Myofascial Pain Syndromes from Trigger Points

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Myofascial pain syndrome (MPS) is a common cause of acute and chronic pain that can complicate other medical illnesses and injuries. It is both defined by and diagnosed by the presence of the myofascial trigger point. Current studies indicate that the trigger point is a dysfunctional motor end plate whose abnormal activity is modulated in some way by the sympathetic nervous system. Pain syndromes arise from trigger points as causes of local pain and of referred pain. Referred pain from a few or from many muscle trigger points produces regional or generalized pain. Treatment requires the elimination of the trigger point by manual therapy or by trigger point injection and correction of the mechanical and medical factors that initiate and perpetuate it.

Myofascial pain syndrome (MPS) constitutes a substantial portion of the pain spectrum, acute and chronic, as both the primary cause of disability, and as a complication arising from other problems such as failed low back surgery, cervical whiplash, overuse, or repetitive strain syndrome. MPS is a very specific type of muscular pain, and is not to be confused with fibromyalgia. It is common after injury, resulting in so-called soft-tissue pain. MPS can be intermittent and mild, or debilitating and totally disabling. It can be acute, but it can still be effectively treated even when it has persisted for years.

Pain syndromes associated with individual myofascial trigger points have been well described. [1,2••] The interactions of individual muscle myofascial trigger points, and the interaction of myofascial trigger points in functional units of muscles, especially in chronic cases, cause regional and widespread pain syndromes. Important postural consequences of dysfunctional muscle units created by myofascial trigger points affect the overall distribution and spread of pain and must be understood for treatment to be effective. For example, a round-shouldered, head-forward posture has significant implications for the position of the jaw, the state of the facial and neck muscles, the relationship of the shoulder and anterior chest muscles, and the low back. The mandible will be repositioned posteriorly, affecting the pterygoid, masseter, and temporalis muscles. Forward shoulder posture is associated with shortening of the pectoralis major and minor muscles and constant tension with their antagonist muscles, the trapezius and rhomboids. The posterior cervical muscles are overloaded, as the head is pulled back (extension of the neck even though the head is forward). The low back is usually flattened, loading the lower back muscles (quadratus lumborum, iliocostalis, and multifidi) and altering the relationships and function of the lumbosacral junction and sacroiliac joints. These changes are associated with the development and maintenance of myofascial trigger points and with the pain affecting different regions (facial and jaw pain, neck and shoulder pain, and low back pain). This example emphasizes the importance of functional muscle units in the development and spread of myofascial trigger points.

The Myofascial Trigger Point

The myofascial pain syndrome is distinguished by the existence of the trigger point, which is characterized by the following features: 1) exquisite tenderness in a taut muscle band; 2) referred pain elicited by stimulation of the trigger point; local twitch or contraction of the taut band; 4)reproduction of the patient's spontaneous pain pattern when stimulated; 5) weakness without atrophy; and 6) restricted range of motion (Table 1). MPS is diagnosed by identification of these features of the myofascial trigger points on physical examination. Treatment involves the inactivation of the myofascial trigger point for immediate relief of pain and to provide a window of time to produce the necessary biomechanical changes that give sustained improvement. The factors in the patient that led to the MPS and that perpetuate it must be eliminated or corrected. These factors include postural dysfunction, impaired spinal joint function, pelvic rotation, leg length inequalities, ergonomic factors associated with work and recreation, and medical systemic factors such as nutritional or hormonal insufficiency.

A particular pain problem is called myofascial when one or more myofascial trigger points reproduce all or part of the individual's pain. As muscles are examined individually for myofascial trigger points, single muscle syndromes emerge. The pain experienced from a myofascial trigger

Table 1. The Physical Features of the Myofascial Trigger Point

1. Exquisite tenderness in a taut muscle band
2. Referred pain elicited by stimulation of the trigger point
3. Local twitch or contraction of the taut band
4. Reproduction of the patient's spontaneous pain pattern when stimulated
5. Weakness without atrophy
6. Restricted range of motion

point is a combination of the local pain from a single muscle and the pain felt in the zone of referred pain. The person suffering from an MPS may complain only of the pain felt in the referred pain zone, but treatment directed only toward the referred pain may be unsuccessful. Myofascial trigger points may refer pain locally. For example, upper trapezius muscle myofascial trigger points refer pain into the ipsilateral posterior neck. Pain from myofascial trigger points may also be felt in remote sites, as in the case of the infraspinatus muscle referral pattern to the arm and hand. MPS is more often the result of myofascial trigger points in many muscles, affecting a region or even appearing as widespread pain involving three or four regions of the body. The spread of myofascial trigger points and therefore of myofascial pain occurs as a result of the development of myofascial trigger points in dysfunctional muscle units and in the muscles in the referred pain zone.

