Diagnosis of Myofascial Pain Syndrome

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KEYWORDS

• Myofascial pain • Trigger points • Active trigger points • Latent trigger points • Muscle • Referred pain • Diagnosis

KEY POINTS

- Myofascial pain is a common condition that occurs as a primary source of pain as well as a comorbid pain with other conditions.
- The source of pain in myofascial pain is the myofascial trigger point that is a small region of hardness and tenderness in a taut band of muscle.
- Many of the pain syndromes are caused by pain referred from the trigger point region.
- The diagnosis of myofascial pain in the clinical setting is best made by palpation of the trigger point, moving in a cross-fiber direction perpendicular to the direction of the fibers.
- Evaluation of the patient must include an assessment of those factors that either predispose the patient to the development of myofascial pain or that are comorbid with it.

INTRODUCTION

Myofascial pain (MP) is a widespread and universal cause of soft tissue pain. Physicians commonly overlook this condition because of lack of awareness and training but it is a relatively simple diagnosis. The central feature of MP syndrome (MPS) is the myofascial trigger point (MTrP), a very small, localized area of muscle contraction that is hard to the touch, and that is very tender. The trigger point is always located on a discrete band of hardness located within a muscle. The diagnosis of MPS is made by palpation of the MTrP.

FEATURES OF THE MTRP

The MTrP is always located on a tight or taut band of muscle. An MTrP that causes pain is always tender to palpation. When stimulated mechanically by palpation or by

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needling, it contracts sharply, referred to as local twitch response (LTR). The taut band limits stretch of a muscle and produces weakness that is rapidly reversed as the trigger point is inactivated. It can activate autonomic activity, such as vasodilation or constriction, goose bumps, or piloerection (**Box 1**).

The MTrP, like other physical sources of chronic pain, refers pain to distant sites and leads to central nervous system sensitization. Central sensitization results in a lower pain threshold and in tenderness, and in an expansion of painful areas, including an increase in MTrPs. MTrPs can be spontaneously painful (so-called "active" MTrPs) or they can be nascent or quiescent (so-called "latent" MTrPs), inactive until physical activity converts them to active MTrPs.

Diagnosis

The diagnosis of MPS is based on a pertinent history and physical examination. Objective means of identifying the MTrP exist, but are generally not used in clinical practice because they are costly, time-consuming, and are not available to most practitioners. Now that high-definition ultrasound (HDUS) is more widely available, there is interest in using it to guide the practitioner in performing injection or deep dry needling of difficult muscles using HDUS guidance.[1] The experienced hand is faster and quite adequate at identifying the site to be needled.

History

MP can be acute pain or chronic muscle pain. The nature of the pain in both cases is dull, deep, aching, and poorly localized. It is rarely sharp and stabbing, although acute episodes of stabbing pain can occur, even on a background of chronic pain. It mimics radicular or visceral pain. Somatic pain from trigger points in the abdomen, for example, can feel like irritable bowel, bladder pain, or endometrial pain. Trigger points in the gluteus minimus muscle refer pain down the side and back of the leg, like L5 or S1 radicular pain. It can be accompanied by a sensory component of paresthesias or dyesthesias but does not present in this manner. Paresthesias, such as tingling, when present, are generally distributed in the dermatome of the nerve root(s) innervating the muscle harboring the relevant trigger point. Pain is often experienced as referred to other regions, such as the head, the neck, or the hip, as referred pain (RP). MP can also be the presenting symptom for radiculopathy, or major joint pain (shoulder or hip). MPS persists long after the initiating cause of pain has resolved. Hence, the story of a remote injury can be relevant.

Box 1
Features of the myofascial trigger point: the first 3 are essential for diagnosis; the last 5 are not required to make a diagnosis

1. Taut band within the muscle
2. Exquisite tenderness at a point on the taut band
3. Reproduction of the patient's pain
4. Local twitch response
5. Referred pain
6. Weakness
7. Restricted range of motion
8. Autonomic signs (skin warmth or erythema, tearing, piloerection [goose-bumps])

Thus, the onset of the pain, the regions involved, the timing and pace of progression, and the quality of the pain, are important elements of the history. In addition, there are certain predisposing factors that make MP more likely to occur. These include iron deficiency (most commonly caused by menstrual blood loss in women, but also from dietary insufficiency), hypothyroidism, and deficiency of vitamin D or B12. Lyme disease, hypermobility, and spondylosis also predispose to the development of MP. Parasitic infections can manifest as widespread trigger point pain. Questions about travel and backpacking are therefore relevant as well.

