.⁶⁷ In many instances the headache has the features of so-called tension headache, but there is increasing acceptance that myofascial trigger points may initiate classical migraine headaches or be part of a mixed tension/migraine headache complex. For instance sterno-cleido mastoid trigger points refer pain to the anterior face and supraorbital area. Upper trapezius trigger points refer pain to the vertex forehead and temple. Trigger points in the deep cervical muscles of the neck may cause post-occipital and retro-orbital pain (see Figure 5).

There is a complex interrelationship between temporomandibular joint dysfunction and myofascial trigger points.⁷ Common trigger points involved in jaw pain syndromes are the masseters, pterygoids, upper trapezius and upper sterno-cleido mastoid.

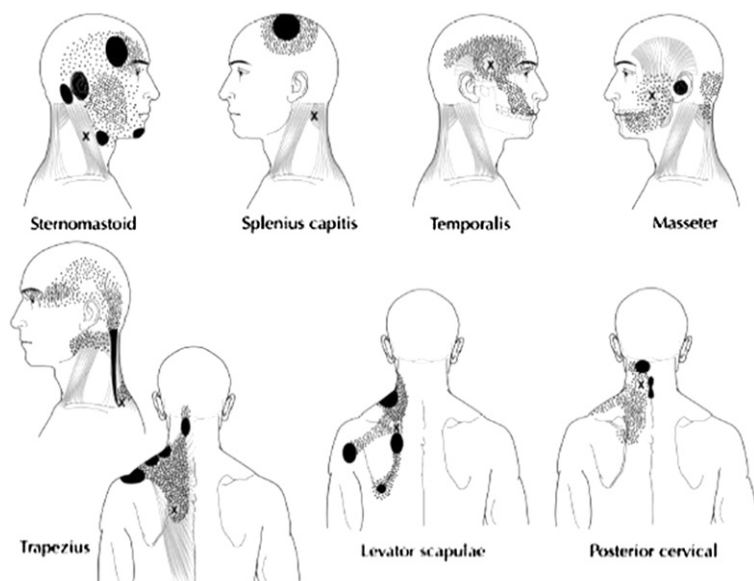


Figure 5. Myofascial pain patterns showing the trigger point (X) and its pain referral pattern (solid black stripping). Adapted from Simons (1984, *Medical Rehabilitation* Basmajian JV, Kirby RL (ed). Baltimore: Williams & Wilkins) with permission.

Low back pain

Acute low back pain has many causes. Some are potentially serious, such as cancer metastases, osteomyelitis, massive disk herniations (e.g. cauda equina syndrome), vertebral fractures, pancreatic cancer and aortic aneurysms. However, the commonest cause of acute back pain is so-called lumbosacral strain. In 95% of cases this resolves within 3 months. In those cases that do not resolve, the development of a chronic low back pain syndrome is usually accompanied by the finding of active myofascial trigger points.¹⁰ Simons describes 15 torso and pelvic muscles which may be involved in low back pain.² The most commonly involved muscle group is the quadratus lumborum (Figure 6); pain emanating from trigger points in these muscles is felt as a band in the low back with occasional radiation in a sciatic distribution or into the testicles. Active quadratus lumborum trigger points often result in difficulties with 'straightening up'. Trigger points involving the iliopsoas are also a common cause of chronic low back pain.⁶⁸ The typical distribution of iliopsoas pain is a vertical band in the low back region and the upper portion of the anterior thigh. Trigger points at the origin of the gluteus medius from the iliac crest are common cause for low back pain in the sacral and buttock with a referral pattern to the outer hip region (see Figure 6).

Neck and shoulder pain

Latent trigger points are a universal finding in many of the muscles of the posterior neck and upper back. Active trigger points commonly involve the upper portion of the trapezius, posterior cervical and suboccipital muscles. Upper trapezius trigger points refer pain to the back of the neck and not uncommonly to the angle of jaw. Levator scapula trigger points cause pain at the angle of the neck and shoulder; this pain is often described as lancinating, especially on active use of this muscle. The deeply located multifidi muscles are difficult to localize by palpation, but have been

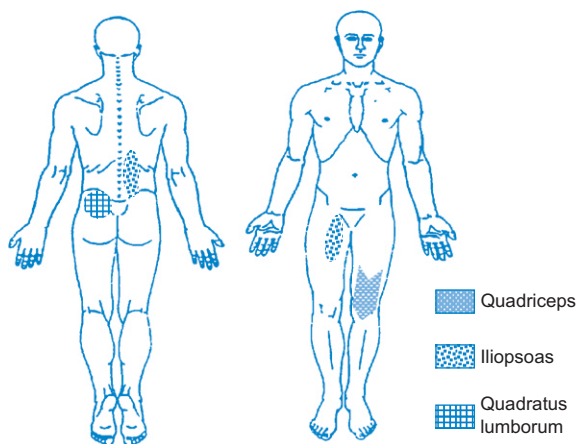


Figure 6. Trigger-point referral patterns from the iliopsoas, quadratus lumborum, and quadriceps muscles that are associated with low back and pelvic region pain.

