

# Classification, Epidemiology, and Natural History of Myofascial Pain Syndrome

Robert D. Gerwin, MD

---

## Address

Johns Hopkins University, Department of Neurology,  
7830 Old Georgetown Road, Bethesda, MD 20814-2432, USA.

**Current Pain and Headache Reports** 2001, 5:412–420

Current Science Inc. ISSN 1531-3433

Copyright © 2001 by Current Science Inc.

Myofascial pain syndrome is a disease of muscle that produces local and referred pain. It is characterized by a motor abnormality (a taut or hard band within the muscle) and by sensory abnormalities (tenderness and referred pain). It is classified as a musculoskeletal pain syndrome that can be acute or chronic, regional or generalized. It can be a primary disorder causing local or regional pain syndromes, or a secondary disorder that occurs as a consequence of some other condition. When it becomes chronic, it tends to generalize, but it does not change to fibromyalgia. It is a treatable condition that can respond well to manual and injection techniques, but requires attention to postural, ergonomic, and structural factors, and toxic or metabolic factors that impair muscle function.

## Introduction

Myofascial pain syndrome (MPS) is a musculoskeletal disorder that can be acute or chronic. It is precisely defined [1••], and its consequences in terms of dysfunction, disability, and financial loss are great. Lack of a pathoanatomic basis for the formation of trigger points (TrPs) has led to a questioning of its existence [2], although an equal lack of a proven basis for migraine headaches for many years did not keep people from suffering from migraine. The pathophysiologic basis for the formation and persistence of the myofascial TrP is under vigorous investigation in a number of countries today. MPS is often compared with, contrasted to, and mistaken for fibromyalgia. In the International Association for the Study of Pain publication, *Classification of Chronic Pain* [3], MPS is mentioned only in connection with fibromyalgia: "We consider myofascial pain syndrome (diffuse or not) to have a somewhat different meaning and think it adds confusion to use the term when discussing fibromyalgia." Nowhere else in this classification is MPS mentioned. This article discusses the

classification of MPS in relationship to other pain disorders. The term prevalence will be used in its accepted meaning of a percentage of a population that has a given condition during a defined period of time. The incidence is the number of persons who develop the condition or who develop new symptoms during a specified time period.

## Classification Definition

Myofascial pain syndrome is defined as pain of muscular origin that originates in a painful site in muscle. This site is characterized by the myofascial TrP [1••]. It is defined by its physical (motor) characteristics and by its sensory features. Not all of the motor and sensory features of the TrP need be present to identify it clinically. There are also autonomic features of the TrP that are not essential to its clinical identification. The minimum essential features that must be present to distinguish the TrP from other types of muscle pain are the taut band with a zone of tenderness in it. Reproduction of part or all of the subject's pain by stimulating the zone of tenderness in the taut band is the most important aspect of the TrP relating it to the subject's pain problem (Gerwin *et al.*, Unpublished data presented at the 4th International Conference on Myofascial Pain and Fibromyalgia, Silvi Marina, Italy, 1998).

The physical or motor sign of the TrP is the taut band. It is a contracted group of muscle fibers that runs from one end of the muscle to the other, except in those muscles where there is anatomic segmentation of the muscle, as in the semitendinosus head of the hamstring, which has a tendinous inscription in its proximal portion dividing the muscle into two (and sometimes three) parts. The taut band is not sustained by  $\alpha$  motor neuron activity, because it does not show motor action potential activity that ordinarily accompanies such activity. Instead, the electromyographic activity that is associated with the TrP in the taut band is the presence of negative-positive potentials known as end-plate noise, resembling miniature end-plate potentials except that they occur up to 1000 times more frequently [4]. This activity, termed spontaneous electrical activity, is uniquely associated with the zone of tenderness in the taut band [5]. The exact nature of this activity and its

relationship to the development of the taut band is presently being investigated. Nevertheless, the argument for the spontaneous electrical activity originating in a dysfunctional motor end plate is compelling.

A second pathophysiologic abnormality uniquely associated with the myofascial TrP is the local twitch response. This is a sharp contraction of the taut band (and of closely related taut bands, but not of the entire muscle in contrast to a tendon reflex) that is a spinal cord reflex, which is initiated by a sharp physical stimulus such as plucking of the taut band by hand or by the insertion of a needle into the trigger zone of the taut band [6].

The sensory manifestation of the TrP is tenderness. Tenderness at the TrP zone is either an increased pain response in relation to the stimulus (hypersensitivity) or a pain response to normally nonpainful stimulation (allodynia). Both phenomena involve central sensitization at the dorsal horn level and higher [7••]. Referred pain is not necessary for the identification of a TrP, but is intrinsic to the reproduction of a subject's pain by stimulating the TrP, because most often perceived pain in the MPS is referred pain as well as local pain.

A muscle with a TrP does not work effectively. The taut band restricts the stretch of the muscle, thereby limiting associated range of motion. Weakness is produced by pain-induced muscle inhibition as well as by muscle shortening. Coordination is affected as reflex inhibition of antagonistic muscle activity is impaired.