Physical Examination

The diagnosis of MPS is made by the identification of an MTrP and relating it to the patient's pain complaint (**Box 2**). A MTrP is identified by palpation. The MTrP is characterized by the presence of a taut band palpable within muscle. The taut band can be palpated in almost every muscle, with some initial guidance and practice. It is also tender when it is the immediate cause of the patient's pain. Thus, MTrP-containing muscle has a heterogeneous feel of hard and soft areas, rather than a homogeneous uniform consistency. The intense contraction of the trigger point results in a sensory phenomenon of localized, exquisite pain that is always associated with the taut band. Moreover, some taut bands are not painful to palpation, but have functional consequences, such as altering the normal sequence of muscle activation.[2] The taut band must be palpated cross-fiber, that is, perpendicular to the direction of the muscle fiber. The direction of muscle fiber may not be obvious, especially in pectoral muscles, the infraspinatus muscle, and the gluteal muscles.

Palpating the Taut Band

An MTrP is always palpated perpendicular to the direction of the muscle fiber so as to detect the taut band. Palpation overlying a firm or bony structure is palpated by compressing the muscle against it (**Fig. 1**). When the muscle can be grasped between the fingers, the muscle is palpated by a pincer grip with the thumb and the index and long fingers (**Fig. 2**). When a taut band is identified, the examining fingers move along the band to identify the small region of greatest hardness, the area of least compliance to compression. It is this area that is most tender. This is the center or heart of the trigger point. Stimulation of this area induces RP. It is in this area that mechanical stimulation elicits the LTR. The farther away stimulation is from this center, the more difficult it is to elicit RP and the LTR, until they cannot be elicited at all.[3] The LTR cannot be elicited at all when the taut band is stimulated 3 cm or more from the trigger point zone. The

Box 2
Procedure for identifying trigger points

1. History and pain diagram: the history identifies the areas affected by pain
2. Examination of muscles whose trigger points can refer pain to the affected areas
3. Palpate the muscle for taut bands, using either flat palpation or pincer palpation
4. Move the fingers along the taut band to find the hardest and most tender spot (the trigger point)
5. Compress the trigger point manually and ask (1) if the spot is tender or painful, and if so, (2) does the pain resemble the patient's usual pain
6. Compress the trigger point for 5–10 seconds and then ask if there is pain or some sensation away from the trigger point (referred pain)

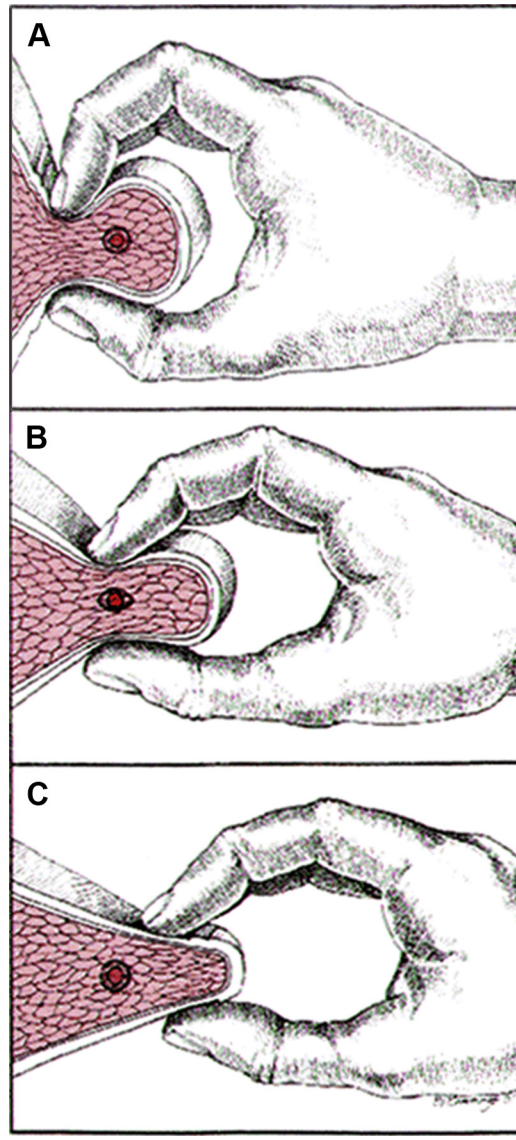

Fig. 1. (*A–C*) The palpating finger rolls over a trigger point taut band. Flat palpation compresses the trigger point between the skin and an underlying hard, bony structure. Palpation is always perpendicular to the direction of the fiber. (*From* Simons DG, Travell JG, Simons LS. Travell & Simons' Myofascial Pain and Dysfunction: The Trigger Point Manual, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 1996; with permission.)