Trigger point physiology

No specific or consistent biochemical changes have been reported in the myofascial trigger point. The known plasticity of the nervous system as it responds to noxious stimulation can explain much of the clinical phenomena that occur. The nature of the myofascial trigger point itself had not been understood until quite recently, when Hubbard and Berkoff [3•] reported spontaneous electrical activity (SEA) in the myofascial trigger point that was not seen in the adjacent nontender muscle. They postulated that the activity was generated in the intrafusal muscle spindle fibers on the basis of preliminary pharmacologic studies that showed an inhibiting effect of the alpha-adrenergic blocking agent, phentolamine. Chen *et al.* found that phentolamine reduced the SEA of the trigger point significantly in the rabbit model [4]. SEA is clustered within a region of the taut band that these authors call the active trigger zone. The abnormal electrical activity comes from the motor end plate, the evidence being the characteristic electrical discharges of end-plate activity. A current hypothesis based on the available experimental data is that the motor end plate is dysfunctional, consistent with about a thousand-fold increase in the release of acetylcholine. This release then results in a sustained, abnormal increase in the activity of the motor end plate [5•], and the abnormal sustained activity is somehow modulated by sympathetic activity.

Tenderness

The foremost characteristic of the myofascial trigger point is the exquisitely tender point in a taut band. This tender point displays both hyperalgesia, which is an excessive response to normally painful stimulation, and allodynia, which is the perception of pain in response to normally nonpainful stimulation. Muscle is supplied with afferent nociceptor nerve endings that are sensitive to pressure and chemical stimulation and that activate dorsal horn neurons. The dorsal horn neurons receive input from multiple muscle sites (receptive fields) and from different deep tissues, including either other muscles or viscera. Sensitization to peripheral noxious stimuli occurs in muscle nociceptors, including dorsal horn neurons, as it does in skin and other tissues.

Substance P is a neuropeptide that modulates nociception by activating nociceptor neurons. Release of substance P by action of nociceptive afferents can enhance the activation of dorsal horn nociceptors, contributing to the sensitization of dorsal horn neurons [6]. Thus, the two key features of the myofascial trigger point, tenderness and referred pain, have their origin in the ability of the nervous system to modulate afferent nociceptive activity. Tenderness is really an expression of sensitization; referred pain is an expression of activation of afferent activity at remote sites. Referred pain is not a phenomenon unique to the myofascial trigger point but is well known clinically throughout medical practice as in anginal pain referred to the neck and down the arm.

These considerations have definite practical implications for MPS, as they suggest treatment approaches that work through inhibiting pain input and transmission from myofascial trigger points. Inactivation of myofascial trigger points in one muscle by the injection of local anesthetic can alter the activity of distant myofascial trigger points [7], showing the importance of these modulating factors.

Local twitch response

The local twitch response (LTR) elicited by mechanical stimulation of the trigger point is an unequivocal sign of a myofascial trigger point, whether active or latent. During the LTR, the taut band in which the tender point is found is the only thing that contracts, not the entire muscle. A burst of high amplitude electrical activity occurs with the LTR. Interruption of the proximal nerve greatly diminishes but does not abolish the LTR. LTRs in taut bands of rabbit skeletal muscle are related to the reflexes at spinal cord level [8]. The LTR is a spinal cord reflex requiring an intact peripheral nerve. It can be elicited by inserting a needle into an myofascial trigger point taut band at the trigger point zone in the process of inactivating a trigger point. The LTR is a sign that the trigger zone has been reached by the treating needle.

