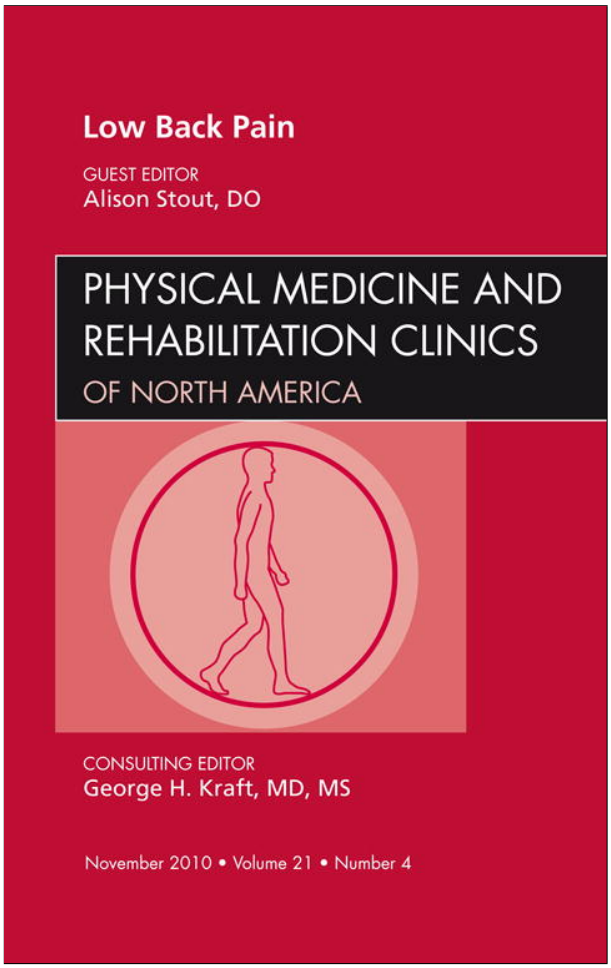

Myofascial Low Back Pain: A Review

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KEYWORDS

• Myofascial pain • Low back pain • Fibromyalgia
• Trigger points

Myofascial syndrome is a common nonarticular local musculoskeletal pain syndrome caused by myofascial trigger points (MTrPs) located at muscle, fascia, or tendinous insertions. Myofascial syndrome affects up to 95% of people with chronic pain disorders[1] and has also been found to be the principal cause of pain in 85% of patients attending a pain center.[1,2] As many as 9 million people in the United States suffer with myofascial pain.[3] Initially described in the 16th century by the French physician de Baillou (who named this regional pain syndrome muscular rheumatism), this condition has received several terms throughout the years including idiopathic myalgia, regional fibromyalgia, and regional soft tissue pain, among others. It was not until the 1950s that Travell and Rinzler referred to these muscle pain patterns as myofascial pain.

Myofascial pain syndrome is characterized by the presence of trigger points, which are hyperirritable tender spots in palpable tense bands of skeletal muscles. Trigger points can be either active, which are tender and spontaneously painful, or latent, which are tender but not spontaneously painful. Snapping palpation of the taut bands may produce a transient contraction of a group of muscle fibers referred as the local twitch response. Local twitch response is caused by activation of local Ia afferents and consequent reflex response of α motor neurons, which indicates the presence of muscle spindles.[4] A patient vocalization or withdrawal from palpation when exquisite tenderness is perceived is referred as the jump sign.[5] Clinically, myofascial pain syndrome can present as painful restricted range of motion, stiffness, referred pain patterns, and autonomic dysfunction.

PATHOPHYSIOLOGY

Several hypotheses proposed have been a topic of debate for the last several years. At present the most accepted theory is the Integrated Trigger Point Hypothesis

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described by Simons.[2,5–8] Simons'[8] integrated hypothesis proposes that a sequence of events including an "energy crisis" of the muscle fibers will cause sustained sarcomere contracture. Decreased levels of adenosine triphosphate caused by reduced blood flow renders the muscle fibers with insufficient energy to return calcium to the sarcoplasmic reticulum, resulting in a rigor state where these muscle fibers are unable to relax. This situation leads to increased metabolic demands, resulting in local temporary hypoxia and the release of noxious histochemicals, which may account for the pain associated with the active MTrPs.[5,6,9]