importance of locating the area of greatest hardness in the taut band, which is the area of greatest tenderness, is that this is the area that is to be treated. Compression of the trigger zone for 5 to 10 seconds can induce referred pain, or pain that is at a distance from the point of stimulation because RP represents central activation or central sensitization. It requires activation of interneurons and spreads rostrally and caudally in the spinal cord, which does not occur instantaneously. Hence, 5 to 10 seconds of

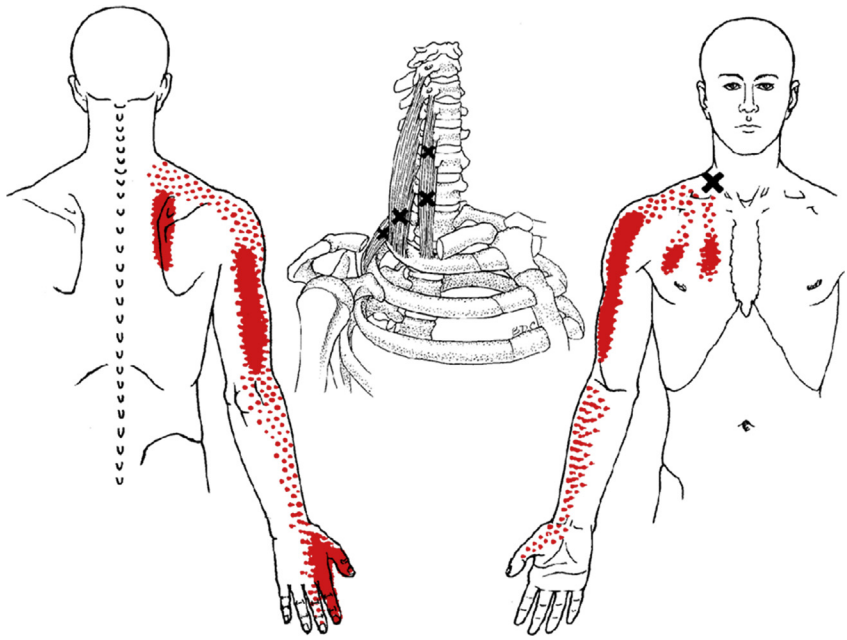

Fig. 2. An example of referred pain, in this case from trigger points in the anterior scalene muscle. (*From* Simons DG, Travell JG, Simons LS. Travell & Simons' Myofascial Pain and Dysfunction: The Trigger Point Manual, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 1996; with permission.)

compression is needed to be certain that the palpated MTrP induces RP. Once the MTrP is identified, determined to be tender, and RP is confirmed, the patient is asked if the pain or tenderness, local or referred, reproduces or is like all or part of his or her usual pain. That relates a particular MTrP to a patient's complaint. Identifying an MTrP that is related to the presenting or another complaint aids in determining which trigger points need to be treated and in what order.

A tender trigger point is an indication that there is hyperalgesia or allodynia. Pain at the MTrP is due to the release of neuropeptides, cytokines, and inflammatory substances, such as substance P, calcitonin gene–related peptide, interleukin-1α, and bradykinin,[4] and protons that create local acidity. Models for *acute* muscle pain have been developed and have yielded information about the generation of local and referred pain.[5] However, most clinically relevant muscular pain syndromes last far longer than the conditions studied in animals or humans under laboratory conditions. Therefore, there is great interest in studying longer-lasting and chronic pain in humans.

Taut Bands

A taut band does not need to be tender because MTrPs are dynamic, being quiescent (when they do not cause pain) or resolve with rest, massage, or stretch. They are activated with movement or action, including psychological stress. When they are quiescent, the MTrP does not reproduce pain (ie, spontaneously cause pain), but that was nonetheless tender to palpation. A taut band that is not tender to palpation will not, of course, reproduce pain unless it does so by causing RP. Such taut bands restrict movement because taut bands are not innocuous with deleterious functional effects.

Lucas and colleagues[2] have shown a latent MTrP disrupts the normal sequence of muscle activation. They can activate central effects, such as decreasing the threshold for pain activation distally.[6,7] They limit muscle lengthening and have a role in activating other MTrPs. Hence, determination of treatment is to be made to treating taut bands, whether latent or not. The decision requires a judgment about whether a taut band is clinically relevant or not. This is not always clear and, therefore, the taut band may be treated more often than not.