Diagnosis

The diagnosis of MPS is made by physical examination. The history provides the context of the pain problem, but

the diagnosis must be based on the identification of the myofascial trigger point by physical examination. The myofascial trigger point is distinguished from simple muscle tenderness by the presence of the taut band and by referred pain. A local twitch response is confirmatory. Restricted range of motion about a joint or a part of the body like the neck or waist is highly suggestive that trigger points are present. The diagnostic process involves an overall evaluation of possible factors that could explain the patient's symptoms. The diagnosis of MPS is not one of exclusion. Rather, it is based on positive findings. Thus, pain in the shoulder must include the usual differential diagnostic considerations of local shoulder dysfunction, including rotator cuff syndrome, impingement syndrome, bursitis, cervical disk disease, and intracapsular disorders, *eg*, degenerative arthritis and adhesive capsulitis. Myofascial trigger points can complicate any of these conditions. A comprehensive examination for myofascial trigger points should include all the muscles that can refer pain to the shoulder region.

Trigger point examination reliability

A study establishing the reliability of the examination of the six major physical features of the myofascial trigger point showed a high inter-rater agreement for all features studied [9•]. There was variation, however, in the degree of agreement among the different features and among the different muscles examined. Some features, such as tenderness and reproduction of usual pain, had very high levels of agreement, but the LTR consistently showed a lower level of agreement, although the agreement present was significant. One should not expect to identify all features of a myofascial trigger point by manual examination in every muscle examined. Some muscles are more difficult to examine than others are, and some features of the trigger point are more difficult to elicit than others. Attempts to quantify the measurement of tenderness have led to use of the pressure threshold meter or algometer [10]. Even with algometry, however, manual examination is needed at least for identification of the taut band that characterizes the myofascial trigger point.

Other laboratory studies

The myofascial trigger point taut band is visible on high resolution B-mode ultrasound. This technique allows visualization of the localized twitch in the taut band of the myofascial trigger point when stimulated by needle insertion [11]. Thus, the physical response of the trigger point to treatment can be documented, and a permanent record can be made.

The characteristic electromyographic activity of the trigger point can be used to identify and document its presence. No other laboratory studies (including x-rays, CT, and MRI) have shown positive results or abnormalities in myofascial pain syndromes. All blood tests are normal unless there are comorbid conditions. Comorbid disorders exist in MPS and can be important causes of chronic MPS that are refractory to treatment, but they are not directly related to the development of the trigger point. Identification of cervical facet joint injury or of hypothyroidism, both potentially comorbid conditions with MPS, requires appropriate laboratory testing. Surface EMG is useful in assessing muscle function in MPS. The evaluation of trigger point activity with dynamic EMG techniques [12] shows asymmetries in the activity of the affected paired muscles and persistent increased muscle activity. Surface EMG can objectively demonstrate muscle-related postural dysfunction and functional muscle unit imbalance in a muscle containing trigger points.

Myofascial Trigger Point Syndromes

Headache

Trigger point syndromes can be grouped according to the area involved. Headache is a common complaint, and one that is closely related to myofascial trigger points. Headache pain can be a consequence of myofascial trigger points located in the shoulder, neck, or facial muscles. The pain is referred from trigger points to various parts of the head and neck, causing symptoms of headache that are often attributed to migraine or sinus headache. The headaches can be constant or throbbing; unilateral, bilateral, or circumferential (bandlike around the head); and vertex, occipital, or retro-orbital. When muscle pain activates peripheral nerve pain receptors in the trigeminal nerve distribution or in the upper cervical spine segments (which have pain input into the subnucleus caudalis of the trigeminal nerve), it is likely that the trigeminovascular system is activated. This process results in a cascade of neuropeptide and vasoactive peptide release (substance P and calcitonin gene-related protein, for example) and in the development of migraine phenomena such as photophobia, diaphoresis, nausea, and dizziness. In this way, muscle trigger points could precipitate migraine phenomena.

