Myofascial Pain
Mechanisms to Management

James Fricton, DDS, MS

KEYWORDS

• Pain • Muscles • Spasm • Myofascial pain • Trigger point • Soft tissue • Chronic pain • Tender muscles

KEY POINTS

- Chronic pain is the main reason for seeking health care, the most common reason for disability and addiction, and the highest driver of health care costs, and it is most often caused by myopain conditions.
- Myofascial pain (MFP) is the most common cause of persistent regional pain such as back pain, headaches, and facial pain.
- MFP is readily diagnosed through identifying differential clinical characteristics and soft tissue palpation.
- Treatments of myopain conditions include:
  - Stretching, postural, relaxation, strengthening, and conditioning exercises
  - Reduction of all contributing factors that strain the muscles and heighten peripheral and central sensitization
  - Counterstimulation treatments to desensitize soft tissues
- Use of a transformative care model that integrates patient training in reducing risk factors and enhancing protective factors with evidence-based treatments using an integrative team of health professionals enhances long-term outcomes.

INTRODUCTION

More than 100 million adults in the United States are affected by chronic pain conditions, costing more than $500 billion annually in medical care and lost productivity.[1–4] Several studies have found that myopain conditions such as myofascial pain (MFP) and fibromyalgia (FBM) are the most common chronic pain condition leading to nearly all chronic pain conditions, including back pain, headaches, neck pain, and jaw pain.[5–10] This finding makes them one of the top reasons for seeking health care, the most common reason for disability and addiction, and the highest driver of health care costs, costing more than cancer, heart disease, dementia, and diabetes. The personal impact of chronic pain in terms of suffering, disability, drug use, depression, and conflict is incalculable. In hopes of improving the condition, medical care often involves expensive and high-risk passive interventions, such as polypharmacy, opioid analgesics, high-tech imaging, implantable stimulators, and surgery. However, more than half of the persons seeking care for pain conditions still have pain 5 years later and many develop long-term disability.[11–19] Because nearly one-third of the population has chronic pain to some extent, most people assume chronic pain is intractable. However, that is not the case. Myopain conditions can be successfully managed in most patients and any poor long-term outcomes are often caused by the lack of recognition and adequate care.[20–26] Thus,

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HealthPartners Institute for Education and Research, University of Minnesota School of Dentistry, 4700 Dale Drive, Edina, MN 55424, USA
E-mail address: frict001@umn.edu

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the principals of cause, diagnosis, and management of myopain conditions are relevant for all health care professionals.

Most Common Chronic Pain Conditions

Everyone, at some point in their lives, has experienced acute muscle pain associated with muscle spasm or repetitive strain. However, when acute pain becomes chronic, patients and their health care professionals often become confused and overlook the muscle in favor of treating other conditions, such as depression, osteoarthritis, or neuropathic conditions. This lack of understanding leads to misdiagnosis, inadequate care, mistreatment, and progression of an acute problem to chronic pain. When behavioral and psychological factors are evident, myopain conditions often become misunderstood, assuming the patient's experience of pain is imagined or exaggerated or caused by the psychosocial issues. However, MFP is a physical pain condition that can be successfully managed.

MFP is the most common cause of persistent regional pain such as back pain, shoulder pain, tension-type headaches, and facial pain, whereas FBM is the most common widespread pain.[20] MFP is a regional muscle pain disorder characterized by localized muscle tenderness, limited range of motion, and regional pain, whereas FBM is associated with soft tissue tenderness, fatigue, stiffness, nonrefreshed sleep, and widespread physical pain.[24] Two prior studies of clinic populations found that MFP conditions were cited as the most common cause of pain, responsible for 54.6% of chronic head and neck pain[6] and 85% of back pain.[7] Another study, in a general internal medicine practice, found that, among those patients who presented with pain, MFP was present in 29.6% of the population and was the most common cause of pain.[8] Symptoms of FBM also seem to be prevalent in the general population, with up to 5% having FBM, and are more prevalent in patients with chronic fatigue (estimated as at least 20%).[4,5,27]

CLINICAL PRESENTATION

Clinical Characteristics

The clinical characteristics of MFP include hard, palpable, discrete, localized nodules, called trigger points (TrPs), which are located within taut bands of skeletal muscle (**Box 1**). TrPs are painful on compression and associated with pain in predictable regional patterns within a referral zone. The pain in the zone of reference is usually located over the tender point (TeP) or spreads out in a referral pattern to distant sites (**Fig. 1**). This tenderness is often referred to as a TrP because palpation of the TeP in the muscle alters the pain in the zone of reference, and, if treated, it resolves the resultant pain.[24] MTrPs can be either active or latent.[24] An active TrP is associated with spontaneous pain in which pain is present without palpation. This spontaneous pain can be at the site of the TrP or referred to more distant sites. However, firm palpation of the active TrP (A-TrP) increases pain locally and usually reproduces the patient's remote pain. A latent MTrP (L-TrP) is not associated with spontaneous pain, although pain can often be elicited in an asymptomatic patient by a mechanical stimulus such as finger pressure over the L-TrP. There are generally no neurologic deficits associated with the disorder unless a nerve entrapment syndrome with weakness and diminished sensation coincides with the muscle TrPs.[6] Blood and urine studies are generally normal unless the pain is caused by a concomitant disorder.[7] Imaging studies, including radiographs

Box 1
Clinical characteristics of MFP

TrPs in taut band of muscle
Tenderness on palpation
Consistent points of tenderness
Palpation alters pain locally or distally

Associated symptoms
Otologic
Paresthesias
Gastrointestinal distress
Visual disturbances
Dermatographia

Pain in zone of reference
Constant dull ache
Fluctuates in intensity
Consistent patterns of referral
Alleviation with extinction of TrP

Contributing factors
Traumatic and whiplash injuries
Occupational and repetitive strain injuries
Physical disorders
Parafunctional muscle tension producing habits
Postural and repetitive strains
Disuse
Metabolic/nutritional
Sleep disturbance
Psychosocial and emotional stressors (direct)