### Differentiation from fibromyalgia

Fibromyalgia is a condition of diffuse muscle tenderness without taut bands [8•,9]. It is defined as a chronic form of myalgia of at least 3 months duration and widespread muscle tenderness. The distinguishing presence of tender points (TPs) in at least three of the four quadrants of the body (right, left, upper, and lower) are reflected in the research criteria of 11 positive out of a possible 18 predetermined TP sites [8•]. The research criteria have slipped into common usage as the clinical criteria for the diagnosis of fibromyalgia. It is important to realize, however, that TPs are not TrPs. They do not have taut bands and they do not refer pain to distant sites (pain referral zones) as TrPs do [10]. The distinction requires careful attention during physical diagnosis, because all TrPs are themselves tender. The distinction between the two physical features requires the search for taut bands with the examining hand. Identification of referred pain, by definition not a feature of the TP, requires the application of pressure on the tender/trigger point for up to 5 to 10 seconds, because referred pain is a delayed response, not an instantaneous development. Therefore, the examiner cannot sweep over the region to be examined simply to determine if the muscle is tender or not. The most important differential diagnosis for fibromyalgia is, in fact, MPS. The distinction can be made only on the basis of careful physical examination, especially when MPS is widespread.

### Regional versus generalized pain syndrome

In contrast to the generalized nature of fibromyalgia, MPS has been characterized as a regional pain syndrome [10]. This usage has been adopted from the early teaching of MPS as single muscle syndromes, and numerous descriptions of new presentations of single muscle or regional MPS [11–13]. Shoulder pain could stem from TrPs in the subscapularis muscle and its functional group. Thus, frozen shoulder is a regional MPS, in addition to any other structural involvement such as a rotator cuff tear. Once both shoulders and a hip are involved, however, the possibility that this could still be the result of TrP-related pain seems to be overlooked because the condition is now generalized.

This distinction between regional and generalized pain, the former being MPS, the latter being fibromyalgia, has persisted in the literature, nonetheless [14]. However, MPS can occur as a generalized condition involving three or four quadrants of the body [15,16]. The spread of TrPs occurs 1) through the axial kinetic chain as a result of postural dysfunction and mechanical stress on muscle, and 2) through activation of TrPs in functional muscle groups, where muscles that act as agonists or antagonists become overloaded or mechanically stressed compensating for the dysfunction of other muscles in the functional muscle unit.

### Central sensitization

Somatic pain can cause rapid changes in the central nervous system, a phenomenon known as central sensitization, which is a manifestation of neuroplasticity, or the remodeling of central processes in response to peripheral stimulation. Central sensitization is important in the development of both tenderness and referred pain, and in the conversion of an acute syndrome to a chronic one, and may be an important factor in the genesis of both fibromyalgia and MPS [17,18]. Central sensitization could explain both the physical findings of widespread tenderness and the generalized hypersensitivity that is seen in fibromyalgia [19], as well as some of the biochemical changes that have been documented in fibromyalgia, such as the elevation of substance P in the spinal fluid [20].

### Primary versus secondary myofascial pain syndrome

Myofascial pain syndrome can also be classified as primary syndromes (Table 1) that are not related to other medical conditions, and secondary syndromes (Table 2) that occur in conjunction with other medical conditions. Primary myofascial syndromes are often the typical overuse syndromes that are named for the structures involved or for common conditions that produce them. Thus, primary myofascial syndromes in the upper part of the body include tennis elbow or lateral epicondylitis, a condition involving the forearm extensor muscles, the extensor carpi radialis, and the supinator muscle. Another upper body primary myofascial syndrome is frozen shoulder, involving the subscapularis muscle, and usually the infraspinatus,

**Table 1. Primary myofascial pain syndromes**

Myogenic headache: chronic tension-type and mixed tension-type/migraine headache
Neck pain
Shoulder pain
Frozen shoulder
Low back pain
Piriformis syndrome
Knee pain
Ankle pain

the latissimus dorsi, the teres major and minor, the supraspinatus muscle, and one or both pectoralis muscles (major and minor). In the lower half of the body, the quadratus lumborum syndrome is a common cause of low back pain, and the piriformis syndrome a common cause of buttock, hip, and lower extremity pain. Primary MPS can also be secondary pain syndromes.

#### *Primary myofascial pain syndrome*

Chronic tension-type headache (myogenic headache) is frequently accompanied by migrainous phenomena like nausea and light sensitivity or photophobia. Such headaches can be thought of as mixed tension-type/migraine headaches because they have features of both at one time or another. One hundred percent of such patients, most of whom had headaches more than 15 days a month, had active myofascial TrPs (Gerwin, Unpublished data). Some patients had cervical spondylosis, but many did not; and some were affected by psychological stresses, but others were not. There did not seem to be a clear role for any specific etiology. Headaches are the expression of referred pain from the muscles of the neck and shoulder to pain referral zones in the head.

Neck pain can be directly caused by TrPs in the muscles of the neck and shoulder (sternocleidomastoid, scalenes, levator scapulae, trapezius, suboccipital and posterior cervical muscles). Postural stresses are among the most common causes of TrP-related neck pain, especially forward head posture and forward rolled shoulders. Posterior displacement of the mandible predisposes to this stressful posture, and can induce a concomitant temporomandibular joint dysfunction syndrome of head and neck pain. Mouth breathers will also adopt this posture, making allergy and nasal congestion a potential factor.

Shoulder pain and restricted shoulder movement can be the result of primary TrP development due to overuse, especially in the subscapularis muscle. The infraspinatus muscle is also frequently involved, as can be the pectoralis major and minor, the two teres muscles, and the latissimus dorsi. The most overlooked muscle in this group, however, is the subscapularis. This can be triggered by injury, but also by poor body biomechanics, especially in external and internal rotation motions of the shoulder.