The most common cause of myogenic headache in the author's experience is referred pain from muscle trigger points. Upper trapezius muscle trigger points cause referred pain to be felt in the temple and along the side of the head above and behind the ear. The trapezius functional muscle group includes the antagonist muscles that flex and rotate the neck. Its functions include extension of the neck against resistance and assistance in lateral rotation and lateral flexion. The sternocleidomastoid muscles, which flex and rotate the neck, frequently have myofascial trigger points in conjunction with the trapezius muscle. The composite headache includes the occiput, the vertex of the head, the supraorbital area, and the forehead, and includes the opposite side, the temple, the side of the head, and the ear, or any combination of these areas (Fig. 1) The splenius capitis and cervicis are also part of the functional muscle unit of the trapezius and the sternocleidomastoid. They act as extensor muscles of the neck in synergy with

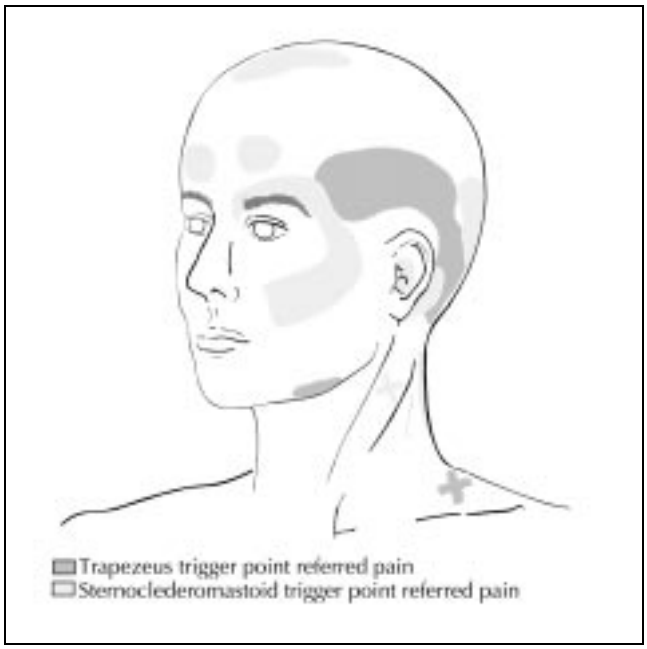

Figure 1. Composite of trigger point referral patterns from the trapezius and sternocleidomastoid muscles that are commonly associated with headache.

the trapezius muscles, as agonists to the contralateral upper trapezius for rotation, and as antagonists to sternocleidomastoid flexor function. They are also likely to have active trigger points along with the trapezius and sternocleidomastoid muscles. Referred pain from the splenius capitis and cervicis add to headache along the side of the head, the vertex of the scalp, and the retro-orbital area. Recurrent headache is frequently the result of myofascial trigger points from these and other anterior and posterior neck muscles.

Shoulder and neck pain

Pain in the neck and shoulder can be the direct result of muscle trigger points. (Fig. 2) Pain in the back of the neck occurs with trigger points in the posterior cervical, and suboccipital muscles. Trigger points in these muscles refer pain into the head but also have local pain. The deep paraspinal multifidi muscles refer pain to the upper neck, the shoulder, and towards the scapula. These muscles may be dysfunctional and are likely to develop myofascial trigger points. This development occurs in situations of postural abnormality or restriction of motion caused by trigger points in the muscles that move the jaw or the muscles that control head flexion, extension, rotation, or lateral bending. The levator scapula contributes to extension of the neck (a function of the bilateral activation of the muscle) and to elevation of the shoulder and rotation of the scapula. Pain from levator scapula trigger points is felt in the angle of the neck and shoulder, medial to the scapula, and into the posterior shoulder. Levator scapula function is synergistic with that of the upper trapezius in elevation of the shoulder and is antagonistic to the rotational direction of force of the trapezius. The levator scapula rotates the glenoid fossa downwards, and the trapezius rotates it upwards. Trigger point pain tends to spread among the muscles that move the scapula. Because myofascial trigger points spread through functional muscle units, trigger point pain will extend through overlapping muscles sharing function or control of a body part.

Injury to the rotator cuff muscles can primarily affect just one muscle, such as the supraspinatus or infraspinatus muscle. The entire rotator cuff complex tends to become secondarily involved. A painful trigger point in the rotator cuff muscle teres major is usually accompanied by a trigger point in the latissimus dorsi, with which it shares adduction and internal rotation of the humerus. The latissimus dorsi attaches superiorly to the humerus but has a common tendon with the teres major. It acts on the scapula through its control of the humerus and the force exerted through the teres major. Inferiorly, its attachment to the lower six thoracic vertebrae and all of the lumbar vertebrae allows it to assist in low back and pelvic movement. Mechanical dysfunction in these movements can lead to the development or perpetuation of trigger points in the primary effectors of low back and pelvic movement and stability. These include the iliocostalis, the quadratus lumborum, and the iliopsoas muscles. Examination of pain in the shoulder region should be accompanied by evaluation of the low back and pelvis region.