associated with referred pain in the upper back shoulders and scapula. As many of the muscles in this area have an important postural function they are commonly activated in office workers and developmental problems causing spinal malalignment (e.g. short leg syndrome, hemipelvis and scoliosis). As the upper trapezius and levator scapulae act synergistically with several other muscles in elevation and fixation of the scapula, it is common for a single trigger point in this region to initiating a spread of satellite trigger points through adjacent muscles which are part of the same functional unit.

Hip pain

Pain arising from disorders of the hip joint itself is usually felt in the groin and the lower medial aspect of the anterior thigh. This distribution is uncommon in myofascial pain syndromes, except for iliopsoas pain. The great majority of patients that complain of hip pain in fact localize their pain to the outer aspect of the hip. In some patients this is due to a trochanteric bursitis, but in the majority of cases it is related to myofascial trigger points in the adjacent muscles. By far the commonest trigger points giving rise to outer hip pain are those in the attachments of the gluteus medius and minimus muscles into the greater trochanter (see [Figure 6](#)).

Pelvic pain

The pelvic floor musculature is a common site for myofascial trigger points.^{16–18} There is increasing recognition by gynecologists and urologists that pain syndromes described in terms of prostatitis, coccydynia, vulvodynia and endometriosis are often accompanied by active myofascial trigger points. One of the most commonly involved intra-pelvic muscles is the levator ani; its pain distribution is central low buttock.

Upper limb pain

The muscles attached to the scapula are common sites for trigger points that can cause upper limb pain.⁶⁹ These include the subscapularis, infraspinatus, teres major and serratus anterior. It is not uncommon for trigger points in these locations to refer pain to the wrist, hand and fingers. Extension flexion injuries to the neck often activate a trigger point in the pectoralis minor with a radiating pain down the ulnar side of the arm and into the little finger. Myofascial pain syndromes of the upper limb are often misdiagnosed as frozen shoulder, cervical radiculopathy or thoracic outlet syndrome.²

Lower limb pain

Rheumatologists often overlook a myofascial cause for pain in the knee and ankle. Trigger points in the tensor fascia lata and ilio tibial band may be responsible for lateral thigh pain and lateral knee pain, respectively. Anterior knee pain may result from trigger points in various components of the quadriceps musculature. Posterior knee pain can result from trigger points in the hamstring muscles and popliteus. Trigger points in the anterior tibialis and the peroneus longus muscles may cause pain in the anterior leg and lateral ankle respectively. Myofascial pain syndromes involving these muscles are often associated with ankle injuries or an excessively pronated foot. Sciatica pain may be mimicked by a trigger point in the posterior portion of the gluteus minimus muscle.

Chest and abdominal pain

Disorders affecting intrathoracic and intra-abdominal organs are some of the commonest problems encountered in internal medicine. For instance, anterior chest pain is a frequent cause for emergency room admissions, but in the majority of patients a myocardial infarction is not found. In some cases the chest pain is caused by trigger points in the anterior chest wall muscles.^{22,70,71} Pectoralis major trigger points cause ipsilateral anterior chest pain with radiation down the ulnar side of the arm – thus mimicking cardiac ischemic pain. A trigger point in the sternalis muscle typically causes a deep substernal aching sensation. Trigger points at the upper and lower insertions of the rectus abdominus muscles may mimic the discomfort of gall bladder and bladder infections respectively. It is important to note that myofascial trigger points may accompany disorders of intrathoracic and intra-abdominal viscera, and thus a diagnosis of an isolated myofascial cause for symptoms should never be made without an appropriate work-up.

PROGNOSIS

Uncomplicated myofascial pain syndromes usually resolve with appropriate correction of predisposing factors and myofascial treatment.⁷² If the symptoms are persistent, due to ineffective management, the development of segmental central sensitization may lead to a stubbornly recalcitrant pain disorder. In some such cases, the spread of central sensitization leads to the widespread pain syndrome of fibromyalgia.^{4,73}

TREATMENT

The effective management of myofascial pain syndromes requires attention to the following issues^{72,74}:

Postural and ergonomic factors

The most critical element in the effective management of myofascial pain syndromes is the correction of predisposing factors (see above). These interfere with the ability of the muscle to fully recover and are the commonest reason for treatment failures.