It has also been hypothesized that the reason for this sarcomere shortening is secondary to an increase in miniature endplate potentials and excessive acetylcholine release, the reason why botulinum toxin may be effective in the treatment of MTrPs.[6,7,10] Besides the mechanism of excessive release of acetylcholine leading to abnormal depolarization, other mechanisms include upregulation of nicotinic acetylcholine-receptor activity as well as genetic or acquired defects of the L-type and N-type voltage-gated Ca^{2+} channel.[7] Excessive calcium release at the sarcoplasmic reticulum through a dysfunctional Ryanidine receptor calcium channel may also cause sustained muscle contraction.[11]

Simons' theory was supported by Shah and colleagues[9] when they measured the levels of biochemical substances at active and latent MTrPs at the upper trapezius and compared them to uninvolved sites at the gastrocnemius muscle. These biochemical substances are associated with the pain, muscle soreness, and inflammation in the soft tissue sites. The selected inflammatory mediators include neuropeptides, cytokines, and catecholamines, and also noted is a decrease in pH. These chemical substances activate the different nociceptors located at muscle, fascia, and joints, and are responsible for the pain associated with the myofascial pain syndrome. Specific substances found by Shah and Gilliams[6] in their study of microdialysis sampling of the trapezius include substance P, calcitonin gene-related peptide, serotonin, norepinephrine, prostaglandins, bradykinins, tumor necrosis factor α, interleukin (IL)-6, IL-8, and IL-1β. The acidic environment secondary to ischemia and local hypoxia inhibits acetylcholinesterase, resulting in an excess of acetylcholine, and activates nociceptors that promote hyperalgesia.[12,13]

The vasodilatory effects of several of the biochemicals released contribute to increased pain at the active trigger point.[6,9] Furthermore, Partanen and colleagues[4] suggested in 2010 that postural stresses and sustained overload of the muscle will cause inflammation of the muscle spindles, resulting in activation and sensitization of intrafusal III and IV afferents.

The pain experienced by these patients may be severe only with minimal palpation, and they may appear as if they are overreacting. This pain response is felt to be secondary to hyperalgesia as a result of sensitization.[4,6] Many of the endogenous substances mentioned may cause peripheral sensitization of nociceptors, which decrease their pain threshold in the peripheral receptors and cause a normally nonpainful stimulus to elicit pain.

ETIOLOGY

There are many factors that have been proposed to result in the development and persistence of MTrP pain. These factors include anatomic abnormalities, various postural habits, vocational activities causing excessive strain on a particular muscle, tendon, or ligament, endocrine dysfunctions, psychological stressors, sleep disorders, and lack of exercise.[14–16]

Mechanical

Postural habits contribute to the development of myofascial pain by causing excessive overload on specific muscle groups, the quadratus lumborum being the most commonly involved.[5] For example, leg crossing will cause the hemipelvis to rise, approximating the iliac crest to the 12th rib, and cause shortening of the ipsilateral quadratus lumborum. A common sleeping position such as lying on one's side with the uppermost leg in adduction will also cause shortening of the quadratus lumborum, and these patients will typically complain that their pain is worse at night.[17] Anatomic considerations include leg length inequality, short arms, and a small hemipelvis. Leg length discrepancy will cause excessive lumbar lordosis and excessive stress at the quadratus lumborum. The compensatory (functional) scoliosis produced by the quadratus lumborum is a necessary lumbar curvature needed to maintain balance, leading to overloading of this muscle. Patients with short arms can be identified by evaluating if the elbows do not reach the iliac crest. When seated, these patients will tend to slump forward or lean to one side of the chair, to be able to place their elbows at the armrest, resulting in excessive strain on the quadratus lumborum and posterior cervical paraspinal muscles.[5] One can also see shoulder tilt to accommodate the spinal curvature and chronic muscle contraction to bring the spine back to midline, which will eventually lead to trigger points.[16]

Medical

Besides mechanical causes of myofascial type pain such as structural, postural, or ergonomic; others include hormonal dysfunction, enzyme deficiencies, immunologic causes, infectious diseases, and nutritional deficiencies. Plotnikof and Quigley[16,18] found that 89% of subjects with chronic musculoskeletal pain had low levels of vitamin D. Deficiency of this vitamin has been associated with musculoskeletal pain, loss of type II fibers, and proximal muscle atrophy. Vitamin B12 and iron deficiency have also been linked to chronic pain, presenting with symptoms such as muscle pain, chronic fatigue, tiredness, and poor endurance. Iron is necessary for the generation of energy through the cytochrome oxidase system, and a deficiency of accessible iron in muscle will result in "energy crisis."[16] Other vitamins such as vitamins C, B1, and B6 have also been associated with diffuse mylagia.[16,18]