A common forearm trigger point pain syndrome is lateral epicondylitis. This condition is usually attributed to tendinitis. It may involve an enthesopathy of the proximal attachment of the extensor carpi radialis longus and brevis and the extensor digitorum. Trigger points in the muscle belly itself accompany the attachment trigger points. Relief of pain is achieved by elimination of the muscle trigger points and the attachment trigger points by manual inactivation or by injection.

Back and hip pain

Trigger points occur in many areas of the low back and pelvic region (Fig. 3). The acute (and chronic) inability to straighten up because of back pain is frequently the result of quadratus lumborum muscle trigger points. This muscle is an extensor and lateral flexor of the waist. Pain from quadratus lumborum trigger points is felt across the low back and is most severe when the patient is upright, whether sitting, or standing, with the back unsupported. Pain can also be felt in the groin, in the testicles, in the back, and in the hip, or it can be felt as sciatica. Bending, turning, and lifting aggravate the pain. This pain is distinguished from hip joint pain on the basis of the location of the pain when the patient is standing. When the pain emanates from the hip joint, there is little back pain and mostly hip, groin, or sacroiliac joint region pain. Quadratus lumborum pain persists when the patient is sitting or recumbent and increases with attempted movement. Hip

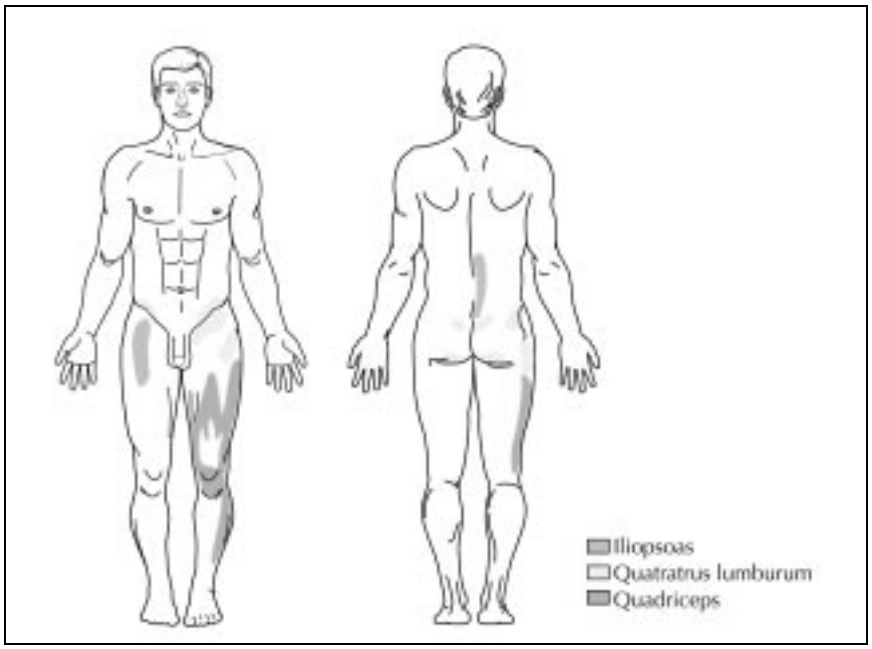

Figure 2. Composite of trigger point referral patterns from the iliopsoas, quadratus lumborum, and quadriceps muscles that are associated with low back and pelvic region pain.