Additional Trigger Point Characteristics

LTR is elicited by mechanical stimulation of the taut band causing local contraction. This is differentiated from a Golgi tendon reflex, which involves contraction of an entire muscle in response to stretch. The LTR is a brief (25–250 ms), high-amplitude, polyphasic electrical discharge. The discharge is attenuated when the stimulation is remote from the trigger zone. The twitch response is dependent on an intact spinal cord reflex arc. Severing the peripheral nerve completely abolishes the local twitch response, whereas transecting the spinal cord does not abolish the twitch response.[8] Thus, the local twitch response is mediated through the spinal cord, and is not affected by supraspinal influences. The twitch response is unique to the MTrP and is not seen in healthy muscle.

Referred Pain

RP represents spread through a central nervous system that has been activated (ie, spread that is facilitated by central sensitization).[5] It is most common in the distribution of the nerve innervating the muscle with the trigger point being activated. Thus, a trigger point arising in the infraspinatus muscle, primarily innervated by the C5 nerve root, tends to refer pain to other C5-innervated muscles, with a spillover to C4 and C6 innervated muscles. The primary referral patterns reflect the arborization and spread of incoming first-order neuronal axons within the spinal cord. The nociceptive spread or arborization of incoming axons is far greater than that of touch and position sense. Hence, there is the potential for greater spread of pain than for perception of touch. Moreover, as incoming nociceptive impulses become more intense and the duration of central nociceptive neurons becomes longer, central sensitization results in a greater extent of increased synaptic efficacy through more distant spinal segments, a result of the neuroplastic changes that accompany central sensitization and RP encompasses ever far-reaching parts of the body. RP can be felt over many spinal cord segments, and in extreme cases can appear to be bizarre and body wide. MTrP points in the ventral (anterior) trunk muscles can even refer to the dorsal (posterior or back) muscles.

Limited Range of Motion

Limited range of motion (ROM) is due to pain on lengthening a muscle harboring an MTrP and to the limitations imposed by the shortened taut band. ROM testing can be misleading because of the potential multiple pathologies that can limit motion about a joint, and, additionally, normal ROM is variable depending on the individual, such as hypermobile individuals.

Examination of ROM can be a useful clue in determining which muscles harbor trigger points. Limited rotation of the head and neck to the left can implicate the left sternocleidomastoid and/or trapezius muscles, or the right splenius cervicis and oblique capitis inferior muscles, all muscles that have to lengthen to accomplish this movement. An additional limitation of head and neck side bending right would focus attention on the left sternocleidomastoid and trapezius muscles.

WEAKNESS

Weakness is often but not always evident in a muscle harboring a trigger point. Weakness in affected muscles is rapidly reversed as the trigger point is inactivated. Hence, weakness does not represent a neuropathic or myopathic process, but appears to be a form of muscle inhibition that may be central. This phenomenon has not been well studied.

AUTONOMIC CHANGES

Vascular dilation and constriction occur as a result of autonomic nervous system activation, resulting in erythema or blanching, and warm or cool areas usually in the distribution of the nerve innervating an affected muscle.

Diagnostic Criteria

There is some debate in the literature as to what is needed to diagnose an MTrP (and hence, diagnose MPS). Articles often state that the diagnosis of MP is made using the criteria of Simons and colleagues.[9] Simons and colleagues[9] described at least 7 features of the trigger point: (1) taut band, (2) exquisite tenderness on the taut band, (3) reproduction of pain, (4) local twitch response, (5) restricted ROM, (6) autonomic symptoms, and (7) RP. To this is often added a nodular hardness at the trigger point. Not all features described are present at any one time, and not all are necessary to identify a trigger point and make a diagnosis. This question has not been answered definitively at this time, but the presence of a taut band that is tender and that reproduces the patient's pain complaint in full or in part is sufficient to base a treatment program. These criteria, a tender, taut band that reproduces the patient's pain, allow the clinician to select a trigger point for treatment. The proof of efficacy is that treatment based on these criteria alone is sufficient to reduce or eliminate pain, which is our experience, although there has not been a study confirming this.

One can ask if identification of a taut band is enough to make a diagnosis. However, to diagnose a pain syndrome, one must have pain, so that it makes sense that tenderness or pain must be elicited by examination so as to diagnosis MPS. However, a nontender taut band should be treated in a patient with trigger point pain syndrome when it is likely to have significant clinical effects, like restricting motion or producing weakness.