**Table 2. Secondary myofascial pain syndromes**

Chronic cervical whiplash neck pain
Temporomandibular joint dysfunction
Secondary frozen shoulder: impingement syndrome, rotator cuff tear
Visceral pain syndromes: nonanginal chest pain, hepatic referred pain, renal/ureteral flank pain
Structural anatomic variations: scoliosis, pelvic torsions, leg-length inequality
Radicular pain
Postlaminectomy syndrome
Rheumatoid arthritis
Osteoarthritis: cervical and lumbar spondylosis, hip or shoulder osteoarthritis
Sjögren's syndrome
Fibromyalgia
Acute trauma: fractures, soft-tissue injury, postoperative pain
Hypothyroidism
Chronic infection: candidiasis, parasitic disease
Vitamin B <sub>12</sub> deficiency

Hip pain is often diagnosed as trochanteric bursitis when it is lateral, but can be caused by TrPs in the quadratus lumborum, gluteus medius, tensor fascia lata, and piriformis muscles particularly. Treatment of TrPs identified in one or more of these muscles often ends the hip pain. Pain in the anterior hip region can be caused by TrPs in the psoas or external abdominal oblique muscles, and the adductor longus and brevis and adductor magnus muscle.

Buckling knee syndrome was described by Travell and Simons [21] as coming from TrPs in the vastus medialis muscle. TrPs in this muscle can form in response to foot instability, or excessive pronation of the foot, which externally rotates the tibia, stressing the vastus medialis and the gluteus medius in the hip region.

Low back pain is most often associated with quadratus lumborum muscle TrPs, often with an accompanying lumbar multifidi and superficial paraspinal muscle TrPs. In addition to poor body mechanics, structural asymmetries such as scoliosis and pelvic torsion contribute to chronic shortening of the quadratus lumborum muscle. TrPs in the psoas muscle often develop when the quadratus lumborum is involved, and the psoas muscle itself is an often overlooked cause of back pain. The quadratus lumborum muscle, because of its attachment to the pelvis, tends to put the pelvis into anterior rotation when it is shortened. Treating quadratus TrPs usually requires treating the hamstring muscles as well, because they are under constant tension when the pelvis is anteriorly rotated.

Piriformis syndrome can be the result of buttock and hip pain caused by TrPs in the piriformis muscle alone, or by compression of the sciatic nerve by the shortened and thickened piriformis muscle. When the sciatic nerve is compressed, pain may be felt in the distribution of the sciatic nerve, involving the back of the thigh, the entire leg

below the knee, and the foot. This is another under-recognized cause of hip, leg, and foot pain that is rapidly reversed when the TrPs are injected with local anesthetic, a valuable diagnostic test.

#### *Secondary myofascial pain syndrome*

Cervical whiplash can be a chronic problem in 20% to 40% of cases, and 50% of those are associated with injury to one or more cervical facet joints [22,23]. In addition to facet joint injury, however, one study showed 100% of patients with chronic whiplash to have symptomatic TrPs that responded to treatment with resolution or significant improvement in their symptoms in 75% of cases (Gerwin and Dommerholt, Unpublished data).

Migraine headache can cause secondary MPS that can be symptomatic. Paroxysmal migraine that is intermittent and infrequent has less TrP-referred pain than chronic tension-type headache. In chronic headache syndromes, it becomes difficult if not impossible to tell which TrPs contributed to the onset of the headache, and which have occurred secondarily. In practice, they all have to be treated, begging the question.

Temporomandibular joint dysfunction always has a component of head and neck TrP pain, both directly due to the TrPs, and also by referred pain into the head and neck. Postural adjustments of the neck made in response to the facial muscle TrPs and the posterior displacement of the mandible contribute to increased muscle tension in the posterior cervical and suboccipital muscles. Treatment of the entire complex of jaw position and head and neck muscle TrPs, and of the meniscus in the temporomandibular joint when it is anteriorly displaced, will usually result in an improvement. Parafunctional behavior such as biting nails, chewing gum, grinding the teeth, or clenching the jaw all affect the head and neck muscles.

Frozen shoulder can be the result of a torn rotator cuff or of an impingement syndrome. The subscapularis muscle is invariably involved with TrPs that impair active or passive abduction and rotation of the shoulder. Inactivation of subscapularis muscle TrPs often improves range of motion. Attention to the myofascial component of this condition remains important during the postoperative rehabilitative phase of treatment.

Viscerosomatic pain syndromes with TrPs include shoulder pain referred from the gallbladder and from the liver, anginal-like chest pain in the postinfarction recovery period caused by TrPs, and flank pain from TrPs induced by ureterolithiasis. In each case, relief may be achieved by elimination of the TrPs. Confusion may occur when postinfarction chest pain is caused by TrPs, because new or recurrent ischemia is always an issue. However, when there is no sign of infarction, the pain may be secondary chest wall TrP pain. Shoulder pain may occur in conjunction with hepatic or diaphragmatic disease, such as cholecystitis, enlarging congenital hepatic cysts, or subdiaphragmatic abscess. Shoulder pain may actually improve with local TrP

therapy, and may or may not recur again, depending on the state of the underlying primary condition. Diagnostic right upper quadrant tenderness may develop after the regional MPS has formed and even after it has been treated successfully. Persistence of muscle wall hypersensitivity and of muscle TrPs may long outlast the acute illness and its resolution, as has been shown for kidney stones [24].

Radicular muscle pain may develop as the first sign of nerve root compression by disc herniation or by foraminal osteophytic nerve root compression. Signs of neurologic impairment may occur days or weeks later. Muscle pain from TrPs may occur at any time in the course of radiculopathy, whether cervical, lumbar, or the much less common thoracic radiculopathy. The diagnosis becomes clear when there is neurologic impairment, but the presence of an abnormal magnetic resonance scan showing disc bulging or herniation or foraminal hypertrophy with root impingement is not enough to make a definite causal relationship.