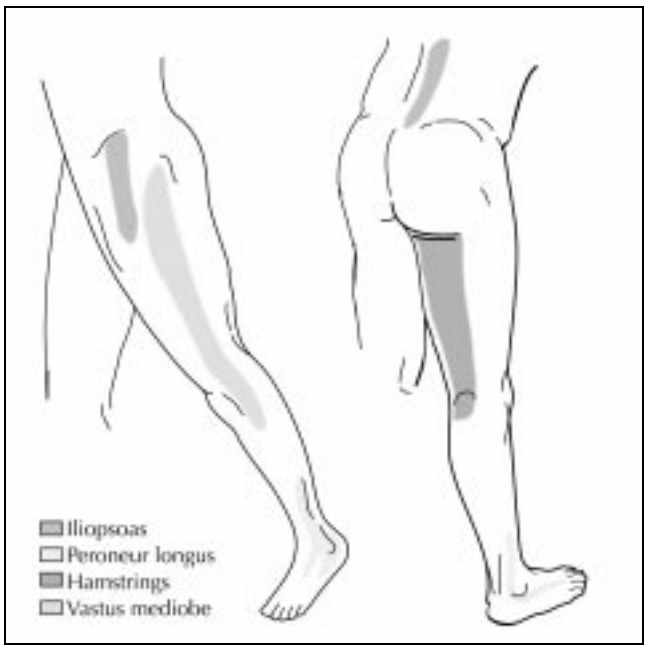

Figure 3. Composite of trigger point referral patterns from the iliopsoas, peroneus longus, hamstring, and vastus medialis muscles that are commonly associated with pelvic region and leg pain.

joint pain is associated with limited range of motion at the hip and relief of pain when sitting or reclining.

Pain in the hip and pelvic region can also be the result of the quadratus lumborum antagonist, the iliopsoas. This flexor of the waist and hip typically causes pain in a vertical distribution in the low back, perceived as across the back when bilateral, and in the anterior thigh. This type of pain is often involved in association with extensors of the back and hips (quadratus lumborum, gluteus maximus, and hamstring muscles). Alleviation of myofascial pain in the low back-pelvic region requires treatment of the iliopsoas muscle. If the iliopsoas trigger point-induced restricted range of motion persists after release of extensor muscle trigger points, back and thigh pain will persist, and a flexion-extension postural dysfunction will result because of the now dominant restriction of flexion in the iliopsoas.

Knee and ankle pain

Anterior knee pain from muscle trigger points results from trigger points in the quadriceps muscle. Myofascial trigger points in the rectus femoris head of the quadriceps can refer pain to the patella and distal anterior thigh. Trigger points in the vastus medialis and vastus lateralis refer pain to the medial and lateral aspects of the knee respectively. The pain they cause can be mistaken for collateral ligament pain.

Back-of-knee pain is usually the result of trigger points in the hamstring muscles, the extensors of the hip, and the flexors of the knee. These muscles are active when the trunk is flexed while standing, controlling flexion. In this way, their function is closely related to iliopsoas function, and the two muscles must be considered together. Trigger points in the small popliteus muscle that attaches to the lateral femoral condyle superiorly, and to the tibia inferiorly, cause pain in the back of the knee and should be considered when pain persists after inactivation of hamstring trigger points.

An everted, pronated foot can result in trigger points in the peroneus longus muscle, referring pain to the anterolateral ankle; in the vastus medialis, referring pain to the knee; and in the gluteus medius muscle, referring pain to the hip. Anteromedial and great toe pain results from trigger points in the anterior tibialis muscle. The same forces that cause an anterior compartment syndrome will cause trigger point formation. Injury to the ankle or foot that

limits movement and impairs dorsiflexion of the foot will overwork the anterior tibialis and result in myofascial pain. This mechanism is exaggerated in women who wear high heels, which increase the plantar flexion of the foot.

Osteopathic concepts and myofascial pain

Osteopathic concepts are only now being emphasized in myofascial pain syndromes in conjunction with myofascial concepts. Understanding the three-dimensional movement of the pelvis, including the sacroiliac joint and lumbosacral junction, increases the ability to diagnose and treat myofascial pain syndromes. Restricted motion of cervical vertebrae, thoracic and lumbar vertebrae, and of the sacroiliac joint results in perpetuation of trigger points. Recurrent thoracolumbar myofascial pain syndromes may require release of segmental spinal hypomobility. Fusion of L5 to S1 results in shifting of the reciprocal movement of the sacroiliac joint when the patient is walking, often producing a myofascial hip and buttock pain syndrome.