Endocrine disorders include hypothyroidism and growth hormone deficiency. Special considerations have been made with hypothyroidism in view of that it promotes a hypometabolic state thought to promote trigger point formation.[16] Low levels of thyroid hormones will affect cellular metabolism, resulting in an inadequate supply of energy for muscle contraction.[16] The same principle of active muscle contraction secondary to inadequate recovery of calcium by the sarcoplasmic reticulum is also seen in McArdle disease. This genetic myophosphorylase deficiency will affect glycolytic metabolism in muscle and will lead to lack of calcium recovery.[19] Finally, infections that have been linked to myofascial pain include chronic Lyme disease, chronic mycoplasma infections, hepatitis C, and enteroviruses.

Assessment

Identification of MTrPs is almost entirely based on history and physical examination. The patient will usually present with a chronic history of localized or regional pain, with resisted range of motion of the muscles involved. It is essential to identify from the history if the muscle pain is more focal as opposed to generalized or widespread. A focal myalgia would suggest mechanical or structural factors as the cause of pain, whereas in a widespread myalgia, laboratory tests are necessary to identify metabolic,

hormonal, or nutritional disorders, or fibromyalgia as the reason for the musculoskeletal pain syndrome.[16] There are also a series of diagrams that the patient can use to identify his or her pain pattern, for a better assessment of widespread versus localized pain.

Physical examination should begin inspecting for postural imbalances, gait, pelvic symmetry, shoulder tilt, leg length discrepancies, and for compensatory functional scoliosis. Evaluating tightness at the hip flexors and hamstrings should be part of the physical examination, as tightness in these muscle groups will promote forward pelvic tilt and an increase in lumbar lordosis, which will result in excessive strain at extensor muscles.[5] Palpation is the most important component of the physical examination to assess for the presence of MTrPs. It is essential to identify if the tender points on palpation produce referred pain patterns or just local tenderness, which is the main difference between trigger points and tender spots. A systematic review in 2009 by Lucas and colleagues[20] on the reliability of physical examination for the diagnosis or trigger points demonstrated that due to a lack of studies and interobserver reliability, physical examination cannot currently be recommended as a reliable test for the diagnosis of trigger points.[2,20–22]

When evaluating patients with suspected myofascial low back pain, muscles that may have trigger points include the iliocostalis lumborum, longissimus thoracis, multifidus, quadratus lumborum, and gluteus medius.[23] Travell and Simons[5] suggest that the quadratus lumborum and gluteus medius are the most frequently involved. Adequate assessment of the quadratus lumborum will require that the patient be lying on his or her side with the uppermost arm abducted above the head with knees bent. Palpation of the gluteus medius should be at the upper lateral quadrant of the buttocks when the patient is lying prone. In a prospective study by Njoo and Van der Does,[22] it was determined that the clinical usefulness of trigger points is increased when localized tenderness and the presence of either the jump sign or patient's recognition of his pain pattern are used as criteria for the presence of trigger points in these muscles.

Also, adequate screening for stress and anxiety is important in patients with widespread musculoskeletal pain. Severe depression, anxiety, and fear avoidance behavior are predominantly associated with patients with low back pain and widespread musculoskeletal pain as compared with patients who are pain free. Other psychosocial factors to consider include low income, early psychological stressors, gender, job satisfaction, and history of musculoskeletal pain in family members.[23]

MYOFASCIAL PAIN VERSUS FIBROMYALGIA

Common differential diagnoses of low back pain include mechanical, sacroiliac joint, discogenic or zygapophysial joint pain; a thorough physical examination will help rule out most of these. When considering myofascial low back pain as the cause of the patient's complaint; special attention has to be made to fibromyalgia, which is also a chronic noninflammatory muscle pain syndrome. Many questions have risen over the years regarding the diagnosis of fibromyalgia, and several have doubted its existence.

