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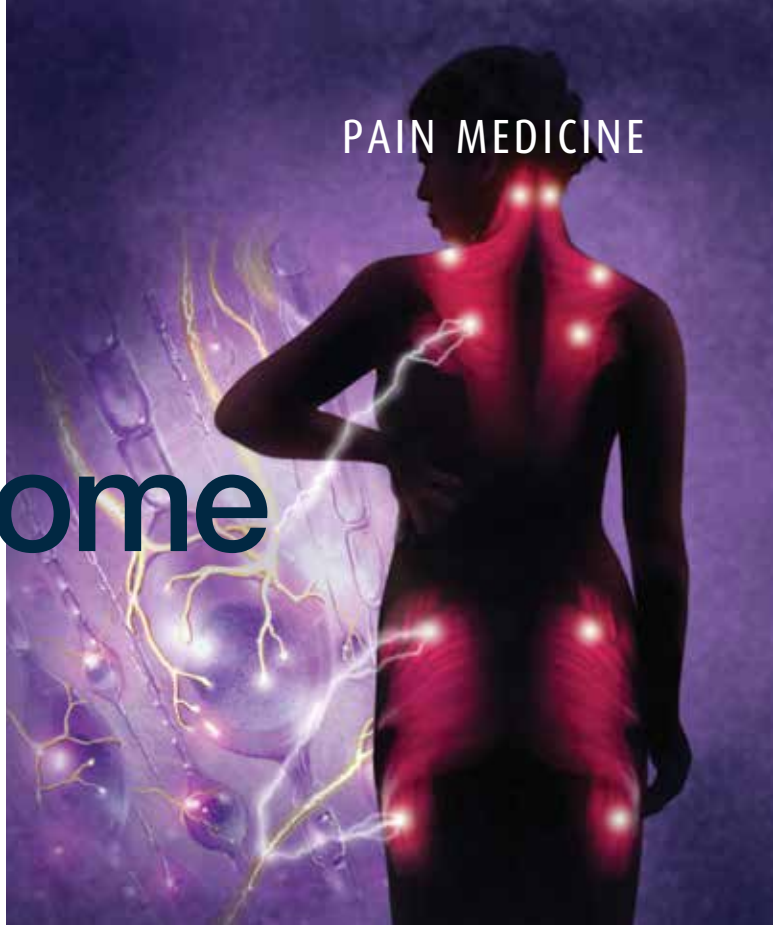

Myofascial pain syndrome

A drug-free perspective

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Myofascial pain syndrome is diagnosed in nearly one-third of patients who have musculoskeletal pain disorders and is characterised by acute or chronic nonspecific pain involving multiple myofascial trigger points. Needle-based interventions and the manual therapies of myofascial manipulation, sustained digital/manual pressure and various massage methods are effective nonpharmacological treatments.

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Myofascial pain syndrome (MPS) is thought to be the leading diagnosis made by pain management specialists and the leading diagnosis in patients with pain presenting to GPs, being diagnosed in nearly one-third of patients who have musculoskeletal pain disorders.[1,2] It is, however, not usually specifically diagnosed in general practice. With increased understanding of the nature and features of its clinical presentation, it is possible for GPs to diagnose myofascial pain early and institute appropriate referral, thus helping prevent the all-too-common development of chronicity associated with the condition.

The mainstay of the treatment of MPS is nonpharmacological but there is a role for pharmacotherapy, including for improving sleep as well as for pain relief. This article focuses on the pathophysiology of MPS and its conservative treatment with needle-based interventions and manual therapies.

WHAT IS MPS?