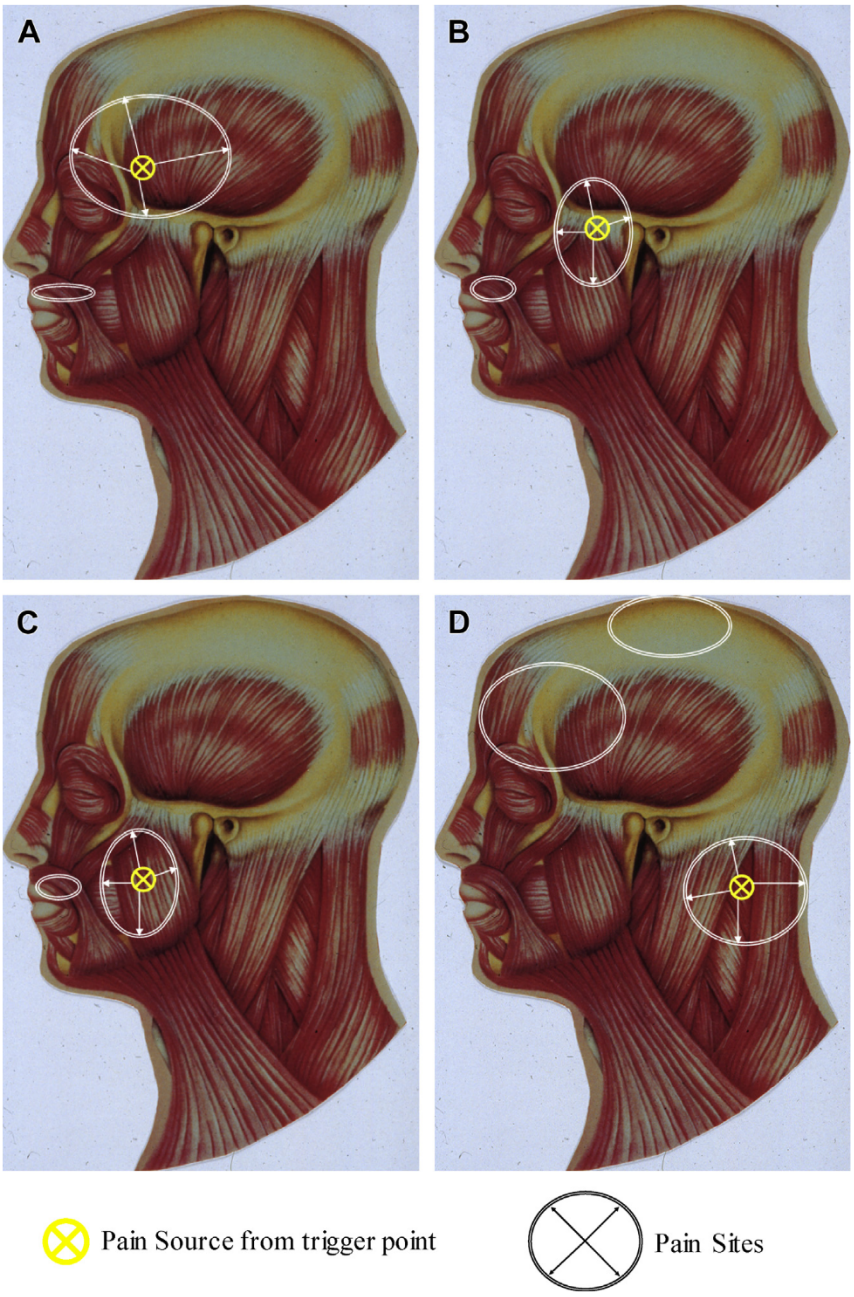

Fig. 1. TrPs with associated patterns of referral in the head and neck. (*A*) The pain source is the anterior temporalis TrP. The pain sites include temple, frontal, and retro-orbital headaches and pain in the maxillary anterior teeth. These muscles are activated by clenching, bruxism, and other oral parafunctional habits. (*B*) The pain source is the deep masseter TrP. The pain sites include preauricular pain, earaches, and pain in the maxillary posterior teeth. These muscles are also activated by clenching, bruxism, and other oral parafunctional habits. (*C*) The pain source is the middle masseter TrP. The pain sites include temple, frontal, and retro-orbital headaches and pain in the maxillary anterior teeth. These muscles are also activated by clenching, bruxism, and other oral parafunctional habits. (*D*) The pain source is the splenius capitis TrP in the posterior cervical area. The pain sites include posterior cervical, vertex headache, and frontal headaches. These muscles are also activated by clenching and forward head posture.

and MRI, do not reveal any pathologic changes in the muscle or connective tissue.

The affected muscles may also have increased fatigability, stiffness, subjective weakness, pain in movement, and slightly restricted range of motion.[5–7,27] The muscles are painful when stretched, causing the patient to protect the muscle through poor posture, bracing, and sustained contraction resulting in persistence of the pain.[8] For example, in a study of jaw range of motion in patients with MFP and no joint abnormalities, the patients showed a slightly diminished range of motion (approximately 10%) compared with normal individuals and pain in full range of motion,[8] which is considerably less limitation than is found with joint locking caused by a temporomandibular joint (TMJ) internal derangement. This postural bracing may also lead to the development of other TrPs in the same muscle and agonist muscles causing the pain to spread to broader regional areas. This development can cause multiple TrPs with overlapping areas of pain referral and changes in pain patterns as TrPs are inactivated.

Although routine clinical electromyographic (EMG) studies show no significant abnormalities associated with TrPs, some specialized EMG studies reveal differences.[28–31] Needle insertion into the TrP can produce a burst of electrical activity that is not produced in adjacent muscle fibers.[31] In 2 experimental EMG studies of TrPs, Simons[30] and Fricton and colleagues[28] found abnormal electrical activity associated with the local muscle twitch response when specifically snapping the tense muscle band containing a myofascial TrP.

The mechanical properties of muscles, including firm consistency, stiffness, and lack of elasticity of muscles containing the TrPs, has also been documented and found to be different from adjacent muscles.[32,33] For example, elastography with ultrasonography imaging confirms these significant tissue abnormalities and that morphologic changes are associated with MTrPs (**Fig. 2**). Changes in elasticity and stiffness of MTrP compared with the surrounding tissue suggest a disruption of normal muscle fiber structure. The increase in local tissue density in the form of contraction knots may result from increased muscle fiber contraction and recruitment and local injury. Furthermore, this possibility was confirmed with magnetic resonance elastography, which showed that the propagation of induced vibration shear waves in TrP bands differs from that in normal muscle tissue. Skin overlying the TrPs in the masseter muscle also seems to be warmer, as measured by infrared emission.[34,35] Although most of these findings are from solitary studies, they provide preliminary evidence of a broad range of objective characteristics that are important in understanding the diagnosis and cause of MFP.

Association with Other Pain Conditions

MFP, particularly in the head and neck, is frequently overlooked as a diagnosis because it is often accompanied by signs and symptoms in addition to pain as well as other pathologic pain conditions.[24] MFP seems to not only mimic many other conditions, such as joint disorders, migraine headaches,