Postlaminectomy syndrome occurs as a result of recurrent root compression by disc herniation at the same or at a different level, by root compression from postoperative scar tissue, or from low back or pelvic-hip region MPS. The piriformis muscle is a common cause of this condition, and frequently overlooked although easy to treat. TrPs in the quadratus lumborum muscle, the deep paraspinal muscles (the multifidi), and the psoas muscle are also very common causes of persistent pain after laminectomy. TrPs in the gluteus medius refer pain down the leg in a dermatomal distribution (L5 or S1), and can be confused for recurrent radiculopathy. Other than the possibility of scar tissue formation, postoperative imaging of the spine shows no cause for the pain. Improvement is usually possible with treatment of the MPS, even when there is scar tissue present.

Hypothyroidism, parasitic disease, recurrent candidiasis, and vitamin B<sub>12</sub> deficiency have on occasion each been complicated by widespread muscle pain with myofascial TrPs. The MPS is usually widespread, and the patients therefore have frequently been diagnosed as having fibromyalgia. Symptomatic TrPs resolve to a great extent when these conditions are treated. Physical therapy may be needed to reach maximum improvement.

Connective tissue disorder frequently has complicating MPS. The importance of recognizing this is that the pain from the MPS can be treated symptomatically in addition to treating the underlying disorder, whether it be rheumatoid arthritis or osteoarthritis. Fibromyalgia has already been discussed. It remains a condition in which secondary myofascial TrPs are common, and can be treated to decrease the overall level of pain. In conclusion, MPS is classified as both a regional pain syndrome and a generalized pain syndrome of musculoskeletal tissue. It is both an acute disorder and a chronic disorder. It is a syndrome to the extent that it is a collection of symptoms, but it is a disease of muscle in the same way that neuropathy is a disease of nerve and arthritis is a disease of articular structures.

## Epidemiology

### Prevalence

The prevalence of MPS in the general population is unknown, in contrast to many studies that have attempted to ascertain the prevalence of fibromyalgia in populations around the world. The current estimate of the prevalence of fibromyalgia in the United States is 2%, with women affected four to seven times more frequently than men [10,25]. This figure may actually be too high, because the diagnosis is based on surveys and physical examination of those self-identified as having chronic, widespread pain. The physical examination was done to identify TrPs. As pointed out earlier in this article, such an examination overlooks the presence of myofascial TrPs that cause widespread pain, and misclassifies such cases as fibromyalgia rather than MPS.

A potentially confusing aspect of epidemiologic considerations is the separation of myofascial TrPs into active and inactive or latent [1••]. This is equivalent to distinguishing between active and inactive osteo- or rheumatoid arthritis, cardiac disease with or without congestive heart failure, or cholelithiasis with or without pain. The active and inactive or latent states are dynamic, the difference lying largely in the sensitivity of the TrP. An active TrP has a lower pressure pain threshold. The state of the TrP depends on a variety of factors, including the extent of physical activity as it does in both the arthritides and in heart disease, but not solely determined by these factors alone. The prevalence of inactive, or silent, clinical conditions is always greater than the prevalence of active disease, which always represents a subset of the total number of inactive and active cases combined. Few studies in myofascial pain make this distinction, and there are very few studies that look at the prevalence of clinically inactive TrPs.

One such study that specifically looked at the prevalence of inactive TrPs, or TrPs that did not necessarily cause pain, evaluated 200 unselected basic airmen (100 men and 100 women) who were examined for hypersensitive areas in the posterior shoulder muscles [26]. No indication is given that any other sign was evaluated than tenderness. The authors comment that "hypersensitive areas" were considered to be TrPs. Fifty-four percent of the women and 45% of the men had hypersensitive areas in these muscles. Referred pain was found in 12.5%, with no gender predominance. This article is often quoted as showing that the prevalence of latent TrPs in a young population is about 50%. In another group of 28 asymptomatic persons accumulated to date as controls for clinical studies of MPS (71% female, 29% male), 18% had inactive TrPs in the trapezius muscle, 11% had inactive TrPs in the anterior scalene, and only 4% had an inactive TrP in the sternocleidomastoid and in the levator scapulae muscle. Active TrPs reproducing pain experienced from time to time occurred in 14% of trapezius muscles, and 11% of levator scapulae muscles, with less in the remaining shoulder region and neck muscles (Gerwin, Unpublished data).

### Sex and gender differences

There is a gender predominance in fibromyalgia with women affected more than men. This difference between men and women is also seen for chronic widespread pain [27], a condition that is closely related to if not a subset of fibromyalgia as it is defined in the epidemiologic literature. As previously discussed, this group probably includes many subjects who have MPS, but were not identified as such because the screening examinations were generally limited to the identification of tenderness only. This difference is also seen in other conditions such as iron deficiency, in which there is a biological reason for the difference (menstruation), and in thyroiditis.

The preponderance of women compared to men in whiplash injuries is multifactorial, and includes the difference in the ratio of size and strength of the neck muscles to head mass as well as the greater exposure of women to urban driving. This difference is reflected in the increased ratio of women to men who suffer whiplash-associated MPS. These differences are both biological (sex-related) and psychosocial (gender-related). Differences in pain responses between men and women reflect both biological differences (as in iron deficiency) that can be said to be sex-related, and differences in cultural, psychological, and social factors that can be said to be gender-related [28].