MPS is a musculoskeletal disorder involving pain in muscles or related fascia and originating from myofascial trigger points. Myofascial trigger points are discrete, focal, hyperirritable spots mostly located in a taut band of skeletal muscle. The characteristic and usually predictable hallmark of myofascial trigger points is that when palpated they give rise to pain locally and/or in a referred area distant from the actual site. A unique feature of myofascial trigger points is the so-called local twitch response (LTR), an involuntary spinal cord reflex contraction of the muscle fibres in a taut band following palpation or needling of the band or trigger point.[3,4,5] Fibromyalgia and MPS have some overlapping features but fibromyalgia is a widespread chronic pain problem whereas

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TABLE. DIFFERENCES BETWEEN MUSCLE PAIN AND CUTANEOUS PAIN

Muscle pain	Cutaneous pain
Poorly localised, aching, pressing, cramping	Well localised, burning, cutting quality
Marked tendency towards referred pain	No tendency towards referral of pain
Affective component harder to tolerate	Affective component easier to tolerate

in MPS the pain is associated with specific trigger points and may be local or referred.

The role of myofascial trigger points in MPS became an accepted part of musculoskeletal clinical practice after the seminal work of Travell and Rinzler in 1952.[6] The importance of myofascial pain was identified by Travell and Simons, who also provided the first classification of diagnostic criteria for trigger points and detailed maps of the pain referral patterns from trigger points.[7]

Myofascial trigger points can be either active or latent: active points provoke a reflection of pain that the patient complains about and describes, and latent points are largely silent until the examiner finds them. Compressing a myofascial trigger point can also produce autonomic phenomena such as visual disturbances, space–perception disturbances, redness and tearing of the eyes, reduction in local vascular activity and skin temperature changes. The implications of such extensive effects are important when examining and treating patients with disorders that are generally not considered to be related to muscular problems.

Common aetiologies of MPS are direct or indirect trauma, pathology in the spine, exposure to cumulative and repetitive strain, postural dysfunction and physical deconditioning. Treating the underlying pathology is the most widely accepted strategy for MPS therapy; if the root cause is not properly treated, myofascial trigger points may reactivate and MPS may persist. However, due to a lack of understanding, clinicians often treat the symptoms (e.g. with medications) rather than the cause.

PATHOPHYSIOLOGY OF MPS

Muscle nociception

Before considering the pathophysiology of MPS it is important to review the fundamental physiology of muscle pain. Chronic pain research provides a theoretical underpinning to the current understanding of MPS.

The role of nociceptive input from muscle in the processes of peripheral and central sensitisation (which are responsible for the transition from normal to abnormal pain perception by the lowering of the activation thresholds of the nerves involved) is well established.[8,9,10] Muscle nociception is more effective than cutaneous nociception at inducing maladaptive neuroplastic changes in the dorsal horn.[11] Such neuroplastic changes support the clinical observation that muscle pain is often difficult to resolve.[12] Furthermore, the unique physiological effects of muscle pain are important when considering the eventual subjective experience of patients experiencing myofascial pain.[12,13,14] This research has demonstrated that muscle pain impairs the descending diffuse inhibitory control mechanisms, meaning that the physiology of muscle pain facilitates the conversion of acute pain to chronic pain.

Another area of research informing our understanding of MPS concerns the specific features of muscle pain.[12,13,15] Since the pioneering work of Melzak and Walls (the gate control theory of pain, 1965), the most common experimental models involving pain research have involved cutaneous and subcutaneous nociceptors.[12,13,16] The studies of Mense established the specific features of muscle pain compared with pain arising from cutaneous and subcutaneous pain receptors.[11] These differences are summarised in the Table. The unique features of muscle pain are well correlated with clinical observations of myofascial pain, which has been described by numerous authors as a dull, deep, cramp-like ache that is hard to localise.[3,6,7,17]

The biochemical milieu of myofascial trigger points

An appreciation of the unique biochemical milieu of myofascial trigger points has been a relatively recent consideration in myofascial pain research. Before Shah's groundbreaking research, most workers in the field conceptualised myofascial trigger points as structural lesions.[12,17,18] The work of Shah and colleagues led to a paradigm shift in the understanding of myofascial pain as they showed that the pathophysiology of myofascial pain is compatible with the current understanding of general pain physiology.[3,4,19]

Shah's study focused on analysis of the biochemical nature of myofascial trigger points. This shift in thinking and the use of in vivo microdialysis enabled the biochemical milieu of myofascial trigger points to be studied in real time, with the result that biochemical differences were shown between muscle containing active myofascial trigger points and muscle with latent or no trigger points.[20] With this technique, Shah was also able to measure the effect of therapeutic techniques such as dry (i.e. noninjection) needling on the biochemistry of trigger points.[8,9,12,14-23] The research is summarised in the Box.