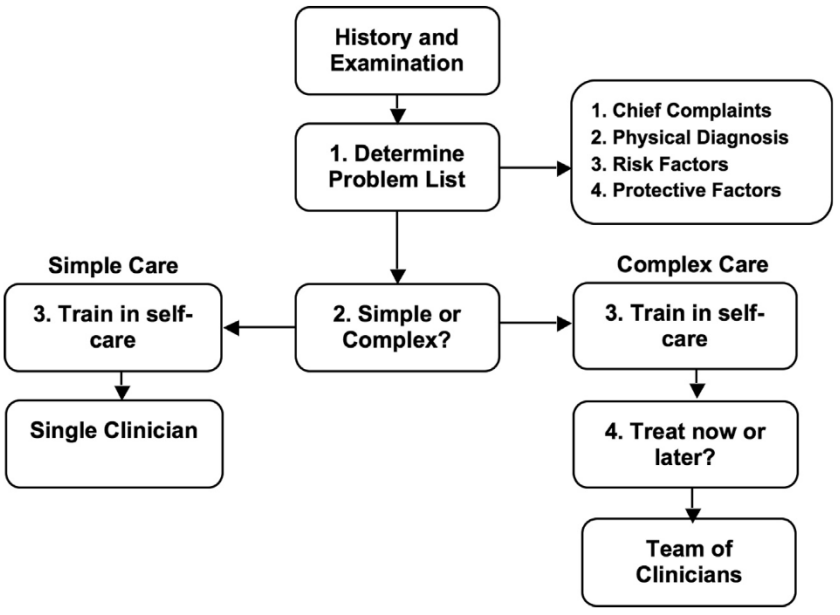

Fig. 2. A decision tree for triaging patients and enhancing outcomes and successful care.

neuralgias, temporal arteritis, TMJ disorders, spinal disk disease, sinusitis, dental pain, and other disorders, but also accompanies other pain disorders.[36–41] For example, TrPs coincide with 14 of 18 TeP sites in diagnostic criteria for FBM.[42,43] TrPs also develop in association with joint disorders such as disc derangements, osteoarthritis, and subluxation.[43–48] MFP is also reported to be found with systemic or local infections of viral or bacterial origin; with lupus erythematosus, scleroderma, and rheumatoid arthritis; and along segmental distribution of nerve injury, nerve root compression, or neuralgias.[44] These findings suggest that muscles may serve as part of the alarm system for regional disorders and may lead to confusion regarding diagnosis of MFP as the primary cause of pain.

Relationship to Other Muscle Pain Disorders

In addition to MFP, there are several other distinct muscle disorder subtypes affecting the masticatory system, including myositis, muscle spasm, muscle contracture, and FBM.[40,41] Perhaps the most pragmatic taxonomy related to differentiating muscle pain disorders is in the Academy of Orofacial Pain's Guidelines for Diagnosis and Management of Orofacial Pain.[40] In this classification, different muscle disorders are descriptively defined by their characteristics and classified as MFP (regional pain and localized tender TrPs), FBM (widespread pain with localized TePs), myositis (regional pain and diffuse tenderness), muscle spasm (brief painful contraction with limited range of motion), contracture (long-standing limited range of motion), and muscle splinting (regional pain and localized tenderness accompanying a joint problem).

Myositis

Myositis is an acute condition with localized or generalized inflammation of the muscle and connective tissue and associated pain and swelling overlying the muscle. Most areas in the muscle are tender, with pain in active range of motion. The inflammation usually has a local cause, such as overuse, excessive stretch, a drug, local infection from pericoronitis, trauma, or cellulitis. This condition is also termed delayed-onset muscle soreness in cases of acute overuse.

Muscle spasm

Muscle spasm is also an acute disorder characterized by a brief involuntary tonic contraction of a muscle. It can occur as a result of overstretching of a previously weakened muscle, protective splinting of an injury, as a centrally mediated phenomenon such as Compazine-induced spasm of the lateral pterygoid muscle, or overuse of a muscle. A muscle in spasm is acutely shortened, painful, and with joint range of motion limited. Lateral pterygoid spasm on one side can also cause a shift of the occlusion to the contralateral side.

Muscle contracture

Muscle contracture is a chronic condition characterized by continuous gross shortening of the muscle with significant limited range of motion. It can begin as a result of factors such as trauma, infection, or prolonged hypomobility. If the muscle is maintained in a shortened state, muscular fibrosis and contracture may develop over several months. Pain is often minimal in the process but it can occur as a result of reactive bracing or clenching to protect the muscle.

Fibromyalgia

FBM is a common rheumatic pain syndrome that resembles MFP but is more widespread and centrally generated. It consists of widespread pain, fatigue, unrefreshed sleep, and cognitive dysfunction such as confusion, forgetfulness, inability to concentrate, and impaired memory. In addition, most patients have tenderness on palpation at definable classic locations on the neck, trunk, and extremities (**Box 2**). The prevalence of FBM in the general population has ranged from 3.7% to 20%.[19,20] Other characteristics of FBM are divided into the frequency that they occur. The characteristics that occur in more than 75% of patients with FBM include chronic fatigue, stiffness, and sleep disturbance, whereas the variety of associated symptoms that occur in less than 25% of patients with FBM include irritable bowel, headaches, psychological distress, Raynaud phenomena, swelling, paresthesias, and functional disabilities.[21,22] It has been shown that central nervous system (CNS)–modulating factors such as stress, sleep disorders, and depression play some role in FBM.[23] Sleep abnormalities have been well documented, but it is unproved whether these are the primary abnormality or an associated or secondary abnormality. More than 75% of patients with FBM are female between the ages of 30 and 60 years.[45] Because FBM commonly occurs with other medical conditions, it is possible that the reported age of onset is artificially high. Therefore, FBM should be suspected in any person presenting with widespread pain because the consequences of prolonged, undiagnosed pain can be considerable.

Examination Findings

Tenderness in the soft tissues is the primary clinical and diagnostic characteristic in MFP and

Box 2
Clinical characteristics of Fibromyalgia (FBM) as defined by the American College of Rheumatology 2010 criteria

1. Widespread Pain Index (WPI) score. History of widespread pain in the past week (0–19): shoulder girdle (left or right), upper arm (left or right), lower arm (left or right), hip (buttock; left or right), upper leg (left or right), lower leg (left or right), jaw (left or right), chest, abdomen, neck, upper back, or lower back
2. Symptom Score (SS) includes adding both primary and secondary symptoms (0–6)
   a. Primary SS (0–9): 1, fatigue; 2, unrefreshed sleep; and 3, cognitive symptoms such as confusion, forgetfulness, inability to concentrate. For each of 3 problems, a score of 0 = no problem; 1 = slight, mild, or intermittent problems; 2 = moderate (considerable problems are often present at a moderate level); and 3 = severe: pervasive, continuous, and life-disturbing problems.
   b. Secondary SS (0–3): 0 = 0 symptoms, 1 = 1 to 10 symptoms, 2 = 11 to 24 symptoms, and 3 = 25 or more symptoms.

Diagnosis of FBM is present if the following 3 conditions are met:

1. (a) The WPI score (part 1) is greater than or equal to 7 and the SS score (parts 2a and b) is greater than or equal to 5 or; (b) the WPI score (part 1) is from 3 to 6 and the SS score (parts 2a and b) is greater than or equal to 9.
2. Symptoms have been present at a similar level for at least 3 months.
3. The person does not have a disorder that would otherwise explain the pain.