Facet joint syndrome

Injury to the cervical facet joint, as in whiplash, can produce a pain syndrome that overlaps and accompanies the myofascial pain syndrome. Persistent or chronic neck or shoulder pain after whiplash demands examination of the facet joints and denervation of the joint if it is found to be the cause of pain. Only then will myofascial trigger point inactivation be effective.

Treatment

Elimination of the myofascial trigger point is the central feature of treatment in the myofascial pain syndrome because this elimination is critical to the relief of pain and the restoration of normal function. Restoration is achieved through the correction of the mechanical and systemic medical dysfunctions that interfere with the ability of the muscle to recover. The trigger point is inactivated either manually or by invasive techniques such as trigger point injections. Simons [2••] *et al.* used the technique of intermittent "cold and stretch" as a way of lengthening the muscle and decreasing or eliminating the trigger point. This technique involves gradual stretching of the muscle while vapo-coolant is applied, until the maximum stretch is achieved or a barrier is reached. The stretch for a particular muscle is always in the opposite direction of the muscle's action. Heat is applied after stretching in order to reduce poststretching discomfort. Other techniques have been developed for the inactivation of the trigger point and the restoration to full length of the muscle. These include trigger point compression, local and regional muscle stretching, myofascial release techniques, the postisometric contraction or relaxation technique (often used with respiratory facilitation) [13], strain-counterstrain, superficial or subcutaneous dry needling [14], and acupuncture, in addition to traditional physical therapy techniques of electrical stimulation and ultrasound. Surface EMG is an effective tool in identifying muscle groups that need to be treated, in assessing the effectiveness of muscle-specific training, and in teaching patients how to control their muscle tension through biofeedback.

Trigger point injection with local anesthetic, or dry needling without anesthetic, are examples of invasive procedures that might be called upon to facilitate the relief of myofascial pain [15]. Inactivation of the myofascial trigger point can be accomplished by insertion of a needle into the trigger zone of the myofascial trigger point. The technique of injection is described in detail by Hong [16]. Procaine is the anesthetic of choice because it is metabolized locally by procaine esterase, and its effective half-life is as short as 10 to 15 minutes. If a local nerve block is inadvertently produced, numbness and weakness clear rapidly. Vitamin C is given for 3 days prior to injection therapy because it reduces bleeding caused by the needle. Risks of trigger point injection include bleeding, organ or vessel puncture, nerve injury, syncope, and allergic reactions. A precise knowledge of anatomy is required of the clinician who employs this technique. Another therapeutic technique now being evaluated is the injection of botulinum toxin into the trigger point [17,18•].

Whatever method is used to inactivate myofascial trigger points, all trigger points in a functional muscle unit should be addressed by a combination of manual techniques and injection if necessary. Stretching the muscles in the affected functional muscle unit is an essential part of the treatment. Moist heat applied following the stretching decreases posttreatment muscle soreness.

Pharmacologic therapy

No studies have looked at drug treatment of the myofascial pain syndrome itself. Analgesics play a role in producing comfort during the acute or treatment phase. Nonsteroidal anti-inflammatory drugs offer analgesic benefit but nothing more specific. The antidepressant drugs offer the same potential relief for chronic MPS as they do in any other chronic pain syndrome. The same principles that apply to treatment of nonmalignant pain of any source are applied to the treatment of MPS.

Psychological factors

A perpetuating factor that must always be considered in relation to the myofascial pain syndrome is psychological stress. In one study involving needle electromyographic evaluation, the trigger point in the trapezius muscle specifically responded to a psychological stressor, whereas the adjacent nontender muscle did not [19•]. Another area of research involves the evaluation of psychological stressors in myofascial pain specifically in women [20].

Conclusions

Myofascial pain syndrome is a painful muscle disorder that occurs following stress or injury in muscle. It is diagnosed by manual examination and treated through a variety of manual and invasive techniques. Correction of underlying or comorbid disorders, including postural and psychological stressors, is a necessary aspect of the treatment.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- • Of special interest
- •• Of outstanding interest

1. Travell JG, Simons, DG: *Myofascial Pain and Dysfunction: The Trigger Point Manual,* Vol 2. Baltimore: Williams and Wilkins; 1992.