Fibromyalgia is a syndrome characterized by chronic widespread muscle tenderness as a result of widespread sensitization. Fibromyalgia may be accompanied by fatigue, sleep disturbances, mood disturbances, depression, and visceral pain syndromes.[16]

The current American College of Rheumatology (ACR) criteria include spontaneous pain present for over 3 months, pain in all 4 quadrants of the body (above and below the waist, right and left of midline) and pain on digital palpation on 11 out of 18 tender

points. Without an adequate physical examination one might confuse myofascial pain syndrome with fibromyalgia. Myofascial pain syndrome is the most common condition that must be considered in the differential diagnosis of fibromyalgia and can also present as widespread myalgia. As stated in a review by Gerwin in 2005 about myofascial pain syndrome and fibromyalgia; "many cases of fibromyalgia are in fact cases of myofascial pain syndrome that have been misdiagnosed as a result of poor muscle palpation techniques that miss the presence of taut bands and referred pain." The main difference between the two is the referral of pain produced when palpating trigger points as compared with tender spots.

In previous studies more than 10 active trigger points were found in more than half of fibromyalgia patients,[24] and active trigger points were found in about 18% of examinations in the predetermined tender points of fibromyalgia.[25] A study by Ge and colleagues[26] in 2009 evaluated if the predetermined sites of examination for tender points in fibromyalgia were frequently associated with MTrPs. Thirty women diagnosed with fibromyalgia as per the ACR criteria were chosen for the study. All of the 18 predetermined tender points were manually palpated and examined with intramuscular needle electromyographic (EMG) examination, as one would expect to see spontaneous electric activity in both active and latent trigger points. The 2 sites bilaterally at the second rib were not included in the EMG examination of thin patients, to avoid any complications. In this study more than 90% of the predetermined tender point sites were either active or latent MTrPs, as evaluated by manual palpation and confirmed by needle EMG registration of spontaneous electrical activity. In conclusion, this study demonstrated that positive tender points at predetermined sites were mostly clinically active and latent trigger points at these predetermined sites, which mimicked fibromyalgia pain.[26]

A new diagnostic tool named the Symptom Intensity Scale (SIS) has been developed for the diagnosis of fibromyalgia. This tool has been used to both diagnose and establish severity of fibromyalgia, without the need to count tender points. The scale consists of 2 parts: a regional pain score, which is the number of anatomic areas out of possible 19 in which the patient feels pain, and a fatigue visual analog scale whereby the patient makes a mark somewhere along a 10-cm line to indicate how tired they are. The SIS has been shown to be an accurate measure for general health, depression, and disability. Although still not recognized by the ACR, several investigators state that it will probably replace the current diagnostic criteria and that tender spots will no longer have to be counted.[27]

DIAGNOSTIC CRITERIA FOR MYOFASCIAL PAIN SYNDROME

Travell and Simons are identified as the principal founders of the diagnostic criteria of myofascial pain. Their proposed criteria include tender spots in a taut band, predicted pain referral pattern, patient pain recognition on tender point palpation, limited range of motion, and the local twitch response. A literature review from 2007 examined the variability of criteria used to diagnose MTrP pain syndrome. The criteria most commonly used by researchers and expert clinicians include all of the previously mentioned by Travell and Simons, except the local twitch response, which has not shown to be a reliable diagnostic test.[28] When comparing the frequency of the commonly used criteria, identifying a tender spot in a taut band is used in 65% of cases, and had been suggested by Travell and Simons to be the most sensitive and specific of all the diagnostic criteria. The frequency of the criteria used include patient pain recognition 53%, predicted pain referral pattern 44%, local twitch response 44%, and limited range of motion 22%. There is still a lack of evidence demonstrating the

reliability of these maneuvers. Further research is needed to test the sensitivity, specificity, and reliability of the current diagnostic criteria.[2]

Other minor criteria proposed include the jump sign, muscle weakness, autonomic responses, reduced skin resistance, pressure algometry readings, patients' being able to identify their trigger points, and alleviation of symptoms by stretch. The combination of the criteria used has been inconsistent but the combination proposed by Simons is still the most commonly used. The most recent modifications of the diagnostic criteria include tender spot in a taut band, patient pain recognition, and painful limitation to range of motion.