Adapted from Wolfe, F, Clauw DJ, Fitzcharles MA, et al. The American College of Rheumatology Preliminary Diagnostic Criteria for Fibromyalgia and Measurement of Symptom Severity. Arthritis Care Res (Hoboken) 2010;62(5):600–10.

other muscle disorders. Tender areas in FBM are termed TePs, whereas in MFP they are termed TrPs. TrPs in MFP are 2-mm to 5-mm diameter points of increased hypersensitivity in palpable bands of skeletal muscle, tendons, and ligaments with decreasing hypersensitivity as the band is palpated further away from the TrP. As noted earlier, TrPs may be active or latent.[30] Active TrPs are hypersensitive and have continuous pain in the zone of reference that can be altered with compression, whereas latent TrPs have only hypersensitivity with no continuous pain. This localized tenderness, elicited with both manual palpation and pressure algometers, has been found to be a reliable indicator of the presence and severity of MFP.[32,33] However, the presence of taut bands seems to be a characteristic of skeletal muscles in all people regardless of the presence of MFP.[34]

Palpating the active TrP with sustained deep single-finger pressure on the taut band elicits an alteration of the pain (intensification or reduction) in the zone of reference (area of pain complaint) or cause radiation of the pain toward the zone of reference. This pain can occur immediately or be delayed a few seconds. The pattern of referral is both reproducible and consistent with patterns of other patients with similar TrPs (see **Fig. 1**). This pattern enables clinicians to use the zone of reference as a guide to locate the TrP for purposes of treatment. In contrast, TePs require a standardized palpation at 18 predefined sites, as noted in **Fig. 2** and **Box 2**.

Many of the TePs in the diagnosis of FBM are in similar locations to many TrPs. For example, Simons[49] points out that 16 of the 18 TeP sites in FBM are at well-known TrP sites. Many of the clinical characteristics of FBM, such as fatigue, morning stiffness, and sleep disorders, can also accompany MFP. Bennett[5] compares these 2 disorders and concludes that they are 2 distinct disorders but may have the same underlying pathophysiology. FBM is characterized by more common CNS-generated contributing factors such as sleep disorders, depression, and stress. In contrast, MFP is distinguished by more common regional contributing factors like localized trauma, posture, and muscle tension habits. MFP generally had a better prognosis for treatment than FBM.

The patient's behavioral reaction to this firm palpation is a distinguishing characteristic of MFP and FBM and is termed a jump sign. This reaction may include withdrawal of the head, wrinkling of the face or forehead, or a verbal response such as "That's it" or "Oh, yes." The jump sign should be distinguished from the local twitch response in MFP, which can also occur with palpation. This latter response can be elicited by placing the muscle in moderate passive tension and snapping the band containing the TrP briskly with firm pressure from a palpating finger moving perpendicularly across the muscle band at its

most tender point. This pressure can produce a reproducible shortening of the muscle band (visible in larger muscles) and associated electromyographic changes characteristic of the local twitch response described later. In locating an active TrP, the jump sign should be elicited and, if possible, alteration of the patient's complaint by the palpation.

In FBM, TePs require direct pressure of about 4 kg/cm^2 over the site instead of the snapping palpation with TrPs. In both TrPs and TePs, palpating pressure over neutral areas such as the middle of the forehead can help the patient distinguish between dull pressure and overt tenderness. This distinction gives the examiner an appreciation of the individual's pain threshold and provides a standard pressure to be placed directly over the sites. TePs often elicit a pain directly over the site of the tenderness without the radiation of pain that is often characteristic of TrPs.

Pain Symptoms

The regional pain found with MFP needs to be distinguished from the widespread muscular pain associated with FBM (**Table 1**). In both cases, the pain is often described as a chronic, dull, aching pain and this is central to the diagnosis. These 2 disorders have many similar characteristics and may represent 2 ends of a continuous spectrum, MFP being generated by regional factors and FBM being generated by centrally mediated factors.

There is evidence that supports the pain of MFP being related to and/or generated by the TrP, particularly if it is distant from the TrP. For example, clinical examination of TrPs shows that in accessible muscles palpation of the active TrPs alters, usually intensifying, the referred pain. In addition, injections of local anesthetic into the active TrP reduce or eliminate the referred pain and the tenderness.[50–52] Treatments such as spray and stretch, exercise, or massage directed at the muscle with the TrP also predictably reduce the referred pain.[53] Other evidence to confirm the relationship includes the use of pressure algometry to show a positive correlation between the scope of tenderness and the severity of pain.[54] In addition, the change in scope of tenderness in response to treatment positively correlates with the change in symptom severity (r = .54).[55]

Pain in FBM is stable and consistent, in contrast with MFP, which can vary in intensity and location depending on which muscles are involved. Patients with FBM most often have pain in the low back, neck, shoulders, and hips.[44,45,56] However, these are areas that frequently affect MFP, reflecting the overlap between the two disorders. These studies have also shown that the pain in FBM is considerably more severe over a larger body area than the pain experienced by patients with other nonlocalized rheumatic disease syndromes.

Table 1
Differences between FBM and MFP

	FBM	MFP
Gender	Female/male: 10:1	Female/male: 1:2
Pain	3 of 4 quadrants	Regional related to muscle involved
General fatigue	Yes	No
Unrefreshed sleep	Yes	Situational
Cognitive symptoms (confusion, forgetfulness, inability to concentrate)	Yes	No
TeP distribution	Widespread	Regional in muscle involved
Stiffness and other symptoms	Widespread	Regional
Prognosis	Moderate	Excellent

Contributing Factors

As with all chronic pain conditions, concomitant social, behavioral, and psychological disturbances often precede or follow the development of MFP and other masticatory muscle pain disorders.[54] Patients report psychological symptoms such as frustration, anxiety, depression, and anger if acute cases become chronic. Maladaptive behaviors such as pain verbalization, poor sleep and dietary habits, lack of exercise, poor posture, bruxism, other tension-producing habits, and medication dependencies can also be seen when pain becomes prolonged. Each of these may complicate the clinical picture by perpetuating the pain, preventing compliance with the treatment program, and causing self-perpetuating chronic pain cycles to develop.

Parafunctional muscle tension–producing habits such as back bracing, neck tensing, and teeth clenching can be generated as a form of tension release as well as a learned behavioral response. The relationship between stress and MFP is difficult to assess because stress is difficult to define and major methodological problems exist

in studying stress. Although no evidence suggests a direct causal relationship between stress and MFP, some studies suggest that a correlation does exist between them. There is a higher than normal incidence of psychophysiologic disorders such as migraine headaches, backache, neck pain, nervous asthma, and ulcers in patients with MFP and other muscle disorders, which suggests similar causal factors.[57–62] Also, higher than normal levels of urinary concentrations of catecholamines and 17-hydroxysteriods, which are commonly associated with a high number of stressful events, were found in a group of patients with MFP dysfunction syndrome compared with controls.[63] In addition, stress management interventions frequently provide significant benefit for patients with MFP and other muscle disorders.

