Myofascial Pain
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Myofascial pain is defined as pain that originates from myofascial trigger points in skeletal muscle. It is prevalent in regional musculoskeletal pain syndromes, either alone or in combination with other pain generators. The appropriate evaluation and management of myofascial pain is an important part of musculoskeletal rehabilitation of regional axial and limb pain syndromes. This article reviews the current hypotheses regarding the pathophysiology of myofascial trigger points and muscle pain. A critical evidence-based review of the pharmacologic, nonpharmacologic, alternative medicine, and exercise treatments of myofascial pain is provided, as well as future research directions.

Overall Learning Objective: To review critically the state of the art knowledge of myofascial pain, including pathophysiology and comprehensive management. Areas of future research are identified.

Key Words: Injections; Muscles; Myofascial pain syndromes; Pain; Rehabilitation; Trigger points, myofascial.

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Traditionally defined, myofascial pain is pain that derives from myofascial trigger points, which are small, highly sensitive areas in muscle that are characterized by hypersensitive, palpable, taut bands of muscle that are painful to palpation, reproduce the patient's symptoms, and cause referred pain. More broadly, many authors, researchers, and pain clinicians speak of muscle pain, sensitization, tenderness, and muscle tension. One example is the commonly described pericranial muscle activity of chronic tension headache. This overlap among clinical syndromes and imprecision in clinical criteria often confuses the interpretation and generalizability of clinical trials in myofascial pain. It is, nonetheless, clinically important and relevant to take a broad view as we endeavor to understand the complex pathophysiology and choose rational treatments for patients with chronic myofascial pain.

By comparison, fibromyalgia is a widespread chronic pain disorder that is characterized by widespread muscle pain, fatigue, sleep disturbance, and 18 paired tender points in a widespread distribution. Although there may be clinical overlap between fibromyalgia and myofascial pain, this report will focus exclusively on myofascial pain.

Epidemiology
Myofascial pain has a high prevalence among individuals with regional pain complaints. The prevalence varies from 21% of patients seen in a general orthopedic clinic, to 30% of general medical clinic patients with regional pain, to as high as 85% to 93% of patients presenting to specialty pain management centers.

An understanding of the pathologic significance of trigger points in symptomatic individuals requires data on the prevalence of trigger points in the general asymptomatic population. A latent trigger point is defined as a trigger point that is tender to palpation and may be associated with restricted range of motion (ROM) and stiffness but is not associated with spontaneous complaints of pain. Active trigger points are those that are associated with a clinical pain complaint. The few studies available suggest that latent trigger points may be present in as many as 45% to 55% of asymptomatic young adults in the shoulder-girdle muscles and in 5% to 45% of lumbar gluteal muscles.

Clinical Characteristics
Patient Presentation and Symptoms
The characteristic symptoms of myofascial pain may begin after a discrete trauma or injury or may be of insidious onset. Patients note localized or regional deep aching sensations, which can vary in intensity from mild to severe. The myofascial trigger points (MTrPs) of each muscle have their own characteristic pain pattern; thus, the distribution of pain can help identify which muscles may contain the responsible MTrPs. Frequently, associated autonomic dysfunction may occur, including abnormal sweating, lacrimation, dermal flushing, and vasomotor and temperature changes. Cervical myofascial pain may be associated with neuro-otologic symptoms including imbalance, dizziness, and tinnitus. Functional complaints include decreased work tolerance, impaired muscle coordination, stiff joints, fatigue, and weakness. Other associated neurologic symptoms include paresthesias, numbness, blurred vision, twitches, and trembling. Later stages can be compounded by sleep disturbance, mood changes, and stress. Patients with chronic MTrPs must be carefully screened for perpetuating factors, such as postural abnormalities, ergonomic factors, or hypothyroidism.

Physical Examination
The physical examination should begin with a careful medical and neurologic examination. This examination should be followed by a meticulous musculoskeletal examination. Posture, biomechanics, and joint function should be analyzed to identify any underlying factors that may have contributed to the development of the local or regional pain.

An active MTrP is usually associated with a painful, restricted ROM. The trigger point should be identified by gentle palpation across the direction of the muscle fibers. The examiner should appreciate a "rope-like" nodularity to the taut band of muscle. Palpation of this area is exquisitely painful and reproduces the patient's local and referred pain pattern.