The local twitch response and predicted pain referral pattern are no longer considered as part of the diagnosis. Other investigators have suggested alleviation of the pain by infiltration of a local anesthetic and pressure algometry readings as part of the diagnosis, but this has not been adopted by many.[2] In a 2009 review of the reliability of physical examination for the diagnosis of MTrPs, firm digital pressure and the patient's feedback on the pain experience are considered the best indicators of the presence of trigger points. In this same review it was concluded that no study to date has reported the reliability of trigger point diagnosis according to the currently proposed criteria in symptomatic patients.[20] Ongoing microdialysis and EMG studies will continue to define and validate the current proposed criteria. There is still poor agreement among investigators as to the most appropriate diagnostic criteria; only recently have interrater reliability studies been reported.[29]

It is well known that the pressure algometer is used by manual medicine practitioners to determine the pressure pain threshold of specific muscles, joints, tendons, ligaments, and bones. The pressure algometer measures the force in pounds or kilograms required to produce pain, and has become useful to quantify pain and track recovery.[30] It is a hand-held instrument with a 1-cm^2 surface area plunger attached to a dynamic force gauge that may be used to assess sensitivity to pressure near a trigger point. Some studies have found high validity with an excellent inter- and intrarater reliability, but algometry is more commonly used in a research setting than clinical practice.[31–33] Other studies have demonstrated that the pressure algometer may have limited validity on determining pressure pain thresholds. Recently a new muscle pain detection device (MPDD) has been developed for identification of trigger points that will also distinguish between primary and referred muscle pain. This new device elicits contractions in muscles in an attempt to identify the muscle pain generator (see the next section for more details).

DIAGNOSTIC EVALUATION

There are no laboratory tests or diagnostic images that can serve as a gold standard for trigger point identification. Physical examination may also be unreliable to adequately diagnose MTrPs. New diagnostic tools such as ultrasonography (US) and magnetic resonance elastography (MRE) have shown promising results to identify and differentiate MTrPs from normal surrounding tissue. US has recently been used to identify trigger points because of its ability to characterize viscoelastic properties of myofascial tissue and identify high resistance arterial flow at trigger point sites.

A study by Sidkar and colleagues[34] evaluated 10 patients with trigger points, as per the diagnostic criteria of Travell and Simons. Active trigger points, latent and normal tissue, were labeled after being identified by physical examination. US was performed on each of the sites by blinded physicians, as well as vibration sonoelastography (VSE). VSE uses external vibration to localize areas of stiffer tissue. On US 2-dimensional (2D) gray imaging, trigger points at the upper trapezius, which were identified

previously by physical examination, appeared as focal dark (hypoechoic) elliptically shaped areas with heterogeneous echotexture. The palpated sites that were labeled as normal appeared as isoechoic with homogeneous echotexture.

Examination of the active trigger points with Doppler flow waveforms revealed abnormalities within the vasculature at active trigger points and adjacent tissue. Doppler flow at the active trigger point sites revealed an increase in vascular resistance as compared with latent and normal tissue sites. This high resistance is secondary to the sustained contracture at these active trigger point sites. US VSE was able to identify areas of stiffer tissue in view of that these areas of stiffer tissue vibrated with lower amplitude as compared with surrounding normal tissue; vibration amplitudes were 27% lower on average.[34] In conclusion, US can be used to identify MTrPs, and VSE can differentiate these sites from normal surrounding tissue and quantify the relative stiffness based on vibration amplitudes.

MRE is another diagnostic tool that can be used to evaluate for MTrPs. MRE was initially developed at the Mayo Clinic and was used primarily to diagnose liver fibrosis by measuring liver stiffness.[35] As initially described by Muthupillai in 1995, it is an noninvasive MR-based contrast imaging technique that applies an oscillating motion to detect tissue vibratory displacements that have been introduced into a tissue by an external source of shear vibration. MRE basically works by measuring the wavelengths of the vibrations sent through tissues. Shear waves travel more rapidly in stiffer tissue and hence display a longer wavelength. Myofascial taut bands, which have higher stiffness as compared with surrounding muscle fibers, will result in longer wavelengths.[36,37] Although MRE is able to identify the difference in wave propagation patterns in a taut band as compared with normal myofascial tissue, the MTrP was not identified in the taut band. 2D US combined with VSE may be a better diagnostic tool than MRE because it can localize MTrPs, provides better mechanical and physical properties of trigger points and surrounding muscle tissue, and is more cost effective.[38]

The MPDD is an electrical device that elicits contractions in muscles in an attempt to identify the muscle that is thought to cause the pain. If the muscle stimulated produces pain, it is believed that this muscle is the pain generator, as compared with normal muscle, where the patient will experience an involuntary painless contraction. This device uses current produced through an aluminum head that moves through the skin and causes the muscle to contract. There are 3 possible diagnostic outcomes when using the MPDD: no pain with the stimulus, pain that disappears with repeated stimulation, or pain that persists with repeated stimulation. When the stimulus does not produce pain, the muscle contracted is not considered the pain generator. Pain that disappears with continuous stimulation is thought to be secondary to tension or spasms, and may be reversible with conservative methods. Finally, pain that persists with a repeated stimulus is caused by histologic changes, such as trigger points within the muscle that produce painful contractions.[39]