Specific data are not available for the prevalence or incidence of MPS among women versus men, although some are available for specific conditions in which myofascial TrPs play a major role. Even in these cases, the results are not always clear. The condition of chronic backache is a case in point. Data are mixed, but in general, women tend to have a greater prevalence of back pain at younger ages than men, the difference vanishing in the older population. However, varying results in different studies do not show a uniform picture, and gender differences may not be as important as social and economic factors [29]. Tension-type headache and muscle tenderness is more common in women than in men [30], a matter of importance when considering the role of the myofascial TrP in the genesis of tension-type headache. The prevalence of temporomandibular disorder or craniomandibular pain is greater in women than in men across all age groups [31,32].

Differences between men and women in relationship to musculoskeletal pain have been recently reviewed [33]. The authors cited evidence that absences from work due to musculoskeletal pain in Sweden were greater for women than men, that women report more severe levels of pain than men, that female neonates express more pain behavior than male neonates, that pressure pain thresholds in women are lower than in men, and that certain painful conditions like temporomandibular joint disorders are more frequent in women taking estrogens. The authors cite both biological (sex) factors such as hormonal differences and differences in body size and muscle makeup (ratio of fast to slow twitch fibers), and psychosocial differences between men and women such as work demands, physical and mental stress at work and at home, social support, and other factors that are gender-based, rather than sex-based, differences.

Trigger points have been identified in a variety of painful conditions, and in some cases the prevalence of active TrPs has been reported. Not all such conditions are identified as MPS, but their description makes it clear that what is reported is a form of regional MPS. For example, one study of pectoral girdle myalgia in a cohort of 100 women showed that 96% had trapezius muscle tenderness in the horizontal and lower nuchal fibers, and 83% had tenderness in the occipital fibers of the trapezius [34]. Tenderness was related to breast weight and the marked indentation of the trapezius muscle by the brassiere strap. Relief was achieved by removing breast weight from the shoulders. Tenderness in the trapezius fibers under such conditions must be related to the presence of TrPs. In another study of 56 patients with chronic mixed tension/migraine headaches, 100% of the subjects had active TrPs, the trapezius muscle being involved in 93% of cases (Gerwin, Unpublished data). This is to be compared to the 18% of normal controls with active TrPs in the trapezius muscle cited above. A similar study of subjects with chronic cervical whiplash showed that 98% of subjects with symptoms lasting longer than 6 months had active trapezius TrPs that reproduced part of their pain picture.

One study that looked specifically at the prevalence of myofascial pain in a general internal medicine practice found that 31% had pain as a reason for their visit, and that 30% of these patients, or 9% of the total population evaluated, had MPS [35]. In a study of 283 consecutive admissions to a university comprehensive pain center, 85% were identified as having MPS as the primary cause of pain [36]. Referral biases to the center (referrals of musculoskeletal pain rather than other kinds of pain syndromes) may have contributed to such a high percentage of MPS among the patients.

### Musculoskeletal pain

A detailed review of the epidemiology of musculoskeletal pain [37] did not distinguish MPS among the causes of low back or other types of pain. Musculoskeletal pain at some time over the lifetime of the individual occurs in an estimated 70% to 80% of the population, but this includes acute as well as chronic cases. Chronic musculoskeletal pain was estimated to affect 10% to 20% of the population. The burden to the individual and to society comes from the population of chronic pain sufferers, not those with acute pain that recover in a short period of time. For example, only 6% of those with low back pain use half of the total health care resources [38••]. Nevertheless, one can only estimate that a substantial percentage of the individuals with chronic musculoskeletal pain may have a significant MPS on the basis of extrapolations of such data, as has been derived from the university comprehensive pain center cited previously [36].

In a general survey of pain conducted by telephone, headache was the most common cause of pain and low back pain was the second most common cause. Back pain

of more than 30 days duration was reported by 14% of the respondents and an annual prevalence of all back pain was 56% [39]. These figures have been confirmed in other population groups [40,41]. The cost of low back pain is considerable, amounting to a considerable percentage of the gross national product, although only a small percentage of the cost is actually consumed by direct health care, the rest being spent on the indirect costs of absenteeism and disability [38••,42]. The difficulty with these data, of course, is that the diagnosis of low back pain does not distinguish among the various causes, even though one might surmise that a muscular basis (*ie*, meaning myofascial pain) is the most likely cause, or at least accompanies other possible causes such as acute or chronic radiculopathy.

Lin *et al.* [43] studied work-related musculoskeletal disorders in Sao Paulo, Brazil. A total of 109 subjects were evaluated, 89.5% female, the mean age 39 years, and the mean duration of symptoms 36 months. A total of 94.5% had an MPS. TPs or TrPs (the distinction was not made for this statistic) occurred in the cervical muscles in 79.9%, in the trapezius muscles in 76%, in the supraspinatus muscle in 70%, and in the infraspinatus muscle in 56%. Wrist and finger flexors and extensors were involved with TPs or TrPs in 70% (extensors) and 79% (flexors) of the subjects. Treatment of the MPS by multidisciplinary techniques that included TP and TrP needling or injection, physical therapy, and psychological interventions resulted in a substantial improvement in pain (50% or greater reduction in the Visual Analogue Scale for pain) in 74% of subjects.