 Interrater reliability studies have suggested that the most reliable physical signs of trigger points are focal tender-
ness and pain recognition. Fricton et al\textsuperscript{12} and Gerwin et al\textsuperscript{20} both found that interrater reliability improves when examiners are well trained before testing. Nonetheless, it should be appreciated that the ability to reliably palpate a taut band or to elicit a twitch response in the muscle varies and depends on the palpation skills and training of the examiner and the depth and size of the muscle (table 1).\textsuperscript{2} The recent study by Sciotti et al\textsuperscript{21} demonstrated that 4 blinded experienced examiners who “trained extensively together prior to the study” were able to reliably (80% agreement) identify the location of latent MTrPs in the upper trapezius muscle. Currently, no internationally agreed-on diagnostic criteria exist for myofascial pain. By comparison, in 1990 the American College of Rheumatology set forth specific criteria for diagnosing fibromyalgia, and these criteria have provided a standard for medical research in that area.\textsuperscript{3}

**DIFFERENTIAL DIAGNOSIS**

The differential diagnosis of myofascial pain is broad, and includes overlapping causes of regional musculoskeletal pain. The following questions may be useful in distinguishing the contribution of myofascial pain to the patient’s symptoms:

1. Is there regional myofascial pain, with trigger points present?
2. Is there regional myofascial pain, with trigger points present?
3. Is there regional myofascial pain, with trigger points present?
4. Is there regional myofascial pain, with trigger points present?
5. Is there regional myofascial pain, with trigger points present?
6. Is there regional myofascial pain, with trigger points present?
7. Is there regional myofascial pain, with trigger points present?
8. Is there regional myofascial pain, with trigger points present?
9. Is there regional myofascial pain, with trigger points present?
10. Is there regional myofascial pain, with trigger points present?
11. Is there regional myofascial pain, with trigger points present?

The differential diagnosis should include (but is not limited to) the following factors:\textsuperscript{1,22}:

2. Inflammatory disorders: polymyositis, polymyalgia rheumatica, rheumatoid arthritis.
5. Diskogenic disorders: degenerative disk disease, annular tears, protrusion, herniation.
7. Mechanical stresses: postural dysfunction, scoliosis, leg length discrepancy, poor body mechanics (eg, unsafe bending and lifting).
8. Nutritional, metabolic, and endocrine conditions: deficiency in vitamins B\textsubscript{1}, B\textsubscript{12}, and/or folic acid; alcoholic and toxic myopathy; iron, calcium, magnesium deficiency; hypothyroidism.
10. Infectious diseases: viral illness, chronic hepatitis, bacterial, or viral myositis.
11. Fibromyalgia or widespread chronic pain.

**PATHOPHYSIOLOGY**

There has been a burgeoning of new information concerning the neurophysiology of MTrPs. Their etiology remains controversial, is not yet adequately established, and deserves more serious research investigation. Current concepts of chronic myofascial pain generally incorporate a complex interplay between peripheral nociception and central sensitization.\textsuperscript{23} The present report reviews current concepts about pathophysiology, organizing them by neuroanatomic location for ease of understanding; however, one should appreciate that these processes are interrelated and should be considered in an integrated fashion.

**Motor Endplate**

An important finding in the pathophysiology of myofascial pain is a pathologic increase in release of acetylcholine (ACh) by the nerve terminal of an abnormal motor endplate under resting conditions—an occurrence supported by electrodiagnostic evidence.\textsuperscript{24,25} This abnormality is considered to be the primary dysfunction in the “integrated hypothesis” proposed by Simons et al,\textsuperscript{13} which postulates a positive feedback loop (see fig 1).

In support of the concept of the abnormal motor endplate, electrodiagnostic studies have demonstrated endplate noise (EPN) significantly more frequently in MTrPs than in the same endplate zone outside of the MTrP.\textsuperscript{24,25} Because EPN is characteristic, but not diagnostic, of MTrPs, the significance of these findings remains disputed.

An increase in EPN has been seen in response to many types of mechanical stimulation of the endplate structure\textsuperscript{26} and does not appear to be specific to myofascial pain. Additionally, chemical stimulation of normal endplates\textsuperscript{27} may produce an endplate noise pattern at as much as 1000 times the normal rate of spontaneous discharge. It is therefore possible that mechanical, chemical, or other noxious stimuli or injury may mediate the abnormal release of ACh.