There are a number of studies that have shown inter-rater reliability of the physical examination of an MTrP, starting with the study by Gerwin and colleagues.[10] Subsequent studies were more sophisticated and showed that clinicians could agree on the identification of the same trigger point, not just the muscle(s) that harbored MTrPs. Sciotti and colleagues[11] showed that examiners could independently identify the same taut band region. Interrater reliability of MTrP palpation in shoulder muscles was demonstrated by Bron and colleagues.[12]

A number of reviews have been published questioning the data and purporting to show that physical examination is not reliable, but these reviews show a fundamental lack of understanding of the anatomy and physiology of the MTrP, which biases the author's conclusions. For example, one such review discounted a number of positive studies because the investigators did not specify that they looked for a "nodule" in the taut band, a feature they said was mentioned as essential by MP "experts."[13] In fact, "all" the experts they were referring to turned out to be Dr David Simons. Simons[9] was referring to the area of tender hardness on the taut band. One has to identify the taut band and to elicit tenderness, but there is no need to identify the region as nodular rather than linear to make a diagnosis. Simons never made a point that identifying a

nodularity in the taut band was necessary for identifying a trigger point. The MTrP may simply be described as a sense of swelling, but often all that the palpating finger feels is hardness on the taut band.

Diagnostic Inactivation of the Trigger Point

An active MTrP is symptomatic and can be assessed for a particular complaint of pain, like headache pain or shoulder or low-back pain. Reproduction of the patient's pain complaint is important in determining if a particular trigger point is clinically significant. When there is doubt about the clinical significance of a particular trigger point, it can be inactivated either manually, by using a laser, by deep dry needling, or by a trigger point injection. An immediate (with 2–3 minutes) unequivocal decrease in pain is good evidence that the MTrP in question is clinically relevant. Sometimes the relief of pain can be dramatic, as in piriformis syndrome, and readily leads to an effective treatment plan (**Box 3**).

OBJECTIVE IDENTIFICATION OF TRIGGER POINTS

Identification of the taut band is now possible with a number of objective techniques. The taut band and the twitch response can be visualized by ultrasound.[14–16] Newer ultrasound devices produce high-resolution images of the taut band, and may be

Box 3
Case history: myofascial syndrome or pinched nerve?

History: A 56-year-old woman complained of pain in the right leg 6 months after lumbar spine surgery to remove a herniated disc and decompress an S1 nerve root. She had continued pain after surgery with no period of decreased pain. Pain was down the outside of the leg to the dorsum of the foot. Her pain was the same after surgery as it was before surgery. Her preoperative imaging studies showed a right-sided L5–S1disc herniation. The postoperative study showed the same herniated disc findings.

Examination: She could not bear weight on the right leg. She came down the hall on a walker, not stepping on the right leg. She had moderately full low-back flexion, limited extension. She had to use her hands to lift the right leg onto the examination table. She had full straight leg raising with pain on the right side, but only to the buttock, not felt in the back; normal 2+ knee and ankle reflexes; there was no sensory loss. Right leg strength could not be assessed because of pain. Examination of the right buttock musculature showed linear hardness and tenderness (increased resistance to palpation) over the right piriformis muscle.

Diagnostic test: A diagnostic inactivation of the piriformis muscle with a trigger point injection using lidocaine 0.25% resulted in a marked decrease in pain within 1–2 minutes. She was able to get off the examination table on her own and walked out of the office without assistance. Pain relief lasted 30 minutes.

Outcome: She was treated with deep dry needling of the piriformis trigger point twice weekly for 6 weeks, by which time she was free of pain, ambulating normally. There was no recurrence for the remaining 10 years of her life.

Comment: The diagnosis of piriformis muscle trigger point was suspected by the history. That she did not have any improvement after surgery suggests that the trigger point was symptomatic at that time. The referral of pain down the leg in a sciatic nerve distribution is consistent with entrapment of the sciatic nerve, peroneal branch, otherwise known as a piriformis muscle syndrome. The dramatic reversal of pain with release of the trigger point by lidocaine injection was highly suggested of a piriformis syndrome. The outcome after treatment confirmed the diagnosis. It is not possible to tell if a component of her pain came from an S1 nerve root compression that was relieved by surgery.

useful in future research studies of the MTrP. MTrPs can also be identified by magnetic resonance imaging.

Magnetic Resonance Elastography

Magnetic resonance elastography is a new technique that can differentiate tissues of varying densities. The technique involves using phase contrast to identify tissue distortion when cyclic energy waves like vibration are introduced into the muscle. Shear waves travel more rapidly in stiffer tissues. The harder taut band can be distinguished from the surrounding normal muscle by this technique.[17,18] MR elastography will likely emerge as an effective tool for the identification of the MTrP's taut band.