The role of hyaluronic acid

It is well known that the viscoelasticity of fascial tissue is altered in MPS. Hyaluronic acid, a large and simple straight-chain carbohydrate polymer, is located in considerable amounts at the interface between deep fascia and muscle. Hyaluronic acid acts as a lubricant, allowing fascia to glide smoothly between muscles and tendons. It has a considerable charge at neutral pH, and is therefore associated with a large

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volume of solvent water, which can additionally cause pressure on nearby structures.

Chemical alterations such as the acidification that occurs in active myofascial trigger points can cause hyaluronic acid molecules to self-associate, altering their viscoelastic properties. This increase of viscosity in fascial tissue stimulates the receptors within fascia to send pain messages to the brain at amounts of stretching within the physiological range, and is responsible for the stiffness often felt by MPS sufferers.[24-26]

CLINICAL SYMPTOMS

MPS is directly induced by myofascial trigger points, which reveal themselves by the symptoms listed below.

- **Muscle pain.** Most commonly this is a sharp, localised pain that is well demarcated and can be elicited by provocative palpation, with radiating or referred pain, described as a deep, dull ache that is hard to localise. These pain reference zones remain stereotypical between individuals. The referred pain may mimic more traditionally recognised dermatomal, myotomal or sclerotomal referred pain patterns.
- **Muscle weakness.** This is due to pain inhibition but is without atrophy.
- **Autonomic and trophic disorders.** Sympathetic nervous system involvement is common in many chronic pain syndromes, including MPS. Symptoms include increased skin temperature, as well as increased sweat secretion, nausea and dizziness.
- **Neuromuscular entrapment symptoms such as weakness, dysaesthesia or hypoaesthesia.** Because neural structures perforate muscles at many sites, muscle fibres that are tense as a result of a myofascial trigger point may exert pressure on nerves, causing less perfusion to the nerve and resultant clinical symptoms.
- **Psychosocial dysfunction.** As with

MODELS FOR THE ORIGIN OF MYOFASCIAL TRIGGER POINTS

The current model: the neurophysiological model

The research of Shah and colleagues investigated the biochemistry of myofascial trigger points, and showed that myofascial trigger points have a unique biochemical milieu compared with normal muscle.[3,4,19]

The research demonstrated that individuals with active myofascial trigger points (spontaneously painful) had significantly elevated levels of endogenous substance P, calcitonin-related gene peptide, bradykinin, 5-hydroxytryptamine, noradrenaline, tumour necrosis factor alpha and interleukin-1 beta in the local muscle area of the trigger points compared with carefully matched asymptomatic controls (individuals with latent trigger points and with no trigger points).[3,4] These substances are released from and act on muscle, nerve and connective tissue, and are associated with nociceptive sensitisation.

Further studies indicated that individuals with active myofascial trigger points had elevated levels of these inflammatory mediators, neuropeptides, catecholamines and cytokines in remote muscle (referred pain).[3] The presence of inflammatory mediators in active trigger points causes local acidification.

Shah's studies also showed needling of active trigger points with elicitation of the twitch response resulted in the lowering of the concentrations of substance P and calcitonin-related gene peptide to within the normal range observed in the control groups.[3,4] This correlates with the clinical observation that dry needling results in symptomatic relief of pain arising from myofascial trigger points.[8,9,12,19,20,21]

These studies provide objective evidence that discriminate between normal muscle tissue and muscle tissue harbouring trigger points on the basis of the altered biochemistry of trigger points. Shah and colleagues also showed that the effect of treatment such as dry needling on trigger points is to normalise this altered biochemistry.

The energy crisis model

The energy crisis model proposed by Simons preceded Shah's neurophysiological model.[12,26] The energy crisis method provided a metabolic explanation for the pathophysiology of trigger points, focusing on the suggested depletion of ATP leading to contracture of affected muscle, which was then maintained by adaptive changes in the muscle's connective tissue.