**Muscle Fiber**

It is hypothesized that increased ACh release could result in sustained depolarization of the postjunctional membrane of the muscle fiber and produce sustained sarcomere shortening and contracture. This maximally contracted sarcomere in the region of the motor endplate, referred to as a “contraction knot” by Simons,\textsuperscript{1} is diagrammed in figure 2. Compelling histologic support for this phenomenon is found in canine models of MTrPs: longitudinal sections of dog trigger points demonstrate this sarcomere shortening, and cross-sections of dog and human MTrPs strongly suggest it as well.\textsuperscript{28,29}

![Fig 1. Positive feedback cycle that summarizes the integrated hypothesis. Increase in release of ACh at motor endplate due to mechanical trauma or chemical stimulation of the nerve terminal induces sustained sarcomere contraction. This occurrence results in localized ischemia, which in turn results in the release of substances that sensitize nociceptors, produce pain, and induce release of neurovasoreactive chemicals. These chemicals lead to increases in ACh release, sustaining the cycle. Adapted with permission.\textsuperscript{1}](image-url)
One consequence of a chronically sustained sarcomere shortening may be greatly increased local energy consumption and reduction of local circulation, a combination that produces local ischemia and hypoxia. An elegant, well-instrumented study of different subjects clearly demonstrated that severe local hypoxia was in the center of tender nodules. The combined tension produced by multiple contraction knots (observed histologically) may account for the increased palpable tension of the taut band. The unrelieved sustained tension of muscle fibers in the taut band produces an enthesopathy at their myotendinous junctions that can be identified as an attachment MTrP. Muscle-stretching techniques will therefore be effective by equalizing sarcomere length throughout the affected muscle fibers and by breaking the feedback cycle.

The localized muscle ischemia stimulates the release of neurovasoactive substances such as prostaglandin, bradykinin, capsaicin, serotonin, and histamine that sensitize afferent nerve fibers in muscle. These sensitized fibers in turn account for local MTrP tenderness.

**Central Mechanisms: Spinal and Supraspinal**

The referred pain resulting from trigger points arises from central convergence and facilitation. It is known from experimental data that, under pathologic conditions, convergent connections from deep afferent nociceptors to dorsal horn neurons are facilitated and amplified in the spinal cord. Referential myotomes occurs due to spreading of central sensitization to adjacent spinal segments. This pattern results in both referred pain and in expansion of the region of pain beyond the initial nociceptive region.

At the level of the central nervous system, spinal neuroplastic changes occur in the second-order neuron pool of the dorsal horn due to persistent pain. These changes produce a lasting increase in the excitability of nociceptor pathways. Central sensitization results are characterized by increased excitability of the neurons and expansion of the receptive pool of neurons. Neurotransmitters involved in the process of central sensitization include substance P, N-methyl-D-aspartate, glutamate, and nitric oxide. In addition, there may also be impairments in supraspinal inhibitory descending pain control pathways releasing inhibitory neurotransmitters such as y-aminobutyric acid, serotonin, and norepinephrine.

**Autonomic Nervous System**

Under pathologic conditions, the neurovasoactive substances such as bradykinin, substance P, serotonin, and histamine also stimulate activity of the local autonomic nervous system fibers to release more ACh, completing the positive feedback loop as diagrammed in figure 1. Endplate potentials (noise and spikes) are increased by stimulation of the autonomic nervous system and reduced by reducing its effect. In experimental data, endplate spike rates were increased by psychologic stress. Spike (and endplate noise) activity is inhibited by phentolamine in human and rabbit studies and by phenoxybenzamine in humans. Both drugs are a-sympathetic blockers. Thus, there seems to be a relationship between autonomic activation and increased motor endplate activity.

The sympathetic nervous system also, presumably, plays a role in the commonly described findings of painful skin rolling, hypersensitivity to touch, temperature and blood flow changes.