Poor muscle health caused by lack of exercise, muscle disuse, or poor posture has also been suggested to predispose the muscle to the development of TrPs and TePs,[64,65] and often arises after muscles have been weakened through immobilization caused by the prolonged use of cervical collars or extended bed rest, for example. Postural discrepancies may also contribute to joint displacement and abnormal functional patterns, which can contribute to abnormal proprioceptive input and sustained muscle contraction in an attempt to correct the poor postural relationships and allow better compensated neuromuscular function. Poor posture caused by a unilateral short leg, small hemipelvis, increased cervical or lumbar lordosis, noncompensated scoliosis, occlusal abnormalities, and poor positioning of the head or tongue have also been implicated.[66]

CAUSE AND PATHOPHYSIOLOGY

The results of this research suggest that an explanatory model can account for the mechanisms in the development of myalgia from its onset to increasing severity found with clinical and chronic cases. It is apparent that both central and peripheral mechanisms are associated with this process but peripheral sensitization may have more prominence in MFP, whereas central sensitization may occur more in FBM.[9]

Peripheral and Central Sensitization

Peripheral strain to the muscles from sustained muscle tension habits, repetitive strain, and injury contribute to localized progressive increases in oxidative metabolism and depleted energy supply (decrease in the levels of ATP, ADP, and phosphoryl creatine, and abnormal tissue oxygenation). This situation results in an increase in muscle nociception, particularly with type I muscle fiber types associated with static muscle tone and posture.[22–24] The muscle tenderness and pain are mediated by type III and IV muscle nociceptors, which may be activated by locally released noxious substances such as potassium, histamine, kinins, or prostaglandins, causing tenderness.[67–69] Phasic modulation of excitatory and inhibitory interneurons supplied by the high-threshold sensory afferents may also become involved, ramping up the peripheral sensitization. Tonic muscular hyperactivity and bracing may be normal protective adaptations to the pain instead of its cause but can still perpetuate the peripheral sensitization. Likewise, interventions that provide counterstimulation to the muscle TrPs, such as massage, temperature change, stretching, electrical stimulation, dry needling, and local anesthetic injections, can decrease peripheral sensitization.

Multiple afferent inputs from the muscle and other visceral and somatic structures in the orofacial area, such as joints, dentition, and periodontal ligaments, converge in lamina I or V of the dorsal horn on the way to the cortex, which can result in both local and referred regional pain.[22] Multiple peripheral and central factors, such as oral habits, anxiety, and stress, may facilitate central input through the modulatory influence of the brain stem, which supports the central biasing mechanism and central sensitization.[56–65] In addition, similar to what happens in the periphery, the central modulatory influences can also decrease central input and inhibit central sensitization. This process may explain the diverse factors that can either exacerbate or alleviate the pain, such as stress, repetitive strain, poor posture, and muscle tension, to increase sensitization; or interventions such as relaxation, medications, counseling, and mindful-based stress reduction that can reduce central sensitization.

Peripheral Changes

The nature of the peripheral neuropathologic and/or dysfunctional processes of MFP TrPs or FBM TePs and the peripheral changes associated with the pain are still not fully understood. Several histologic and biochemical studies have been completed on biopsies of tender muscle sites in patients with both generalized and regional muscle complaints. These studies suggest that there are localized progressive increases in oxidative metabolism, particularly in muscle fiber type I with depleted energy supply, increases in metabolic by-products, and resultant muscle nociception at the periphery. This condition results in

local and referred pain in the CNS that can be altered by a central biasing mechanism that either amplifies or suppresses the pain.

Injury to Muscle Fiber Type I

Each skeletal muscle has different proportions of muscle fiber types that group into 3 broad categories: type I, type IIA, and type IIB (**Table 2**).[70] Type IIC and IIM are involved in development and are not frequently seen in the adult masticatory muscles. Type I muscle fibers are functionally associated with static muscle tone and posture. They are slow-twitch, fatigue-resistant fibers with a high number of mitochondria needed for oxidative phosphorylation used in energy metabolism. Type II fibers are functionally associated with increased velocity and force of contraction over brief periods. They are fast-twitch fibers that fatigue easily, are rich in glycogen, and use anaerobic glycolysis for energy metabolism. These fibers can transform from one type to another depending on the demands placed on the muscle. For example, Uhlig and colleagues[70] found signs of fiber transformation from type I to type IIc in cervical muscles associated with pain and dysfunction after spondylodesis. This finding is consistent with the transformation associated with prolonged inactivity caused by the injury. Furthermore, Mayo and colleagues[71] found decreases in the cross-sectional diameter of muscle fiber types I and II in the masticatory system in rhesus monkeys undergoing maxillomandibular fixation.

Thus, transformation caused by inactivity and pain can decrease both the percentage and size of type I fibers available to maintain normal postural and resting muscle activity. In contrast, an increase in demands of postural muscle activity may result in an increase in type I fibers and a decrease in type II fibers, as found by Bengsston and colleagues[72] in patients with muscle pain.[73] If the increased demand placed on the type I fibers by repetitive strain from activities such as clenching or shoulder tensing is beyond normal physiologic parameters, the intracellular components of these fibers will be damaged. This damage will result in hyperpolarization outside the muscle caused by high levels of K^+ from sustained motor unit activity and K^+ pump damage, damage to the actin and myosin myofilaments, disruption of the sarcoplasmic reticulum and the calcium pump, and decrease in local blood flow. Specific factors that are important in initiating this process included both direct macrotrauma and indirect microtrauma from repetitive muscle strain factors.

Metabolic Distress at the Motor End Plates

In explaining the local nature of MFP TrPs, Simons[74,75] suggested that the damage to the muscle occurs primarily at the motor endplates creating an energy crisis at the TrP. He suggested that this crisis occurs from a grossly abnormal increase in acetylcholine release at the endplate and generation of numerous miniature endplate potentials. This situation results in an increase in energy demand, sustained depolarization of the postjunction membrane, and mitochondrial changes.[50] Hubbard and Berkoff[75] found spontaneous EMG activity at the TrP.[76] Hong[77] and Torigoe found that the EMG characteristics of the local twitch response are generated locally without input from the CNS. Also, botulinum toxin A injections, which act on the neuromuscular junction only, have also been shown to be effective in MFP TrPs.[78]

Histologic studies also provide some support to this mechanism.[67,68,79–82] They have shown myofibrillar lysis, moth-eaten fibers, and ragged red type I fibers with deposition of glycogen and abnormal mitochondria, but little evidence of cellular inflammation hypothesis.[22,68] Studies of muscle energy metabolism found decreases in the levels of ATP, ADP, and phosphoryl creatine and abnormal tissue oxygenation in muscles with TrPs.[73] El-Labban and colleagues[79] showed histologically that TMJ ankylosis results in degenerative changes in masseter and temporalis muscles. It has been hypothesized that these changes represent localized progressive increases in oxidative metabolism and depleted energy supply in type I fibers, which may result in progressive abnormal muscle changes that initially include reactive dysfunctional changes occurring within the muscle, particularly muscle fiber type I and surrounding connective tissue.[80]