2.•• Simons DG, Travell JG, Simons LS: *Myofascial Pain and Dysfunction: The Trigger Point Manual,* vol 1, edn 2. Baltimore: Williams and Wilkins; 1999.

Latest version of the standard and best textbook in the field. Chapter 2 in this volume presents the latest data on the mechanism of trigger point formation and physiology. Chapter 4 is the best available statement on the causes of chronic myofascial pain.

3.• Hubbard DR, Berkoff GM: **Myofascial trigger points show spontaneous needle EMG activity.** *Spine* 1993, **18:**1803–1807.

First to describe the spontaneous electrical activity in the trigger point and to compare it to adjacent normal muscle.

4. Chen, JT, *et al.*: **Phentolamine effect on the spontaneous electrical activity of active loci in a myofascial trigger spot of rabbit skeletal muscle.** *Arch Phys Med Rehabil* 1998, **79:**790–794.

5.• Hong C-Z, Simons DG: **Pathophysiologic and electrophysiologic mechanisms of myofascial trigger points.** *Arch Phys Med Rehabil* 1998, **79:**863–872.

Most up-to-date presentation of the physiology of the trigger point. The data are similar to those presented in references [1] and [2].

6. Mense S: **Pathophysiologic basis of muscle pain syndromes.** In *Myofascial Pain: Update in Diagnosis and Treatment.* Edited by Fischer AA. Philadelphia: W.B. Saunders; 1997:23–53.

7. Carlson CR, *et al.:* **Reduction of pain and EMG activity in the masseter region by trapezius trigger point injection.** *Pain* 1993, **55:**397–400.

8. Hong C-Z, Torigoe Y, Yu J: **The localized twitch responses in responsive taut bands of rabbit skeletal muscle are related to the reflexes at spinal cord level.** *J Musculoskeletal Pain* 1995, **3:**15–33.

9.• Gerwin RD, *et al.:* **Interrater reliability in myofascial trigger point examination.** *Pain* 1997, **69:**65–73.

Reports on a study that established the inter-rater reliability of the myofascial examination and therefore confirmed its credibility as a diagnostic tool.

10. Fischer AA: **New developments in diagnosis of myofascial pain and fibromyalgia.** In *Myofascial Pain: Update in Diagnosis and Treatment.* Edited by Fischer AA.. Philadelphia: W.B. Saunders;1997:1–21.

11. Gerwin RD, Duranleau D**: Ultrasound identification of the myofacial trigger point.** *Muscle Nerve* 1997, **20:**767–768.

12. Donaldson CCS: **The evaluation of trigger-point activity using dynamic EMG techniques.** *Am J Pain Manage* 1994, **4:**118–122.

13. Lewit K: **Treatment of myofascial pain and other dysfunction disorders.** In *In Pain Research and Clinical Management: Progress in Fibromyalgia and Myofascial Pain*. Edited by Værøy H, Merskey H. Amsterdam: Elsevier; 1993:375–392.

14. Baldry PE: *Acupuncture, Trigger Points and Musculoskeletal Pain*. Edinburgh: Churchill Livingstone; 1993.

15. Lewit K: **The needle effect in relief of myofascial pain.** *Pain* 1979, **6:**83–90.

16. Hong C-Z: *Myofascial trigger point injection. Critical Reviews in Physical Medicine and Rehabilitation.* 1993.

17. Alo KM, *et al.:* **Botulinum toxin in the treatment of myofascial pain**. *Pain Clin* 1997, **10:**107–116.

18.• Cheshire WP, Abashian SW, Mann JD: **Botulinum toxin in the treatment of myofascial pain syndrome.** *Pain* 1994, **59:**65–69.

First study to show that botulinum toxin can inactivate myofascial trigger points, thus opening this approach as a treatment possibility and illuminating the nature of the trigger point, which is affected by botulinum toxin.

19.• McNulty W, *et al.:* **Needle electromyographic evaluation of trigger point response to a psychological stressor.** *Psychophysiology* 1994, **31:**313–316.

Demonstrates the responsiveness of the trigger point to psychological stressors, a response that is not seen in normal muscles away from the trigger point.

20. Zautra AJ, *et al.:* **The evaluation of myofascial face pain and its relationship to psychological distress among women.** *Health Psychol* 1995, **14:**223–231.11.