Ultrasound

The combination of vibration sonoelastography with ultrasound imaging localizes hypoechoic, elliptical, focal areas that corresponded to a palpable trigger point nodule in the trapezius muscle.[15] The trigger point taut band can be imaged in latent and active trigger points. A nonsymptomatic trigger point has not been systematically sought, although changes in "normal muscle" that may represent such taut bands may be seen. A reversal of capillary flow that most likely represents shunting of blood away from an area of trigger point–induced vascular compression has also been shown by ultrasound sonoelastography.[19]

Thus, there are a number of ways in which trigger points can be imaged objectively. The practical application of these approaches is just beginning to be explored, like ultrasound guidance of the needle when treating a deep muscle like the psoas muscle. Computerized tomography and electromyographic localization of the trigger point are presently available for guidance in trigger point needling, but rarely used. Ultrasound localization of the trigger point for needling guidance is presently being evaluated, and may be useful for muscles that are difficult to palpate. These techniques have already confirmed beyond a doubt the existence of the MTrP that previously was identifiable only by palpation.

ELECTROMYOGRAPHY

Hubbard and Berkoff[20] described a signature electromyographic signal associated with the trigger point as a persistent, low-amplitude, high-frequency discharge found at the MTrP region in active MTrP. This activity, which initially came to be known as spontaneous electrical activity (SEA), is associated with the MTrP region.[21,22] As the recording electrode is moved away from the trigger zone, the SEA diminishes. Likewise, the SEA diminishes as the needle is placed outside the taut band.[3] A needle placed 1 cm away from the trigger zone and outside the taut band does not display SEA.[20]

The electrical activity associated with the trigger point most likely arises from the motor endplate,[9] and has been named endplate noise by Simons.[23] There has been controversy about the nature of this electrical activity, but the low-amplitude, constant discharges are consistent with the small, monophasic negative waveform of less than 50 μV called miniature endplate potentials (MEPPs). The higher-amplitude waveforms seen only in active trigger point zones are consistent with endplate spikes.[24] MEPPs are thought to be the result of spontaneous release of acetylcholine from motor nerve potentials. Botulinum toxin reduces the endplate noise in rabbit myofascial trigger points, supporting the postulated role of acetylcholine release from the motor nerve terminal in the generation of endplate noise.[25] The low-amplitude activity is thought to be the result of the release of acetylcholine sufficient only to generate subthreshold endplate depolarization in close proximity to the electrode. The endplate spike of

several hundred microvolts' amplitude represents temporal summation of MEPP sufficient to reach or exceed the membrane threshold value.[24] The studies by Hubbard and Berkoff,[20] and subsequently others, compared the spontaneous electrical activity at the trigger zone with a control point a centimeter or so away from the index electrode. The absence of electrical activity at the control electrode confirms the lack of anterior horn cell motor action potentials and therefore establishes the resting state of the muscle. Endplate noise is 5 times more frequent in endplate zones in the trigger point than in endplate regions outside the trigger point zone and the taut band.[21]

Endplate noise intensity is directly correlated with the degree of trigger point irritability as measured by pressure pain threshold.[26] Thus, motor endplate activity is greater in active, spontaneously painful trigger points, than in latent trigger points. Greater endplate activity and consequently greater focal muscle sarcomere compression can be thought of as being associated with greater local muscle injury and local release of nociceptive substances.

Hence, endplate noise is now well described as an electrical phenomenon unique to the trigger point. Nevertheless, electromyography is not used for the clinical identification of trigger points because it is not readily available to many clinicians treating trigger points, it is costly, and it is a far less efficient way to identify MTrPs than palpation. One can examine the entire body for trigger points in minutes, something that is not feasible by any laboratory means of detecting MTrPs.

LABORATORY

Laboratory studies are not used to make a diagnosis of MPS or of the MTrP because there is no laboratory or imaging study that has clinical utility in diagnosis. Rather, laboratory studies are used to identify conditions that seem to be associated with MPS. The most common conditions that are thought to predispose to, and perpetuate, MTrPs, are iron deficiency, hypothyroidism, vitamin D deficiency, vitamin B12 deficiency, parasitic infestation, and recurrent vaginal yeast infections. A list of symptoms that should make one think of underlying disease and the tests that are commonly used are found in **Table 1**.

MULTIPLE DEFICIENCY STATES

If 2 or more nutritional (iron, vitamin D, vitamin B12) deficiency states are present or if one is present, but does not respond to supplementation, malabsorption syndromes must be considered. Antitissue transglutaminase (anti-TTG) immunoglobulin (Ig)A antibodies are present and increased in 90% of cases of malabsorption syndrome. Serum IgA is also measured, as 3% of the population has little to no IgA. In that case, one must specify that the anti-TTG antibodies should be IgG antibodies. In a few cases, patients have had normal anti-TTG antibodies, but responded to a gluten-free diet by eliminating their pain symptoms (Gerwin RD, unpublished data, 2013).