Activation of Muscle Nociceptors

The resulting metabolic by-products of this damage, whether by high potassium concentration and hyperpolarization outside the muscle caused by K^+ pump damage, high calcium concentration caused by damage to the sarcoplasmic reticulum, or inflammatory mediators from tissue damage, peripheral sensitization of nociceptors in the muscle, fatigue, and disuse can result.[81] Localized tenderness and pain in the muscle involve type III and IV muscle nociceptors and have been shown to be activated by noxious substances, including K^+, bradykinin, histamine, or prostaglandins, that can be released locally from the damage and trigger tenderness.[67–69,82–84] Note that the K^+ activated a higher percentage of type IV muscle

Table 2
Characteristics of muscle fiber types I, IIA, and IIB in skeletal muscles. Type IIC and IIM are primarily involved in growth and development and are not often seen in skeletal muscles

	Major Fiber Types		
	Type I (Red)	**Type IIA (Pink)**	**Type IIB (White)**
Staining	Weak: ATPase (light pink) Strong NADH-TR (dark pink)	Strong: ATPase (light pink) Strong NADH-TR (dark pink)	Strong: ATPase (light pink) Weak NADH-TR (dark pink)
Contraction speed and fatigue	Slow twitch Without fatigue Gradual recruitment to maximal force	Fast twitch Fatigue resistant Higher threshold to recruitment	Slow twitch Fatigue resistant Develops highest muscle tension
Cellular characteristics	Low glycogen High number of mitochondria High oxidative enzyme levels Slow myosin	Low glycogen Low number of mitochondria Low oxidative enzyme levels Fast myosin	Rich in glycogen Low number of mitochondria Low oxidative enzyme levels Fast myosin
Morphology	1. Less in deep masseter with short face 2. More with loss of teeth	1. More in deep masseter with short face 2. Less with loss of teeth	1. Hypertrophy with long face 2. Less with loss of teeth
Function	Posture Sustained low-force contraction Increased muscle length does not alter function or morphology	Long-term use Sustained high-force contraction Increased muscle length does not alter function or morphology	Strength Brief high-force contraction Increased muscle length does not alter function or morphology
Response to electrical stimulation	At 50 Hz: Type I to II Increase glycogen level Decreased mitochondria	At 10 Hz: Type II to I Decrease glycogen level Increased mitochondria	At 10 Hz: Type II to I Decrease glycogen level Increased mitochondria
Metabolism	Oxidative phosphorylation	Glycolytic	Glycolytic

Data from Graff-Radford SB, Reeves JL, Jaeger B. Management of chronic head and neck pain: effectiveness of altering factors perpetuating myofascial pain. Headache 1987;27(4):186–90.

nociceptors than other agents, providing support that localized increases in K^+ concentration at the neuromuscular junction may be responsible for sensitization of nociceptors. This peripheral sensitization is thought to play a major role in local tenderness and pain, which, together with central sensitization, produces hyperalgesia in patients with persistent muscle pain.

Central Nervous System Changes

The afferent inputs from type III and IV muscle nociceptors in the body are transmitted to the CNS through cells such as those of the lamina I, V, and possibly IV of the dorsal horn on the way to the cortex, resulting in perception of local pain.[85,86] In the trigeminal system, these afferent inputs project to the second-order neurons in the brain stem regions, including the superficial lamina of trigeminal subnucleus caudalis, as well as its more rostral laminae such as interpolaris and oralis.[87,88] These neurons can then project to neurons in higher levels of the CNS such as the thalamus, cranial motor nuclei, or the reticular formation.[88] In the thalamus, the ventrobasal complex, the posterior group of nuclei, and parts of the medial thalamus are involved in receiving and relaying somatosensory information.[89] These inputs can also converge with other visceral and somatic inputs from tissues such as the joint or skin and be responsible for referred pain perception.[90]

Central Biasing of Nociceptive Input

Both FBM and MFP need to be considered as a primary disorders of central pain perception. Although nociceptive input from the periphery do occur, they have been shown to be modified by multiple factors in their transmission to the CNS. For example, low-intensity and high-intensity electrical stimulation of sensory nerves or noxious stimulation of sites remote from site of pain suppress nociceptive responses of trigeminal brain stem neurons and related reflexes.[88] This finding provides support that afferent inputs can be inhibited by multiple peripherally or centrally initiated alterations in neural input to the brain stem through various treatment modalities such as cold, heat, analgesic medications, massage, muscular injections, and transcutaneous electrical stimulation.[68]

Likewise, persistent peripheral or central nociceptive activity can result in an increase in abnormal neuroplastic changes in cutaneous and deep neurons. These neuroplastic changes may include prolonged responsiveness to afferent inputs, increased receptive field size, and spontaneous bursts of activity.[76,89–92] Thus, peripheral inputs from muscles may also be facilitated or accentuated by multiple peripherally or centrally initiated alterations in neural input with further sustained neural activity such as persistent joint pain, sustained muscle activity habits, or postural tension, or CNS alterations such as depression and anxiety that can support the central sensitization, further perpetuating the problem. This sensitization may be subserved by several neuropeptides; for example, substance P, serotonin, acetylcholine, and endorphins. Serotonin, or 5-hydroxytryptamine, is a CNS neurotransmitter that has been shown to have an inverse relationship to the pain of FBM and, with substance P, has been shown to be at increased levels in the cerebral spinal fluid of patients with FBM.[72,73]

These biochemical changes underlie an integrated central biasing mechanism in the CNS that dampens or accentuates peripheral input.[59] This mechanism may explain many of the characteristics of MFP and other muscle disorders, including the broad regions of pain referral, the recruitment of additional muscles in chronic cases, the interrelationship between muscle and joint pain, and the ability of many treatments, including medication, spray and stretch, massage, and TrP injections, to reduce the pain for longer than the duration of action.

There is evidence that patients with FBM may also have an abnormality associated with the immune system that may distinguish patients with FBM from patients with MFP and support the more systemic nature of FM. Several studies have found that most patients with chronic fatigue and immune dysfunction syndrome fulfill the criteria for FBM and that they may have several serum abnormalities of immune function.[67–69] It is suggested that, in some patients with FBM, a herpes simplex viral infectious process initiates the symptoms that lead to a chronic disturbances in both immune system functioning and the mechanisms of sleep and small fiber neuropathic pain. Thus, an antiviral, valacyclovir, in combination with an antiinflammatory medication, such as celecoxib, has been initially shown to be helpful for FM.[83,84]

EVIDENCE-BASED MANAGEMENT

Treatment of MFP can range from simple cases with transient single-muscle syndromes to complex cases involving multiple pain areas and many interrelating contributing factors, including the presence of FBM. Many systematic reviews and randomized controlled trials (RCTs) have shown success in treatment of MFP using a wide variety

of techniques, such as exercise, TrP injections, myotherapy, vapocoolant spray and stretch, transcutaneous electrical nerve stimulation, biofeedback, posture correction, tricyclic antidepressants, muscle relaxants and other medications, and addressing perpetuating factors.[4,6,7,27,78,93–103] For the masticatory muscles, the use of intraoral appliance therapy has also been helpful.[104–110]