**Table 1: Interrater Reliability Studies for Identification of MTrPs**

<table>
<thead>
<tr>
<th>Study</th>
<th>Results</th>
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<th>Results</th>
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<tr>
<td>Experienced But Untested Examiners</td>
<td></td>
<td>Wolfe et al</td>
<td>(\kappa = .35^*)</td>
</tr>
<tr>
<td>Nice et al</td>
<td>(\kappa = .38)</td>
<td>Lew et al</td>
<td>10% and 21% concordance</td>
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<td>Inexperienced But Trained Examiners</td>
<td></td>
<td>Njoo et al</td>
<td>(\kappa = .49)</td>
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<tr>
<td>Haieh et al</td>
<td>Taut band .22</td>
<td>Twitch response .12</td>
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<td></td>
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<td>Referral pain .34</td>
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<tr>
<td>Experienced and Trained Examiners</td>
<td></td>
<td>Gerwin et al</td>
<td>(\kappa = .74^*)</td>
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<tr>
<td>(4 examiners)</td>
<td>Taut band, 3 muscles nearly complete agreement, 2</td>
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<td></td>
<td>Muscles, (\kappa = .40), .46</td>
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* Kappa values are mean values reported by Simons et al. The Simons table shows individual examination values on which the mean value was based.
abnormal sweating, reactive hyperemia, dermatographia, and altered pilomotor responses that are associated with myofascial pain.39

TREATMENT

Pharmacologic Treatment of Myofascial Pain

Given the considerable clinical overlap among myofascial pain, fibromyalgia, regional soft tissue pain, and tension headache, agents beneficial in an associated syndrome may prove useful in treating myofascial pain. In the absence of controlled data specifically examining drug efficacy in myofascial pain, clinicians often extrapolate from these associated disorders.

Nonsteroidal anti-inflammatory drugs. There is minimal literature evaluating the use of nonsteroidal anti-inflammatory drugs (NSAIDs) for the treatment of chronic muscle pain and myofascial pain. Several studies40-42 have found a small supplemental benefit of NSAIDs for management of pain in fibromyalgia, if used in combination with alprazolam, amitriptyline, or cyclobenzaprine. Interestingly, NSAIDs continue to be popular among patients, and in a recent study by Wolfe et al,43 NSAIDs were considered more effective than acetaminophen for pain management by patients with fibromyalgia.

Tramadol. Tramadol is a combination of a weak opioid agonist as well as an inhibitor of the reuptake of serotonin and norepinephrine in the dorsal horn. There are no published controlled trials of tramadol for the treatment of myofascial pain; however, several studies44-48 support its efficacy in fibromyalgia, chronic low back pain (LBP), and osteoarthritis, all of which are commonly seen in association with myofascial pain.

Antidepressants. Tricyclic antidepressants such as amitriptyline are shown to be effective for chronic tension-type headache, fibromyalgia, acute LBP, and intractable pain syndromes with muscle spasm.49-52 Selective serotonin reuptake inhibitors have not been specifically studied for myofascial pain, although efficacy has been documented in fibromyalgia for improving pain, sleep, and global sense of well-being.53,54

Alpha2 adrenergic agonists. The 2 major alpha2 adrenergic agonists available for clinical use are clonidine and tizanidine. Tizanidine acts centrally at the level of the spinal cord to inhibit spinal polysynaptic pathways and to reduce the release of aspartate, glutamate, and substance P.54,55 Additionally, tizanidine has supraspinal effects that increase nociceptive thresholds and inhibit responses of spinal neurons.56 In 1 open-labeled study57 of tizanidine in a mixed population of patients with myofascial pain syndrome or fibromyalgia, tizanidine treatment reduced pain. There are no published double-blind placebo-controlled trials of tizanidine in the treatment of myofascial pain or fibromyalgia. The efficacy of tizanidine in the treatment of acute low back and neck pain has been documented.58,59

Anticonvulsants. To date, there are no controlled trials of anticonvulsants in the treatment of myofascial pain or fibromyalgia. One open label study60 of gabapentin in the treatment of chronic daily headache found possible efficacy.

Botulinum toxin. Botulinum toxin type A appears to be emerging as a promising but expensive new agent with efficacy in chronic myofascial pain syndromes. Chesire et al61 performed a small, randomized, double-blind, placebo-controlled trial of botulinum toxin type A and demonstrated a reduction of at least 30% in visual analog pain scales, verbal pain descriptors, palpable muscle firmness, and pressure pain thresholds in the botulinum toxin group compared with a placebo (saline) group. In a recent study by Porra et al62 botulinum toxin type A was compared with steroid injection for the treatment of chronic myofascial pain and was found to provide a greater improvement in symptoms at 30 and 60 days posttreatment. Not all studies have found efficacy: Wheeler et al63 were unable to demonstrate a statistically significant difference between botulinum toxin and placebo for the treatment of refractory unilateral cervicotothoric myofascial pain.