If iron deficiency is found, the cause must be identified. Iron supplementation alone is not an adequate approach. Iron deficiency is generally seen in only women, as a result of menstrual blood loss with insufficient dietary iron intake. Male iron insufficiency is uncommon. There must be a specific reason found, like excessive use of nonsteroidal anti-inflammatory drugs causing gastritis, or cancer.

DIFFERENTIAL DIAGNOSIS

The diagnosis of MPS with trigger point tenderness and referred pain is enough to initiate treatment at a symptomatic level. However, the diagnosis of MPS is only the

Table 1
Commonly used laboratory tests in the evaluation of myofascial pain syndromes

Condition	Symptoms	Test	Threshold
Hypothyroidism	1. Diffuse muscle pain 2. Widespread MTrPs 3. Deep coldness 4. Fatigue 5. Constipation	1. TSH 2. Serum cholesterol	TSH >2.25 μIU/mL
Iron insufficiency	1. Diffuse muscle pain 2. Widespread MTrPs 3. Fatigue 4. Deep coldness	1. Serum ferritin 2. Serum iron, IBC, percent saturation 3. Hemoglobin, hct, differential count	1. Ferritin <25 ng/mL 2. Low serum iron, high IBC, saturation <18% 3. hct <28, MCH and MCHC low
Vitamin D insufficiency	1. Diffuse pain 2. Widespread MTrPs 3. Fatigue 4. Weakness	1. 25-OH vitamin D 2. PTH if vitamin D is very low	1. 25-OH vitamin D <30 ng/mL 2. PTH if 25-OH vitamin D <18 ng/mL
Vitamin B12	1. Diffuse pain 2. Weakness 3. Impaired vibration and position sense in the great toes	1. Vitamin B12 level 2. CBC	1. Serum vitamin B12 level <350 pg/mL 2. Macrocytic anemia
Parasitic infestation	1. Diffuse myalgia 2. Widespread MTrPs 3. Gastrointestinal symptoms	1. 3 stools on different days for ova and parasites	Stool positive for ova and/or parasites
Candida	1. Recurrent vaginal itching and discharge 2. Widespread pain 3. Diffuse muscle tenderness	1. History of recurrent episodes of vaginal candida 2. History of repeated use of antibiotics	Vaginal examination may or may not be positive for candida

Abbreviations: CBC, complete blood count; hct, hematocrit; IBC, iron-binding capacity; MTrP, myofascial trigger point; PTH, parathyroid hormone; TSH, thyroid-stimulating hormone.

beginning of the diagnostic and treatment process. Additional steps are necessary to fully address a patient's pain. The cause of the MP must be explored by history, physical examination, and by laboratory testing. Thus, the evaluation of a patient with apparent MPS must consider those conditions that have a similar presentation and that constitute the differential diagnosis of MPS (Table 2).

LOOK-ALIKES

One problem associated with the diagnosis of MP is that referred pain from MTrPs can be similar to referred pain from other conditions and from pain generated directly by other conditions. For example, one of the first signs of radiculopathy can be the development of MTrPs that precede the appearance of signs of neurologic impairment. Trigger point pain can mimic radicular pain. The clinician must be aware of these 2 possibilities. Referred pain that radiates into upper and lower limbs from muscle trigger points also typically affects the neck or back, the shoulder, or hip region. Thus, the pain pattern from the MTrP is no different from radicular pain from nerve