However, the difficulty in management lies not in the selection of which treatment to use but in how to educate, engage, and empower patients to reduce the lifestyle risk factors that contribute to its persistence and treatment failure. Results from clinical studies reveal that many patients with MFP have seen many clinicians, and have received numerous medications and multiple other singular treatments for years without receiving more than temporary improvement.[111,112]

These and other studies of chronic pain suggest that, regardless of the pathogenesis of chronic pain, a major characteristic of some of these patients is the failure of traditional biomedical approaches to resolve the problem. Several new strategies are needed in the care model to improve outcomes of chronic pain, including:

- Using an inclusive problem list (physical diagnoses, protective factors, and risk factors) to personalize the care strategy
- Determining the complexity of the patient with risk assessment and using a decision process (tree) to stratify care based on complexity to increase the potential for successful management
- Use a transformative care model that provides patient training on an equal and integrated basis with evidence-based treatment
- Recognizing the role of the health care provider as an agent of change and shift to new chronic illness paradigms

Each of these is discussed later.

Determine a Complete Problem List

The first step in helping patients shift their understanding of their illness so they learn to achieve health and wellness is to establish a complete problem list. The problem list includes both the physical diagnoses (the physical problem responsible for the chief complaint and its associated symptoms) and the list of contributing factors that initiate, perpetuate, or result from the disorder and complicate the problem. Multidimensional risk assessment helps to determine which contributing factors are present. Specific risk factors for chronic pain are included in **Table 3**. These risk factors may range from peripheral factors, such repetitive stress-strain and postural habits, to central mediating factors, such as anxiety and depression, comorbid conditions, somatization, and catastrophizing. Protective factors, such as level of exercise, healthy diet, sleep, coping, self-efficacy, patient beliefs (eg, perceived control over pain), and social support, reduce vulnerability to chronic pain and can create more positive outcomes.

Match the Complexity of Management to the Complexity of the Patient

High treatment failure rates dictate the critical need to match the level of complexity of the management program with the complexity of the patient; the more risk factors and training needed, the more complex the care team. **Fig. 2** describes a hierarchical approach from acute to simple to complex management. Failure to address the entire problem, including all involved muscles, concomitant diagnoses, and risk factors, may lead to failure to resolve the pain, delayed recovery, and perpetuation of the pain. In addition, managing only those patients whose complexity matches the care strategy available to the clinician improves success. Simple cases with minimal behavioral and psychosocial involvement can typically be managed by a single clinician. Complex patients with many risk factors should be managed within an interdisciplinary pain clinic setting that uses a team of clinicians to address different aspects of the problem in a concerted fashion. In both simple and complex cases, each clinician needs to recognize and address the whole problem and use a transformative care model to maximize the potential for a successful outcome.

The difficulty in long-term management of chronic pain often lies not in treating the muscle but in the complex task of training the patient to change the identified risk factors because they can be integrally related to the patient's physical characteristics, attitudes, emotions, lifestyles, and social and physical environment. Interdisciplinary teams integrate various health professionals in a supportive environment to accomplish both long-term treatment of illness and modification of these contributing factors. Many approaches, such as habit reversal techniques, biofeedback, and stress management, have been used to achieve this result within a transformative care approach.

Transformative Care Model

Combining evidence-based biomedical treatments with robust patient training to reduce risk

Table 3
The 7 realms of risk factors and protective factors involved in management of MFP

Realm	Description	Protective Factors that Protect from Delayed Recovery and Chronic Pain	Risk Factors that Increase Risk of Chronic Pain
Body	Physical and physiologic structures of the body	Balanced relaxed posture, stretching, strengthening, and conditioning exercise	Poor posture, tight weak muscles, hypomobile or hypermobile joints, poor conditioning, injury, genetic risk, and comorbidities
Lifestyle	Regular lifestyles and behaviors	Protective diet, good pacing, reduce repetitive strain, staying active, restful sleep, chemical free, and compliance with protective actions	Poor diet, sedentary life, prolonged sitting, poor sleep, hurrying, repetitive strain habits, high-risk behaviors, chemical use
Emotions	Positive and negative feelings and affect	Sustained positive emotions, such as feelings of joy, excitement, calmness, confidence, happiness, and contentment	Prolonged negative emotional experiences: anger, anxiety, sadness, fear, guilt, and depression
Society	Relationships with others	Positive relationships with family, friends, colleagues, community, social support, helping others, work wellness, rewarding recovery	Poor relationships, routine conflict, loss, abuse, posttraumatic stress, low social support, secondary and tertiary gain
Spirit	Beliefs and purpose in life	Purpose, direction, beliefs, faith, hope, self-compassion, self-esteem, inner strength, determination	Stress, feeling lost, burnout, disbelief, cynicism, doubt, hopelessness, resignation
Mind	Thoughts and attitudes	Broad understanding of conditions, resilience, self-efficacy and self-control, accepting responsibility, having realistic expectations, and engaging in active coping	Ignorance or limited understanding of broader problem, low resilience, low self-efficacy/control, refusal of responsibility, poor compliance, unrealistic expectations, and passive coping
Environment	Physical environment	Clean, organized, safe environment and interaction with the environment that is protective, cautious, and careful	Living within an unclean, chaotic, disorganized, negligent, and dangerous environment increases risk of injury and accident

From Preventing chronic pain. International MYOPAIN Society. Available at: https://www.preventingchronicpain.org/drupal/pcpnet/rsrch_cause; with permission.

factors and enhance protective factors can transform a person's life from one beset by illness to one characterized by health and wellness.[14,15] This principle is the basis for a transformative model of care. When self-management is combined with these evidence-based biomedical treatments, outcomes can be dramatically improved and patients are less dependent on the health care system. Transformative care includes the use of risk assessments to identify risk factors as part of the problem list. Personalized care strategies include integrative teams that can be supported by health coaches, social support networks, on-line and in-person patient training programs, and dashboards to document patient engagement and patient-centered outcomes. A transformative care management strategy for masticatory MFP includes 2 components:

1. Treatment with intraoral appliance therapy, medication, physical therapy, and other treatments to reduce the pain and muscle dysfunction.
2. Training on exercises to improve flexibility, function, and pain with cognitive behavior therapy to reduce risk factors and promote protective factors (see **Box 1**).

The short-term goal is to reduce pain by reducing muscle strain and restoring the muscle to normal length, posture, and full joint range of motion. The long-term goal is to prevent delayed recovery by developing a daily exercise routine, such as yoga; maintaining balanced, relaxed postures; and making changes in risk factors permanent. Thus, patient compliance with a new daily routine is important. This outcome can best be achieved if the health care provider becomes an agent of change.