There may be both a peripheral and central mechanism that explains the apparent efficacy of botulinum toxin in the treatment of chronic myofascial pain. First, the blockade of ACh release at the neuromuscular junction reduces muscle hyperactivity, which in turn may decrease local ischemia. Second, if, as theorized, trigger points are sustained by excessive ACh release and sarcomere shortening, then botulinum toxin may disrupt the abnormal neurophysiology of the trigger point. Evidence has also been found of retrograde uptake of botulinum toxin type A into the spinal cord and nucleus raphe, structures that modulate expression of neurotransmitters important in pain perception (eg, substance P, enkephalins).64

Although the efficacy of botulinum toxin type B has not yet been studied to the extent that type A has, it may prove to have similar efficacy. Studies in this area are ongoing, and a greater understanding of the mechanism(s) and efficacy of botulinum toxin in myofascial pain will likely become available soon.

Nonpharmacologic Treatment of Myofascial Pain

Postural, mechanical, and ergonomic modifications. Although standard clinical practice includes efforts to correct postural and ergonomic abnormalities, there is little direct data to support this approach in treating myofascial pain. One study, by Komiyama et al,65 combined postural training and behavioral therapy in the treatment of myofascial oral pain and found that the subjects receiving the combination therapy were able to regain free unassisted mouth opening earlier than those treated with behavioral therapy alone; however, the differences in outcome were clinically minor.

The occupational medicine literature provides evidence that injuries are more common when workers are subjected to greater loads and have undesirable postures during their work.66 Occupational muscle pain syndromes are theorized to occur as the result of repetitive microtrauma and myofascial shortening. Correction of awkward postures is a standard part of treatment of these disorders,67 although long-term efficacy studies are lacking.

Stress reduction. Stress reduction techniques, including cognitive-behavior programs, meditation, progressive relaxation training, and biofeedback, are often incorporated into chronic pain rehabilitation programs. Studies specifically addressing the efficacy of these techniques for myofascial pain are few. In 1986, Crockett et al68 compared a multifaceted relaxation program with physiotherapy with dental splinting and with transcutaneous electric nerve stimulation (TENS) for management of chronic facial and masticatory myofascial pain; they found equivalent results and good response among all treatment groups. Electromyographic biofeedback as well as meditation-based stress reduction programs seem to be beneficial in fibromyalgia.69,70

Acupuncture. A growing body of evidence supports the efficacy of acupuncture in myofascial pain and fibromyalgia. The limited amount of high-quality data suggests that real acupuncture is better than sham for relieving pain, improving global ratings, and reducing morning stiffness in fibromyalgia.71 The 1997 National Institute of Health consensus statement on acupuncture72 concluded that "acupuncture may be useful as an adjunct treatment or an acceptable alternative to be included in a comprehensive management program" in the treatment of fibromyalgia, myofascial pain, LBP, osteoarthritis, and lateral epicondylitis. Birch and Jamison73 found relevant
acupuncture (over points relevant to myofascial neck pain) to be superior to both NSAID treatment and irrelevant acupuncture (superficial needling over points not related to neck pain) in a group of 46 patients with chronic myofascial pain. Interestingly, a remarkably close correspondence has been found between acupuncture points and trigger points, with 71% of trigger points sharing location and pain distribution patterns with acupuncture points. Questions that need to be answered in future randomized controlled trials include the duration of benefit of acupuncture, the optimal acupuncture techniques, and the value of booster treatments for the treatment of myofascial pain.

**Massage, TENS and ultrasound.** Studies suggesting efficacy of massage as part of a treatment program for myofascial pain are scant. In a study by Gam et al., massage combined with stretching exercises was better than the control group in reducing the number and intensity of myofascial trigger points. There was only a mild reduction of neck and shoulder pain. In a recent article by Hernandez-Reif et al., massage therapy was found effective in reducing pain, increasing serotonin and dopamine levels, and reducing symptoms associated with chronic LBP; however, this study did not specify the etiology of the pain.