### Natural History of Myofascial Pain Syndrome

The myofascial TrP has two components: the taut band and the sensation of pain. The taut band is a motor abnormality, and the phenomenon of pain is a sensory consequence. The taut band can exist without pain, but pain from a TrP cannot exist without a taut band. The taut band is the first sign of the muscular response to biomechanical stress. The origin of the taut band is still not clear, but an abnormality of the motor end-plate complex of the myoneural junction, comprised of the synaptic terminal that releases acetylcholine, the synaptic cleft, and the postsynaptic muscle membrane, is likely. An excess release of acetylcholine is thought to be responsible for the development of abnormal end-plate noise that is associated with dysfunctional motor end plates [4]. Dysfunctional motor end plates are found in a disproportionate number in the myofascial taut band [44], and may be the underlying abnormality that results in the development of the taut band by causing focal areas of sarcolemmal shortening in the muscle fiber [7••]. Muscle contraction is the result of a calcium-dependent and energy-requiring sliding overlap of actin and myosin molecules in the muscle sarcomere. Separation of the actin and myosin molecules is also energy-dependent. Lengthening of the muscle requires the separation of

breaking of bonds between actin and myosin, and a sliding apart of the molecules. When sarcomeres are too densely contracted, there can be distortion and destruction of the myosin-titin complex at the Z-band, which can shorten the overall muscle fiber length. Restoration of the resting state of the muscle can still occur, however.

This synopsis of muscle contraction is meant to illustrate the dynamics of muscle contraction at the molecular level, and to show that the development of the taut band is based on the contraction of the muscle. A current hypothesis is that excessive release of acetylcholine from an abnormal motor end plate complex can lead to focal areas of intense sarcolemmal contraction, and that this might be the basis of the taut band. Such focal contraction does not necessarily mean that there is increased tension in the remainder of the muscle fiber, where the remaining sarcomeres must of necessity be elongated, but that the hardness that is felt as the taut band may represent the stiffness of the cytoskeleton of the muscle fiber. In any event, the development of the taut band is considered to be the first manifestation of the TrP, and the taut band is a structure that is dynamic, that can form, disappear (or recover), and form again, depending on the state of contraction and of the myoneural complex.

When the muscle is under sufficient mechanical stress, or the motor nerve is sufficiently stressed (*eg*, compressed), taut bands may reform, increase in number or in size, and result in the release of nociceptive substances (*eg*, bradykinin, substance P, potassium) that activate peripheral nociceptive nerve endings. A proposed mechanism for the release of these substances is the development of local muscle ischemia as a result of capillary compression by the taut bands. Activation of the peripheral nerve endings in turn activates the dorsal horn cell in the spinal cord. Rapid sensitization of the dorsal horn cell occurs, resulting in hypersensitivity and referred pain [1••,7••]. This is a reversible process that is responsive to the level of activity of the muscle. As muscle stress decreases, TrPs can decrease in number and can diminish in activity. As the level of stress or activity increases, taut bands increase in number and "irritability," and become tender to palpation, or spontaneously painful with activity. A very active TrP will be painful at rest. Thus, the boundary between latent and active TrPs is actually very fluid and dynamic, and TrPs will increase and decrease the amount of pain they produce, depending on the demands placed on the muscle and its ability to meet those demands.

### Acute myofascial pain syndrome

Acute MPS are more likely to be localized or regional, and to resolve spontaneously or with simple treatment (heat, alternating heat and cold, stretching, physical therapy, TrP needling, or injection with local anesthetic). However, often the problem is recurrent and intermittent, particularly if there is an underlying condition that is not correctable, such as a structural or environmental one. Once the

underlying condition is addressed and alleviated, the frequency of recurrences may diminish. For example, correction of a leg-length inequality-based scoliosis may reduce the frequency of recurrent low back pain or shoulder pain. However, in low back pain in which 95% of persons who stopped working because of back pain returned to work within 6 to 12 weeks, patients still had considerable pain 6 to 12 months after seeking medical care [38••]. Thus, even though statistics about return to work suggest that the majority of persons with acute low back pain have a limited disability and therefore a limited pain syndrome, the truth is that many of the persons with acute musculoskeletal pain syndromes develop chronic pain. How much this is related to MPS, undetected or uncorrected medical conditions, or to unidentified or uncorrected ergonomic work stresses, is unknown. Nevertheless, limited acute MPS syndromes do occur, especially in otherwise healthy individuals after an accident or injury. A typical case is that of a previously healthy young man who had a chest pain after a football scrimmage. His single muscle pectoralis major syndrome was readily treated by TrP injection and physical therapy, and never recurred.

### Chronic myofascial pain syndrome

Recurrent or chronic MPS tends to be more widespread than acute or recent onset syndromes. Spread of TrPs to involve other muscles and other regions occurs through alterations in the axial kinetic chain, through functional muscle units, and through satellite TrPs in referred pain zones.

Axial kinetic chain abnormalities are structural musculoskeletal stresses that develop up and down the skeletal chain. For example, shortened hamstring muscles cause the pelvis to go into posterior rotation. If the hamstring shortening is unilateral, pelvic rotation may be unilateral, causing pelvic torsion. Pelvic torsion will cause a leg-length inequality because the acetabulum is not centered in the innominate bone. The leg on the side of posterior rotation will act as functionally shorter and scoliosis will occur. Shortening of the psoas and quadratus lumborum will occur on the side of the pelvis that is higher. There will be increased tension and active shortening of the superficial paraspinal muscles on the side of the high hip, and increased tension and shortening of the trapezius, levator scapular, and scalene muscles on the contralateral side with the compensatory high shoulder. In this manner, a chronic hamstring muscle injury can cause neck and shoulder pain.

Functional muscle units are muscles that work together to exert a force vector or to stabilize a part, either as agonists or synergists, or as antagonists in opposition. When one muscle in a functional unit develops a TrP, it ceases to work effectively as it is weakened and loses the ability to lengthen, which allows normal range of motion about a joint. Other muscles in the functional unit must compensate for the weakness or impaired range of motion, and are thereby exposed to overuse, or are chronically shortened,

or both. These muscles are prone to develop TrPs. However, muscles commonly have two or more actions, and the functional muscle unit changes with the action of the muscle. TrPs can extend through affected muscles from one functional unit to another. For example, the trapezius muscle shares extension of the neck with the splenius capitis and semispinalis muscles, lateral bending of the neck with the scalenes, and shoulder elevation with the levator scapula. A TrP in the trapezius muscle can result in TrPs in all three of these muscles if the condition is chronic. Spread of TrPs through functional muscle units can turn a regional pain syndrome into a generalized pain problem affecting three or four of the body quadrants.