In a randomized control study in 2010 by Hunter and colleagues,[39] the effectiveness of MPDD versus manual pressure was evaluated, based on the outcome of standardized injections to muscles identified by each technique. Subjects were injected at several sites identified by a blinded physician by either manual pressure or MPDD. The sample size included 40 patients with a minimum 3-month history of back or neck pain. Painful sites were identified in half of the patients by manual pressure and half with the MPDD. There were 45 muscles identified by manual pressure that were determined to benefit from injection and 46 muscles selected for injection by MPDD. When compared with MPDD, of the 45 muscles identified with manual pressure 24% had no pain, in 42% the pain disappeared with repeated stimulus, and

only 15 muscles were identified that would benefit from injection. After treatment with 2 mL of 1% lidocaine injection, the MPDD group reported statistically significant improvements from baseline in pain, mood, and disability scores at 1 week and 1 month. Although relatively new, the MPDD demonstrates promising results in identifying muscle pain generators, for better precision of injection, as compared with manual pressure palpation.

TREATMENT

Multiple modalities and approaches have been used for the treatment of MTrPs with varying degrees of success. These methods include manual therapies such as pharmacologic management, physical therapy modalities, myofascial release, injection therapies, dry needling, and alternative medicine treatments such as acupuncture. The most common and effective treatment options used in daily clinical practice as well as other alternative treatment options such as acupuncture and herbal medications are presented in this section. The most important concept overall is treating the underlying etiologic pathology responsible for trigger point activation—far more important than treating the actual trigger point(s). In many cases, the active MTrP will automatically inactivate as soon as the underlying pathology is adequately treated.[40] Important mention has to be made of several of the principles proposed by Hong concerning inactivation of trigger points. First of all, the trigger point being targeted should be confirmed as the pain generator. This targeting might be difficult because there may be many adjacent latent trigger points that will be painful on palpation. Identification of active versus chronic trigger points should also be assessed. Furthermore, the active trigger point should not be inactivated in the acute stage because it will subsequently disappear on its own once the acute lesion is adequately treated, although inactivation might be considered if the pain is severe or intolerable. When the etiologic lesion cannot be targeted locally because of severe pain from the active MTrPs, treatment of inactive distal (satellite) trigger points can be considered. This action may result in decreased sensitivity of the surrounding muscle tissue, and will later allow for treatment of the acute lesion and active trigger points. Finally, for optimal results, perpetuating factors should be avoided. The patient should be educated on proper posture, home exercises, and self-care techniques. Special considerations have to be made to underlying anatomic abnormalities that contribute to the persistence of myofascial pain, such as limb length discrepancies and compensatory functional scoliosis.

Manual therapy is one of the most common treatment options for myofascial pain. Manual therapy is often the first line of treatment before going on to other more invasive techniques such as injections or needling. Manual therapy will mostly consist of myofascial release, deep pressure massage, osteopathic manipulative treatment, and a popular technique called spray and stretch. This technique was initially described by Simons, and consists of passively stretching the targeted muscle and simultaneously applying a vaporized cooling liquid spray such as Fluori-Methane or ethyl chloride. This temporary anesthesia allows the muscle to be stretched passively toward normal length and helps inactivate the trigger point.[3,41]

Postisometric relaxation is another effective technique whereby the patient is asked to contract the involved muscle 10% to 25% of maximum, followed by relaxation and stretching.[40] Deep digital pressure (ischemic compression) is no longer recommended by Simons in the most recent edition of the trigger point manual, in view that it theoretically contributed to additional local ischemia. The best approach is by applying

a "press and stretch" technique, which is believed to restore abnormally contracted sarcomeres to their normal resting length.[7]