TENS treatment has shown mixed results in the treatment of myofascial pain. One single-blind study compared TENS with sham TENS in 10 patients for treatment of myofascial pain and found no benefit for pain reduction; however, the study did use subthreshold TENS parameters. By comparison, Graff-Radford et al. compared 4 different TENS settings to no-stimulation control in a double-blind study and found that high-frequency, high-intensity TENS reduced myofascial pain. Ultrasound in combination with massage and exercise has been tested in 1 randomized controlled trial. In this study, ultrasound, massage, and exercise had no additional benefit over sham ultrasound with massage and acupuncture for the treatment of MTrPs.

**Exercise for Myofascial Pain**

Stretching exercises form the basis of exercise treatment of myofascial pain. This treatment addresses the muscle tightness and shortening that are closely associated with pain in this disorder and permits gradual restoration of normal activity. Slow, sustained stretch throughout the available ROM is the most effective approach. Strengthening exercise is contraindicated for the initial treatment of MTrPs. Electromyographic studies have shown that muscles with active MTrPs start out as fatigued, fatigue more rapidly, and recover more slowly. Strengthening exercises performed early in a rehabilitation program for myofascial pain may result in substitution of other muscles, and risk overloading the muscle, causing pain and contributing to worsening myofascial pain.

Patients should be encouraged to remain active but to perform daily activities in a gentle, lightly loaded manner. When a movement leads to pain, the patient should stop at that point and slowly, gently explore extending the movement just a little further to help release the muscle tightness. Clinical experience suggests that leaving a muscle in the shortened position aggravates MTrPs.

One controlled, blinded study compared the effects of a 5-day home program of muscle stretching exercises and self-massage (using a Thera Cane® device® with an active ROM program for neck and back myofascial pain). Both groups showed some improvement. Stretching program showed significantly more improvement than subjects in the active ROM program in analog scale pain reports ($P<.001$) and in pressure pain threshold readings ($P<.001$), though not in percentage of time in pain. The study was not designed to distinguish the relative contributions of self-massage from those of slow stretching exercises. Critical in-depth reviews of treatment literature by the Philadelphia Panel for Evidence-Based Clinical Practice found support for the use of therapeutic exercise in LBP, neck pain, and knee pain.

After pain from MTrPs is reduced and ROM restored, a graded stabilization and strengthening program should be undertaken to maximize functional outcome. Aerobic exercise should also be included as part of an overall musculoskeletal and cardiovascular fitness program to prevent recurrence.

**Trigger Point Injection for Myofascial Pain**

Stretching exercises are the mainstay of myofascial pain management, and injection therapy should be reserved to supplement or augment these exercises. When MTrP injection is used as the primary therapy, patients are at risk for becoming dependent on this treatment for pain relief. Educating patients about the effectiveness of manual techniques and instruction in the specific techniques empowers patients to self-manage effectively their symptoms. With increasing relief of pain and increasing function, resumption of normal activity helps to further inactivate their symptoms. When injection proves necessary for initiating therapy or dealing with a recalcitrant area of myofascial pain, a series of injections should be initiated, and the patient informed of the limited role of this treatment in the long-term management of myofascial pain. Often, 3 consecutive visits for injection are recommended in chronic myofascial pain, with reassessment after the third visit to evaluate the efficacy of the injections and to determine if further injections are necessary.

MTrP injections may employ several medications, including no medication (dry needling), short- or long-acting anesthetics, steroids, and botulinum toxin. Injections may use a number of different techniques: slow search, fast in-fast out, superficial dry needling, intramuscular stimulation, twitch-obtaining intramuscular stimulation, and needleling with preinjection blocks. Several theories exist regarding the mechanism of action of injections for myofascial pain.

Dry needling of the MTrP provides as much pain relief as injecting lidocaine but causes more postinjection soreness. Effectiveness of needling depends on the needle eliciting local twitch responses. Presumably, the needle mechanically disrupts and terminates the dysfunctional activity of involved motor endplates, with or without injection. Longer-acting anesthetics are more myotoxic without a proven increase in MTrP pain relief. The effectiveness of injecting steroids in MTrPs is controversial and without a clear rationale because little evidence exists to support an inflammatory pathophysiology for MTrPs. Injection of botulinum toxin appears to be an emerging treatment for myofascial pain. In a recent systematic review article on needling therapies for MTrPs, Cummings and White conclude that, based on current medical evidence, the "nature of the injected substance makes no difference to the outcome and that wet needling is not therapeutically superior to dry needling." This is clearly an area in which more research is needed.