This model has significant internal inconsistencies, and these have undermined the acceptance of myofascial pain concepts in the broader medical pain management community.[23]

The models compared

The work of Shah and colleagues supercedes the energy crisis model by focusing on the neurophysiological changes rather than the metabolic changes associated with the development of myofascial trigger points.

Despite the fundamental differences in the two models, both have identified hypoxia in the trigger point region as an important underlying pathophysiological change leading to the development of a trigger point. The neurophysiological model, however, relies on the measurable reduction in the pH of the biochemical milieu of the myofascial trigger point identified by Shah as evidence of hypoxia, whereas the energy crisis model supposes hypoxia without any objective evidence.

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Figure 1. Dry needling of a myofascial trigger point in the shoulder.

many chronic pain conditions, concomitant social, behavioural and psychological disturbances may precede or follow the development of pain. Patients may report psychological symptoms such as frustration, anxiety, depression and anger if acute pain becomes chronic through inadequate treatment. Further to these generalised effects of chronic pain, the specific input that muscle nociception has to the limbic and prefrontal cortex (the areas of the brain associated with the affective components of pain) helps explain the emotional distress often experienced by patients suffering chronic myofascial pain.[11]

DIAGNOSIS

There are no specific diagnostic imaging criteria for MPS and the diagnosis is based on the history and physical examination.

Electrodermal activity measurement, thermography, sonography, echography, dynamic ultrasound and MRI have been investigated for the objective assessment and measurement of MPS, but none has been particularly reliable at providing an objective method of diagnosing trigger points.[27,28] However, promising early further research by Shah and colleagues points to possible future diagnostic imaging techniques.[19,29] This work has focused on using a combination of three diagnostic ultrasound techniques: greyscale (2-dimensional [2D]), vibration sonoelastography and Doppler. These techniques were used experimentally to determine the characteristics of myofascial trigger points in the upper trapezius muscle compared with surrounding soft tissue. On 2D ultrasound, myofascial trigger points appeared as focal hypoechic (darker) areas within a heterogeneous echo texture, and on sonoelastography they appeared as focal regions of reduced vibration amplitude, indicating a localised area of stiffer tissue compared with surrounding soft tissue. Doppler ultrasound showed that myofascial trigger points have a unique vascular environment, with blood flow reversal in diastole in active trigger points indicating that active points have a highly resistant vascular bed. Shah postulated this observation could be due to blood vessel compression caused by local muscle contracture and/or biochemically medicated vasoconstriction of the blood vessels that was brought about by the unique biochemical milieu of myofascial trigger points identified in previous studies.

Although holding promise, these diagnostic techniques for distinguishing active myofascial trigger points from latent trigger points and normal myofascial tissue are not currently available to most clinicians treating myofascial pain. Manual palpation therefore remains the most commonly used method of identifying myofascial trigger points in everyday clinical practice. These trigger points tend to occur in characteristic locations in individual muscles.[22] Diagnosis by palpation is based on three main criteria:

- identifying the taut band belonging to the myofascial trigger point
- finding the most tender spot within the band
- reproducing the pain and other symptoms recognised by the patient on provocation by pressure, traction or needling.

Manual palpation may be seen as an imprecise methodology for diagnosis but its reliability is enhanced when the features of local tenderness and symptom reproduction are the focus of the examination.[23] A recent study confirmed this observation.[23]

CONSERVATIVE TREATMENT OF MPS

The nonpharmacological treatment of MPS is based on an understanding of the underlying pathogenesis of myofascial pain – i.e. an altered biochemical milieu caused by muscle nociceptive activity associated with local hypoxia. It has been shown that local myofascial trigger point stimulation via insertion of a solid filament needle or sustained digital/manual pressure evokes antinociceptive effects by modulating underlying mechanisms, which is an important consideration when managing MPS.[3,4,19]

Needle-based interventions

Clinicians who treat patients with MPS should be aware of the evidence for the effectiveness of needle-based interventions used in the management of MPS and related disorders.