Satellite TrPs develop in the pain referral zone of primary TrPs. The satellite TrPs have all of the properties of TrPs, including a referral zone of their own. They may persist even after the primary TrP has been eliminated. They may also affect other muscles in their functional muscle unit.

Myofascial pain syndrome can persist for long periods of time, or wax and wane in intensity, but never go away entirely if not treated effectively. The average duration of chronic pain (6 months or longer) prior to being evaluated at one pain center was 63 months, the range being 6 to 180 months [45]. A total of 45% of patients with chronic MPS have three or four body quadrants affected (Gerwin, Unpublished data), belying the notion that MPS are regional. Chronic MPS is just as likely to be widespread as it is to be localized. Correcting underlying structural, postural, and ergonomic factors, and addressing medical conditions that impair muscle function like hypothyroidism, and treating the myofascial TrPs directly, can result in a cure or in significant improvement in a large percentage of patients. Significant improvement or complete elimination of pain was achieved in 75% of patients with myofascial syndromes associated with chronic cervical whiplash (Gerwin and Dommerholt, Unpublished data). This author has not seen the evolution or transformation of MPS into fibromyalgia, although 75% of patients with fibromyalgia may have significant MPS at one or more times during the course of their illness [45].

## Conclusions

Myofascial pain syndromes are muscle pain syndromes that are classified as musculoskeletal disorders. They have a defined pathophysiology that leads to the development of the characteristic taut or hard band in muscle that is tender and that refers pain to distant sites. MPS can be regional or generalized. If an MPS becomes chronic, it tends to generalize, but it does not become fibromyalgia. It can be classified both as a primary disorder without other medical illness, or as a secondary pain syndrome that occurs as a result of another process. MPS may persist long after the initiating event or condition has passed, but it is nonetheless a muscle disease that can be satisfactorily treated.

## References and Recommended Reading

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

- Of importance
- Of major importance

1. •• Simons DG, Travell JG, Simons LS: *Myofascial Pain and Dysfunction: The Trigger Point Manual*, vol 1, edn 2. Baltimore: Lippincott Williams & Wilkins; 1999.  
This is the major text in the field of myofascial pain. It details the principles of diagnosis and treatment, as well as outlines what is currently known about the nature of TrPs.
2. Carlsson CA, Nachemson A: *Neurophysiology of Back Pain: Current Knowledge in Neck and Back Pain: The Scientific Evidence of Causes, Diagnosis, and Treatment*. Edited by Nachemson A, Jonsson E. Philadelphia: Lippincott Williams & Wilkins; 2000:149–163.
3. Merskey H, Bogduk N: *Classification of Chronic Pain*, edn 2. Seattle: IASP Press; 1994.
4. Simons DG: **Do endplate noise and spikes arise from normal motor endplates?** *Am J Phys Med Rehabil* 2001, **80**:134–140.
5. Hubbard DR, Berkoff GM: **Myofascial trigger points show spontaneous needle EMG activity.** *Spine* 1993, **18**:1803–1807.
6. 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.
7. •• Mense S, Simons DG: *Muscle Pain*. Baltimore: Lippincott Williams & Wilkins; 2001.  
Currently the most up-to-date exposition of pain of muscle origin, written by the researcher who has done much of the work on the central and peripheral responses to muscle pain, and by the person who developed or guided the major advances in myofascial pain studies.
8. • Wolfe F, Smythe HA, Yunus MB, et al.: **The American College of Rheumatology criteria for the classification of Fibromyalgia.** *Arthritis Rheum* 1990, **33**:160–172.  
Presents the basis for the diagnosis of fibromyalgia, and is widely used. Intended to standardize research criteria for the diagnosis, it has been widely adopted for clinical use.
9. Rau C-L, Russell IJ: **Regional versus generalized muscle pain syndromes.** *Curr Rev Pain* 1999, **3**:85–95.
10. Russell IJ: **Fibromyalgia syndrome.** In *Muscle Pain*. Edited by Mense S, Simons DG. Baltimore: Lippincott Williams & Wilkins; 2001:289–337.
11. Saggini R, Giamberardino MA, Gatteschi L, Vecchiet L: **Myofascial pain syndrome of the peroneus longus: bio-mechanical approach.** *Clin J Pain* 1996, **12**:30–37.
12. Delacerada FG: **A comparative study of three methods of treatment for shoulder girdle myofascial syndrome.** *J Orthop Sports Phys Ther* 1982, **4**:51–54.
13. Zohn DA: **The quadratus lumborum: an unrecognized source of back pain, clinical and thermographic aspects.** *Orthop Rev* 1985, **14**:163–168.
14. Friction J: **Myofascial pain.** In *Fibromyalgia and Myofascial Pain Syndromes*. Edited by Masi AT. London: Bailliere Tindall; 1994:857–880.
15. Gerwin RD: **Myofascial pain and fibromyalgia: diagnosis and treatment.** *J Back Musculoskeletal Rehabil* 1998, **11**:175–181.
16. Gerwin RD: **Differential diagnosis of myofascial pain syndrome and fibromyalgia.** *J Musculoskeletal Pain* 1999, **7**:209–215.
17. Henriksson KG, Mense S: **Pain and nociception in fibromyalgia: clinical and neurobiological considerations on aetiology and pathogenesis.** *Pain Rev* 1994, **1**:245–260.
18. Mense S: **Descending antinociception and fibromyalgia.** *Z Rheumatol* 1998, **57**(suppl 2):23–26.
19. McDermid AJ, Rollman GB, McCain GA: **Generalized hypervigilance in fibromyalgia: evidence of perceptual amplification.** *Pain* 1996, **66**:133–144.
20. Russell IJ, Orr MD, Littman B, et al.: **Elevated cerebrospinal fluid levels of substance P in patients with the fibromyalgia syndrome.** *Arthritis Rheum* 1994, **37**:1593–1601.