Hong's fast in-fast out technique elicits local twitch responses more quickly than other techniques and presumably reduces needle trauma to muscle fibers by the twitch movement. Baldry et al. recommend superficial dry needling, which they speculate may inactivate MTrPs through stimulation of...
cutaneous A delta fibers. Chu,9 based on Gunn’s work,90 reports a technique in which neurogenically evoked muscle twitches relieve myofascial-type pain. The needling and infiltration technique described by Fischer and Imamura,91 who used a preinjection block, permits more thorough injection of the trigger point and taut band region with reportedly less patient discomfort.

All these techniques rely on an accurate identification of MTrPs by means of palpation. There is no definitive evidence that 1 technique is superior to another in long-term outcome. Cummings and White92 remark, “Because no technique is better than any other, we recommend that the method safest and most comfortable for the patient should be used.” Very slim acupuncture needles may have the advantage of minimizing tissue trauma and allowing the practitioner to needle 4 to 6 trigger points at 1 session.

FUNCTIONAL OUTCOMES IN MYOFASCIAL PAIN

Outcome studies of myofascial pain and its treatment are few. As noted previously, physical therapy of MTrPs was effective in at least 1 well-designed study.80 In another study92 of pain, disability, and psychologic functioning in chronic LBP subgroups, patients with LBP of myofascial origin demonstrated similar or slightly worse outcomes than those with disk herniation, as measured by several standardized questionnaires on pain and disability. Patients with myofascial pain have less accurate beliefs regarding their pain symptoms, express more dissatisfaction with physician efforts to treat their pain, and report a receiving dearth of information from their physician.83

Last, a 1998 study by Heikkila et al94 investigated the outcome from a multidisciplinary rehabilitation program for patients with whiplash and myofascial pain. After the rehabilitation period, 49% of patients had improved their coping skills, and this figure rose to 63% after 2 years. In addition, 46% of patients had increased their life satisfaction. The myofascial pain group also decreased their sick-leave time.94

Future Trends and Research

Successful clinical research on myofascial pain requires that an authoritative definition of diagnostic criteria be developed for this disorder. Existing diagnostic criteria are often poorly identified and vary from study to study. The International Myofascial Pain Society is now conducting an international multicenter study95 to address this fundamental issue of generally agreed-on diagnostic criteria. The lack of a practical laboratory or imaging test for MTrPs contributes to the difficulty in objectively identifying individuals with this disorder. An emerging technology, tissue impedance imaging, may prove useful. It clearly images neuromas and nerves and at least some MTrPs. Another possible test is the identification of reduced-amplitude, magnetic-evoked cerebral potential when the muscle at the MTrP is stimulated.96

An understanding of the prevalence and risk factors for myofascial pain is also needed as a basic building block of future research. Studies of the prevalence of MTrPs as a function of age and their prevalence as a significant cause of the pain in regional diagnoses like headache, frozen shoulder, and LBP are needed. Establishing good interrater reliability of blinded examiners is essential to the success of such studies.

A better understanding of MTrPs requires further investigation into the pathophysiology of myofascial pain. The pathologic processes responsible for the taut band are more complex than previously envisioned.97 The linkage between autonomic function and ACh release also needs clarification.

Well-controlled, double-blinded randomized trials of treatments are needed to establish effective therapy for patients with myofascial pain. The impact of MTrPs on muscle function needs further evaluation as well.1

**SUMMARY**

Myofascial pain is a common cause of regional musculoskeletal pain. It may develop from intrinsic postural or biomechanic factors and may be the primary cause of pain. In other cases, myofascial pain may occur in combination with other underlying pain generators such as disk herniation or radiculopathy, which sensitize the myotome. The pathophysiology appears complex and to involve multiple levels of the peripheral and central nervous system. Many pharmacologic and nonpharmacologic treatments are used in the management of acute and chronic myofascial pain, but, as yet, empiric data to guide treatment choice are limited. Research is needed to define better the trigger point identification, and functional outcome studies are needed to assess rigorously the efficacy of treatments.

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