Dry needling is a treatment modality that is minimally invasive, easy to learn with appropriate training, and carries a low risk (Figure 1).[30,31] It can be performed superficially (superficial dry needling – SDN) or intramuscularly (deep dry needling – DDN). In SDN the needle is inserted subcutaneously to a depth of 3 to 10 mm.

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The deep method of dry needling is considered more effective than the superficial method for the treatment of pain associated with myofascial trigger points. DDN results in significantly better analgesia than SDN, although post-treatment soreness is often reported for a period of 24 to 48 hours, depending on the sensitivity of the individual being treated.[31,32] It is suggested, however, that SDN is used over body regions with a potential risk of significant adverse events, such as the lungs and large blood vessels.

Despite a growing body of literature exploring the aetiology and pathophysiology of myofascial trigger points, the exact mechanisms of the therapeutic effects of trigger point dry needling are not understood. Simons, Travell and Simons indicated that the therapeutic effect of trigger point dry needling is the mechanical disruption of the trigger point contraction knots.[22]

When using invasive procedures like trigger point dry needling, eliciting LTRs is essential.[21] Not only is the treatment outcome much improved, but LTRs also confirm that the needle was indeed placed in a taut band, which is particularly important when needling myofascial trigger points close to peripheral nerves or viscera.[33] Dry needling techniques have been shown in studies to normalise the biochemical milieu associated with effective pain relief by means of eliciting an LTR (see the Box).[3,4,19]

Manual therapy methods

Manual therapy techniques are used to address the adaptive connective tissue changes that develop with MPS, and include myofascial manipulation, sustained digital/manual pressure and various massage methods (Figure 2).[34]

As mentioned earlier, the stiffness often felt in MPS is due to hyaluronic acid molecules aggregating in the altered biochemical milieu of myofascial trigger points. Myofascial manipulation and sustained digital/manual pressure have been shown to reverse this aggregation through the local increase in subcutaneous temperature that they cause promoting alkalinisation locally, with a resultant decrease in viscosity of the hyaluronic acid-containing extracellular matrix in the area. This restores the normal gliding properties between fascia and muscle as well as regenerating the activation of mechanoreceptors embedded in fascia. Sufferers report an increase in range of motion and a decrease in pain as a result.[26]

Figure 2. Myofascial manipulation being performed on the subscapularis muscle.

Other methods

Examining the postural alignment in space of a person with MPS and their multisegmental movement is an essential component in the conservative treatment of MPS, and helps understanding of the underlying biomechanical dysfunction that can bring about the sensory–motor complexity that is MPS. Once identified, correction of postural faults, restoration of joint range of motion through dry needling and manual therapy methods, and neuromuscular conditioning through therapeutic exercise can achieve maximal functional outcomes and long-term analgesia.

Where appropriate, modification of work practices (at work and at home) must be undertaken to prevent reactivation of MPS through pathological biomechanics, poor physical ergonomics or faulty sporting technique.

CONCLUSION

Myofascial pain is a common nonarticular musculoskeletal pain disorder characterised by the presence of myofascial trigger points that can create both local and referred pain. Although the underlying pathophysiology involves local tissue hypoxia, it is the altered biochemical milieu of the trigger point that maintains nociceptive activity in the trigger point region, and results in both peripheral and central sensitisation of the body's pain processing systems. This sensitisation combined with inhibition of the descending pain inhibitory pathways and specific input to the prefrontal cortex and limbic system by muscle nociceptors results in the distressing chronicity experienced by many patients with myofascial pain.

Despite its complexity, myofascial pain is relatively easily diagnosed by clinical evaluation. Treatment techniques such as dry needling have been shown to provide effective pain relief, and manual therapy techniques help relieve the associated stiffness.

Patients with MPS commonly present in general practice but the condition is not usually specifically diagnosed. With increased understanding of the nature and features of the clinical presentation of myofascial pain, it is possible for GPs to diagnose it early and institute appropriate referral, thus helping prevent the all-too-common development of a chronic pain state. MT

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A list of references is included in the website version (www.medicinetoday.com.au) and the iPad app version of this article.

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