Table 2
Differential diagnosis of regional pain syndromes

Pain Region	Examine for Signs and Symptoms of Regional Disorders	Muscles with Trigger Point Referred Pain Patterns that Reproduce the Regional Pains in Column 2
Head and neck	Headache features (laterality, character of headache [sharp, stabbing, dull, throbbing, etc.] and clinical feature as dizziness, photophobia, and phonophobia); the presence of neurologic signs (weakness, absent tendon reflexes, sensory loss). Range of motion of neck, loading tests for facet joints, imaging for spondylosis and instability	Upper trapezius, levator scapulae, posterior cervical (splenius capitis and cervicis, semispinalis, and oblique capitis inferior), sternocleidomastoid, facial muscles (masseter, temporalis)
Shoulder	Shoulder and acromioclavicular joint dysfunction signs, shoulder impingement, and rotator cuff syndrome signs	Trapezius, supraspinatus, levator scapulae, supraspinatus, infraspinatus, posterior serratus superior, rhomboids, subscapularis, teres major and minor, latissimus dorsi, deltoid, pectoralis major and minor
(Noncardiac) Chest	History and signs of tracheobronchial and esophageal disease, including carcinoma, cardiac disease, especially angina	Pectoralis major, abdominal obliques rectus femoris, back muscles
Low back	Spondylo-arthropathies, spondylolisthesis, disc disease, spinal stenosis, myelopathies (cord compression, tethered cord), hypermobility syndromes	Psoas, quadratus lumborum paraspinal muscles (iliocostalis, longimus thoracis, multifidi), abdominal oblique, rectus femoris
Pelvic/hip	Internal organ disease: painful bladder, irritable bowel, endometriosis, menstrual cramps, prostatitis, vulvovaginitis, carcinoma, radicular pain from the lumbo-sacral spine	Abdominal muscles, psoas, quadratus lumborum, gluteal muscles, including the piriformis muscle, thigh adductors, including the pectineus muscle, hamstrings, especially the upper semitendinosis muscle, the short extensor muscles of the thigh (obturators, gemellae)
Knee	Intrinsic knee joint disease, radicular pain from the low back	Quadriceps muscle (especially the vastus medialis) for medial knee pain, vastus lateralis for lateral knee pain, hamstrings and gastrocnemius muscles for back of knee pain
Ankle/foot	Intrinsic joint pain, radicular pain from the low back	Anterior and posterior leg muscles, (gastrocnemius, soleus, fibularis, anterior tibialis, long flexor and extensor muscles of the leg), intrinsic foot muscles

root compression and is similar to the pain referral from facet joint arthropathy in that it often has an axial component and referral down a limb. In fact, the problem is more complicated because either or both conditions can occur as comorbidities with MTrP pain (see the following section, Comorbidities).

COMORBIDITIES

MTrPs can occur in association with a large number of other clinical conditions, both conditions then occurring as comorbidities (**Box 4**). This applies even if it is thought that the MTrP is a result of another condition. Increasing awareness of mild hypothyroidism with thyroid-stimulating hormone levels as low as 2.25 μIU/mL has led to the identification of individuals with widespread trigger point pain that resolves with the administration of thyroid hormone. Awareness of the coexistence of MTrPs with internal organ disease can uncover such diverse etiologies as cancer, infection (parasitic and Lyme disease are 2 outstanding examples), acute and chronic radiculopathy, nerve entrapment, endometriosis, painful bladder syndrome, and Ehlers-Danlos syndrome, to name some comorbid conditions. Common comorbid conditions are listed in **Box 4**. In some cases, trigger points may persist along with the conditions that may have led to their creation. This is particularly true of spinal disorders, such as radicular syndromes and facet arthropathies (see the preceding section, Differential diagnosis). The astute clinician will pursue the underlying cause or predisposing conditions that need to be addressed for a successful outcome. Patients who experience relief from their pain when treated by such an astute clinician are eternally grateful.

Box 4
Common conditions that are comorbid with myofascial pain syndrome

1. Migraine headache
2. Tension-type headache
3. Temporomandibular joint disorder
4. Fibromyalgia
5. Hypermobility syndromes
6. Painful bladder syndrome
7. Irritable bowel syndrome
8. Pelvic pain syndrome
9. Vulvovaginitis
10. Prostatitis
11. Endometriosis
12. Dysmenorrhea
13. Hypothyroidism
14. Vitamin D deficiency
15. Vitamin B12 deficiency
16. Iron deficiency
17. Parasitic infection
18. Celiac disease of malabsorption

In some cases, MTrPs can produce a well-described clinical condition, such as migraine or chronic tension-type headache, and then coexist with it as a comorbid condition.[27,28] In this case, elimination of the trigger point may be essential to resolving the headache if it is the primary trigger for the headache.

Rounded shoulders and forward head posture asymmetrically shorten the trapezius and sternocleidomastoid muscles, creating a mechanical dysfunction that promotes the development of trigger points in these muscles.

SUMMARY

MTrP is common and readily identified by a trained examiner. The diagnosis is made by suspecting the possibility of MPS from the history then confirmed by identifying the MTrP by physical examination. Provocative factors and factors that may perpetuate the condition are investigated. Comorbid conditions are identified and treated. Thus, the evaluation of the patient is comprehensive and is not limited simply to identification of a specific MTrP causing a specific pain. A comprehensive evaluation is necessary to reach a diagnosis and develop a successful comprehensive treatment plan.

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