Stretching

The muscles involved in myofascial pain syndromes are shortened due to the aforementioned focal contractions of sarcomeric units. It is thought that these focal contractions result in prolonged ATP consumption, and that the restoration of a muscle to its full stretch length breaks the link between the energy crisis and contraction of sarcomeric units. Effective stretching is most commonly achieved through the technique of spray and stretch.⁷⁴ This involves the cutaneous application, along the axis of the muscle, of ethyl chloride spray while at the same time passively stretching the involved muscle. Other techniques to enhance effective stretching include trigger point to pressure release, post-isometric relaxation, reciprocal inhibition, and deep stroking massage.²

Strengthening

Muscles harboring trigger points usually become weak due to the inhibitory effects of pain. A program of slowly progressive strengthening is essential to restore full function and minimize the risk of recurrence and the perpetuation of satellite trigger points.

Trigger-point injections

Injection of trigger points is generally considered to be the most effective means for their direct inactivation.⁷⁵ A peppering technique using a fine needle to inactivate all the foci within a trigger-point locus is a thought to be a critical element of successful trigger-point therapy (see Figure 7).⁷⁶ Accurate localization of the trigger point is confirmed if a local twitch response is obtained⁷⁷; however this may not be obvious when needling deeply lying muscles. Successful elimination of the trigger point usually results in a relaxation of the taut band. Although dry needling is effective, the use of a local anesthetic (1% lidocaine or 1% procaine) helps to confirm the accuracy of the injection and provides instant gratification for patients.⁷⁸ There is no evidence that the injection of corticosteroids provides any enhanced effect. A beneficial role for botulinum toxin in trigger-point injections has not so far been conclusively demonstrated⁷⁹, but may have a role in treatment-resistant situations.⁸⁰

Medications

Currently there is no evidence that any form of drug treatment eliminates myofascial trigger points.⁷⁴ Non-steroidal anti-inflammatory drugs (NSAIDs) and other analgesics usually provide moderate symptomatic relief. Tricyclic antidepressant drugs, which modulate pain at the central level, are often of benefit, especially in those patients with an associated sleep disturbance. In the author's experience, tizanidine (a muscle

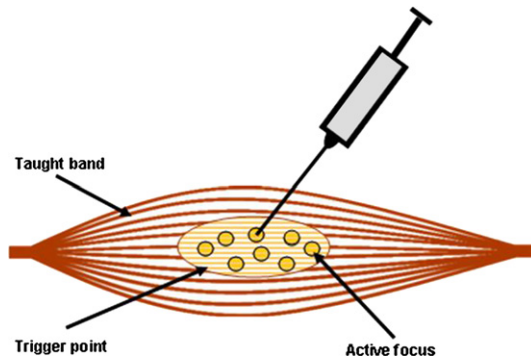


Figure 7. When injecting a trigger point it is useful to envisage several active foci that need to be individually punctured by the advancing needle. To this effect a 'peppering' technique is recommended in which the syringe is held between the thumb and last two fingers, while resting the wrist on the patient's body; the index finger depresses the plunger. Resting the volar part of the wrist on the patient's body provides a measure of safety in case the patient jumps or moves unexpectedly. Adapted from Hong (1994, *Journal of Musculoskeletal Pain* 2: 29–59) with permission.

relaxant which also ameliorates pain by activating α_2 -adrenergic receptors) is often a useful adjunct in difficult-to-treat myofascial pain syndromes.

Psychological techniques

In severe myofascial pain syndromes that are not responding to treatment, it is not unusual for patients to become anxious and depressed. These mood disorders need to be recognized and appropriately treated. Persistent muscle tension exacerbates the pain of myofascial trigger points.^{44,81} In such recalcitrant cases it is often necessary to use additional management techniques, such as EMG biofeedback, cognitive behavioral therapy, and hypnotic/meditation relaxation techniques.

Practice points

- myofascial pain should always be considered in the differential diagnosis of difficult-to-understand pain syndromes
- most healthy people harbor latent trigger points; training is necessary to distinguish these from active myofascial trigger points that are causing pain
- myofascial pain may result in a referred pain pattern that mimics radicular pain
- pain arising from muscle is a potent cause of altered central neuroplasticity, and if untreated it may lead to more widespread pain, including fibromyalgia

Research agenda

- the development and validation of clinical diagnostic criteria for myofascial pain
- the correlation of clinical features of myofascial trigger points with histology, neurophysiology and biochemical changes
- a survey of the prevalence of myofascial pain problems in rheumatology practice
- a long-term follow-up study comparing different management strategies (i.e. stretching, trigger-point injections, physical therapy, botulinum injections, microcurrent therapy etc)

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