Pharmacologic therapy, transcutaneous electrical nerve stimulation, and thermotherapy are useful only for controlling pain symptoms. Pharmacotherapy includes muscle relaxants, nonsteroidal anti-inflammatory drugs (NSAIDs), analgesics, and tricyclic antidepressants such as amitriptyline. The latter are recommended in myofascial pain characterized by sleep disturbances.[5,42] Muscle relaxants include clonazepam, whose primary mechanism of action is enhancing GABAergic inhibition, and cyclobenzaprine, which is a centrally acting serotonin receptor antagonist that suppresses muscle spasms without interfering with muscle function. Leite and colleagues[43] have demonstrated cyclobenzaprine to be slightly superior when compared with clonazepam. Topical NSAIDs have been used in pain control for acute soft tissue damage, although their effectiveness in treating myofascial pain has not been fully studied. A randomized, double-blind, placebo-controlled study demonstrated that a 1-week treatment with a diclofenac patch produced significantly greater pain reduction and earlier mobilization of the involved muscles when compared with placebo (menthol patch). However, it did not affect pain threshold on MTrPs.[44]

US, iontophoresis, phontophoresis, and high-voltage galvanic stimulation are used by many professionals, but none of these techniques are supported by scientific evidence regarding their efficacy in eliminating MTrPs.[42] Of all available treatment options for MTrPs, the best evidence exists for trigger point injection and dry needling.[41] The main goal of treatment is to inactivate the trigger point and loosen the taut band.[45] The indications for a trigger point injection is clinical localization of active trigger points in patients with chronic low back pain with myofascial pain syndrome, who have failed to respond to medications and/or a course of active physical therapy, or when a joint is mechanically locked.[46,47] When the decision is made to proceed with injection or needling, there are several precautions to keep in mind. Contraindications to trigger point injections include bleeding disorders, anticoagulation, local infection, aspirin ingestion within 3 days of injection, and acute muscle trauma.[3,5] Bleeding tendencies will result in increased capillary hemorrhage, which will contribute to postinjection soreness.[5] If possible, the patient should be placed prone or supine to decrease the risk of vasovagal depression. Other complications include local muscle necrosis if corticosteroid is the substance being injected, pneumothorax, cervical epidural abscess, and intrathecal injection. Using botulinum toxin within trigger points may result in excessive weakness, flu-like symptoms, and transient numbness of the ipsilateral limb, and has not been shown to be superior to injection with Marcaine.[48–50]

Lidocaine is a frequently used anesthetic, although there are many different types of anesthetics or injection solutions that can be used. Procaine is also a preferred anesthetic because of its short action and minimal systemic toxicity with the absence of local irritation. Procaine also has the distinction of being the least myotoxic of all injectable anesthetics.[3] Other anesthetics cited in the literature for the treatment of MTrPs include prilocaine, mepivacaine, bupivacaine, levobupivacaine, and ropivacaine. A study in 2000 suggested that ropivacaine 0.5% was better tolerated than dry needling, bupivacaine 0.5%, bupivacaine plus dexamethasone, or ropivacaine plus dexamethasone. Corticosteroids should NOT be included as part of treatment, as there is no evidence of added benefit and local muscle necrosis is a potential risk.

Needle size is frequently 25- or 27-gauge, but a needle as large as 21-gauge has been reported. Thick subcutaneous muscles such as the gluteus maximus or paraspinals will usually require a 21-gauge, 2.0-in needle.[5] Needle length may be dependent on the

depth of the muscle through subcutaneous tissue, but is reported from 0.75 to 2.5 in. The 21-gauge, 2.5-in needle will be required to reach deep muscle such as the gluteus minimus and quadratus lumborum.[3] The number of trigger points injected varies, as does the volume of solution injected. Volume may depend on the pharmacologic dosing limits of the injected mixture and the total number of trigger points. Common clinical practice is to use 0.5 to 2 mL per trigger point, which may be injected in one location of maximal tenderness or an angular array of sites. The technique recommended by Hong and Hsueh was modified from Travell and Simons. This approach described holding the syringe in the dominant hand while palpating the trigger with the thumb or index finger of the opposite hand. Needle insertion was into the subcutaneous tissue adjacent to the trigger point at a 50° to 70° angle to the skin, aiming at the taut band. Multiple insertions in different locations in different directions from the subcutaneous layer were "fast in" and "fast out" to probe for latent triggers; this technique kept a straight track of needle insertion and avoids the possibility of muscle fiber damage.[51] Each thrust coincided with the injection of 0.02 to 0.05 mL of injectate to a total of 0.5 to 1 mL in each trigger point. Compression of the point for 2 minutes allowed hemostasis, which was followed by stretching of the muscle. Some studies have emphasized that stretching the muscle after the injection will increase efficacy of treatment. Travell and Simons recommended full active range of motion of the muscles injected so that they could reach their fully shortened and lengthened position.