21. Travell JG, Simons DG: *Myofascial Pain and Dysfunction: The Trigger Point Manual*, vol 2, edn 2. Baltimore: Williams & Wilkins; 1992.
  22. Barnsley L, Lord S, Bogduk N: **Whiplash injury.** *Pain* 1993, 58:283–307.
  23. Lord SM, Barnsley L, Wallis BJ, Bogduk N: **Chronic cervical zygapophysial joint pain after whiplash. A placebo-controlled prevalence study.** *Spine* 1996, 21:1737–1744.
  24. Vecchiet L, Giamberardino MA, eds: **Referred muscle pain and hyperalgesia from viscera.** In *Muscle Pain, Myofascial Pain, and Fibromyalgia: Recent Advances*. New York, NY: Haworth Medical Press; 1999:61–69.
  25. Wolfe F, Ross K, Anderson J, et al.: **The prevalence and characteristics of fibromyalgia in the general population.** *Arthritis Rheum* 1995, 38:19–28.
  26. Sola AE, Rodenberger ML, Getys BB: **Incidence of hyper-sensitive areas in posterior shoulder muscles.** *Am J Phys Med Rehabil* 1955, 34:585–590.
  27. Macfarlane GJ: **Fibromyalgia and chronic widespread pain.** In *Epidemiology of Pain*. Edited by Crombie IK et al. Seattle: IASP Press; 1999:113–123.
  28. Fillingim RB, ed: *Sex, Gender, and Pain. Progress in Pain Research and Management*, vol 17. Seattle: IASP Press; 2000.
  29. LeResche L: **Epidemiological perspectives on sex differences in pain.** In *Sex, Gender, and Pain*. Edited by Fillingim RB. Seattle: IASP Press; 2000:233–249.
  30. Jensen R, Rasmussen BK, Pedersen B, Olesen J: **Muscle tenderness and pressure pain thresholds in headache. A population study.** *Pain* 1993, 52:193–199.
  31. Goulet J-P, Lavigne GJ, Lund JP: **Jaw pain prevalence among French-speaking Canadians in Quebec and related symptoms of temporomandibular disorders.** *J Dent Res* 1995, 74:1738–1744.
  32. Drangsholt M, LeResche L: **Temporomandibular disorder pain.** In *Epidemiology of Pain*. Edited by Crombie IK et al. Seattle: IASP Press; 1999:203–233.
  33. Rollman GB, Lautenbacher S: **Sex differences in musculoskeletal pain.** *Clin J Pain* 2001, 17:20–24.
  34. Ryan EL: **Pectoral girdle myalgia in women: a 5-year study in a clinical setting.** *Clin J Pain* 2000, 16:298–303.
  35. Skootsky SA, Jaeger B, Oye RK: **Prevalence of myofascial pain in general internal medicine practice.** *West J Med* 1989, 151:157–160.
  36. Fishbain DA, Goldberg M, Meagher BR, et al.: **Male and female chronic pain patients categorized by DSM-III psychiatric diagnostic criteria.** *Pain* 1986, 26:181–197.
  37. Magni G: **The epidemiology of musculoskeletal pain.** In *Progress in Fibromyalgia and Myofascial Pain*. Edited by Voeroy H, Merskey H. Amsterdam: Elsevier; 1993:3–21.
  38. ●● Linton S: **The socioeconomic impact of chronic back pain: is anyone benefiting?** *Pain* 1998, 75:163–168.
- This editorial is a very complete accounting of the economic problems produced by chronic back pain. It serves well as a model for understanding the impact chronic pain has on society as well as on the individual.
39. Taylor H, Curran NM: *The Nuprin Pain Report*. New York: Louis Harris and Associates; 1985.
  40. Skovron ML, Szpalski M, Nordin M, et al.: **Sociocultural factors and back pain: a population-based study in Belgian adults.** *Spine* 1994, 19:129–137.
  41. Hillman M, Wright A, Rajaratnam G, et al.: **Prevalence of low back pain in the community: implications for service provision in Bradford, UK.** *J Epidemiol Community Health* 1996, 50:347–352.
  42. Gerwin RD: **Management of persons with chronic pain.** In *Management of Persons with Chronic Neurologic Illness*. Edited by Ozer MN. Boston, MA: Butterworth-Heinemann; 2000:265–290.
  43. Lin TY, Teixeria MJ, Fischer AA, et al.: **Work-related musculoskeletal disorders.** In *Myofascial Pain: Update in Diagnosis and Treatment*. Edited by Fischer AA. Philadelphia: W.B. Saunders Company; 1997:113–118.
  44. Simons DG, Hong C-Z, Simons L: **Prevalence of spontaneous electrical activity at trigger spots and control sites in rabbit muscle.** *J Musculoskel Pain* 1995, 3:35–48.
  45. Gerwin R: **A study of 96 subjects examined both for fibromyalgia and myofascial pain [abstract].** *J Musculoskel Pain* 1995, 3(suppl 1):121.