Hameroff and colleagues[52] compared injection of bupivacaine 0.5%, etidacaine 1%, and saline into triggers in the neck and low back. Anesthetic improved pain and activities of daily living at 1 week better than saline, but there was no difference in the type of anesthetic. Malanga and Wolff[53] compared mepivacaine 0.5% with saline in acute low back pain, and found no significant difference in pain resolution at 2 weeks. One quality trial by Garvey and colleagues[54] showed no difference between dry needling, injection of lidocaine, lidocaine plus steroid, or vapocoolant spray with acupressure in the short term. A double-blind controlled study by Hong and Simons[55] compared lidocaine injection versus dry needling for MTrP treatment. These investigators noted that lidocaine injection and dry needling were equally successful in treating MTrPs. 0.5% lidocaine was preferred over dry needling because it reduced the intensity and duration of postinjection soreness. It was also concluded that the best response to injection and immediate relief was found when the "local twitch response" was provoked by impaling the active point; little treatment effect was noted if no local twitch response during needling or lidocaine injection was elicited. Relief after injection or needling lasted for a short period of time and then returned gradually. Persistence or recurrence of pain might have been secondary to underlying pathologic lesions that had not been addressed, such as intervertebral disk lesions.[51,55]

A randomized controlled study similar to that done by Hong in 1994[51] was carried out by Ay and colleagues[45] in 2009. These investigators compared the efficacy of anesthetic injection and dry needling methods for myofascial pain syndrome, and significant improvement in pain relief was obtained with both. With dry needling, additional pain was not significant and was found to be as effective as local anesthetics in the inactivation of trigger points. Both of these injection methods were effective by causing local mechanical disruption; this leads to relaxation of taut bands, decreased pain, increased local blood flow, and improved range of motion, and causes fibrotic scar formation on trigger points.

In highly resistant trigger points botulinum toxin can be used, but its high cost does not support its use. Other investigators recommend the use of botulinum toxin in patients with chronic myofascial pain resistant to physical therapy including dry

needling and oral pharmacotherapy over at least 1 month.[56] Advantages include presynaptic block of acetylcholine release from motor nerve endings, which will promote prolonged muscle relaxation for 3 to 4 months' duration.[57,58] Some studies have demonstrated the analgesic and antinociceptive effects of botulinum toxin in animal pain models.[59] Despite the theoretical advantages of botulinum toxin, a randomized, double-blind, placebo-controlled study by Ferrante and colleagues[60] did not demonstrate significant differences compared with placebo with respect to pain scores, pain thresholds with pressure algometry, or use of rescue medication. There is still concern regarding excessive muscle weakness. For example, weakening neck flexors without simultaneously weakening neck extensors can lead to postural abnormalities and increase pain.[60]

There are several alternative treatment options for myofascial pain syndrome such as acupuncture, chiropractic manipulation, and herbal medications. A recent review of the published evidence for the treatment of myofascial pain demonstrated acceptable evidentiary support by common chiropractic techniques for the treatment of myofascial pain.[61] Acupuncture's analgesic effects may be mediated by pain perception block by gate control theory whereby a pain input may be inhibited by another sensory input such as needling, elevating opioid peptides in the central nervous system, and noxious inhibitory control. Dry needling itself causes this same effect.[62] A controlled randomized trial demonstrated significant differences between the effects of trigger point acupuncture and sham acupuncture on pain and function in patients suffering from chronic low back pain.[63] Another new approach to treating trigger points is through the use of herbal medications. Some of the natural medicines that may be used include lavender, rosemary, passionflower, lemon balm, and marijuana, all of which contain the compound linalool. This compound inhibits end plate activity by reducing acetylcholine release and by modifying nicotinic acetylcholine receptors.[64]

SUMMARY

Myofascial pain is a common process resulting from a variety of causes. The diagnosis is usually made clinically, although there are recent advances in imaging that will allow for better research and may have future clinical benefits. The underlying cause (often related to muscular imbalances) should be assessed by a comprehensive physical examination and should be treated by the practitioner using a comprehensive rehabilitation program. Additional treatment options include pharmacologic, needling with or without anesthetic agents or nerve stimulation, and alternative medicine treatments such as massage and herbal medicines. Repeated trigger point injections should be avoided and corticosteroids should not be injected into trigger points. Ongoing research will continue to expand our knowledge concerning the treatment and management of myofascial pain, and provide new views concerning etiology and diagnosis.

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