The Health Care Provider as an Agent of Change

Health care providers need to recognize that they are part of the patient's system of health and/or illness. In some cases, dependency on medications, repeated use of interventions and surgery, secondary gain from care-seeking behavior, and rebound pain from drugs can be part of the patient's cycle of problems. If clinicians understand their integral role in the cycle of self-perpetuating illness, they can be part of the solution and help initiate change. Because of the long history of the biomedical model of care, patients often expect to have a passive role in care. To change this, new paradigms described in **Table 4** need to be conveyed to the patient as part of the evaluation. Embracing patient-centered health care paradigms that foster responsibility, education, motivation, self-efficacy, social support, strong provider-patient relationships, and long-term change encourages a passive, dependent patient to become an empowered, engaged, and educated patient.[1,2]

Muscle exercises

The most useful exercise techniques for muscle rehabilitation include muscle stretching;

Table 4
Shifting clinical paradigms associated with transformative care involves each member of the team following the same concepts by conveying the same messages implicit in their dialogue with the patient

New Paradigm	Statement that Shifts to New Paradigm
Understand the whole patient	We will help you identify all diagnoses, risk factors, and protective factors associated with your condition
Each patient is complex	Multiple conditions and interrelated contributing factors may initiate, result from, and increase or decrease risk of illness. Each needs to be addressed as part of management strategy
Self-responsibility is key to recovery	You have more influence on the problem than any treatment provided. Will you take ownership and control of the condition?
Self-care	You will need to make daily changes to improve your condition
Education and training	We will teach you how to make changes to improve your condition
Long-term change	Change only occurs over time, and it may take months for the changes to have a large impact on reducing pain and symptoms
Strong provider-patient partnerships	We as health professionals will support you as you make changes to improve your pain condition
Personal motivation	Will you be able to make the changes needed to achieve wellness?
Social support	You may need help to make the changes required for recovery
Fluctuation of progress	Expect ups and downs during the recovery process

posture; strengthening exercises; and, particularly for FBM, cardiovascular fitness. In patients with both MFP and other muscle disorders, a home program of active and passive muscle stretching exercises reduces the muscle tenderness, whereas postural exercises reduce its susceptibility to flare-ups caused by physical strain. Strengthening and cardiovascular fitness exercises improve circulation, strength, and endurance of the muscles.[6,99,113]

Evaluating the present range of motion of muscles is the first step in prescribing a set of exercises to follow. For example, in the head and neck, range of motion should be determined for the jaw and neck at the initial evaluation. A limited mandibular opening in the jaw indicates whether there are any TrPs within the elevator muscles: temporalis, masseter, and medial pterygoid. If mandibular opening is measured as the interincisal distance, the maximum range of opening is generally between 42 and 60 mm, or approximately 3 knuckle widths (nondominant hand). A mandibular opening with TrPs in the masseter is approximately between 30 mm and 40 mm or 2 knuckle widths. If contracture of masticatory muscles is present, the mandibular opening can be as limited as 10 to 20 mm. Other causes of diminished mandibular opening include structural disorders of the TMJ, such as ankylosis, internal derangements, and gross osteoarthritis.

Passive and active stretching of the muscles increases the opening to the normal range as well as decreasing the pain. Passive stretching of the masticatory muscles during counterstimulation of the TrP can be accomplished through placing a properly trimmed and sterile cork, tongue blades, or other object between the incisors while the spray-and-stretch technique is accomplished. Rapid, jerky stretching or overstretching of the muscle must be avoided to reduce potential injury to the muscle.

Postural exercises are designed to teach patients mental reminders to hold the body in a balanced, relaxed position and to use body positions that afford the best mechanical advantage. This approach includes static postural problems such as unilateral short leg, small hemipelvis, occlusal discrepancies, and scoliosis, or functional postural habits such as forward head, jaw thrust, shoulder phone bracing, and lumbar lifting. In a study of postural problems in 164 patients with head and neck MFP, Fricton and colleagues[103] found poor sitting/standing posture in 96%, forward head in 84.7%, rounded shoulders in 82.3%, lower tongue position in 67.7%, abnormal lordosis in 46.3%, scoliosis in 15.9%, and leg length discrepancy in 14.0%. In improving posture, specific skeletal conditions such as structural asymmetry or weakness of certain muscles need to be considered. In the masticatory system, patients should be instructed to place the tongue gently on the roof of the mouth and keep the teeth slightly apart. In the cervical spine, a forward or lateral head posture must be corrected by guiding the chin in and the head vertex up. The shoulders naturally fall back if the thorax is positioned up and back with proper lumbar support. Patients need to be instructed in proper posture for each position (sitting, standing, and lying down), as well as in movements that are done repetitively throughout the day, such as lifting or turning the head to the side. Sleeping posture on the side or back is particularly important for patients who wake up with soreness. Improved posture is also facilitated by regular physical conditioning. Patients need to be placed on a conditioning program to facilitate increased aerobic capacity and strength. Aerobic programs, such as exercise classes, regular running, walking, biking, or swimming, improve comfort, endurance, and functional status of patients with MFP.

Muscle treatments

There are many methods suggested for providing repetitive stimulation to tender muscles. Massage, acupressure, and ultrasonography provide noninvasive mechanical disruption to inactivate the TrPs. Moist heat applications, ice pack, Fluori-Methane, and diathermy provide skin and muscle temperature change as a form of counterstimulation. Transcutaneous electrical nerve stimulation, electroacupuncture, and direct current stimulation provide electric currents to stimulate the muscles and TrPs. Acupuncture, TrP injections of local anesthetic, corticosteroids, or saline cause direct mechanical or chemical alteration of TrPs. However, the 2 most common techniques for treating TrPs are the spray-and-stretch technique and TrP injections, and these are discussed here.

With the spray-and-stretch technique, an application of a vapocoolant spray such as Fluori-Methane over the muscle with simultaneous passive stretching can provide immediate reduction of pain, although lasting relief requires a full management program.[24,53] The technique involves directing a fine stream of Fluori-Methane spray from the finely calibrated nozzle toward the skin directly overlying the muscle with the TrP. A few sweeps of the spray are first passed over the TrP and zone of reference before adding sufficient manual stretch to the muscle to elicit pain and discomfort. The muscle is put on a progressively increasing passive stretch while the jet stream of spray is directed at an acute angle 30 to 50 cm (1–1.5 feet) away. It is applied in

1 direction from the TrP toward its reference zone in slow, even sweeps over adjacent parallel areas at a rate of about 10 cm/s. This sequence can be repeated up to 4 times if the clinician warms the muscle with a hand or warm moist packs to prevent overcooling after each sequence. Frosting the skin and excessive sweeps should be avoided because these may reduce the underlying skeletal muscle temperature, which tends to aggravate TrPs. The range of passive and active motion can be tested before and after spraying as an indication of responsiveness to therapy. Failure to reduce TrPs with spray and stretch may be caused by (1) inability to secure full muscle length because of bone or joint abnormalities, muscle contracture, or the patient avoiding voluntary relaxation; (2) incorrect spray technique; or (3) failure to reduce perpetuating factors. If spray and stretch fails with repeated trials, direct needling with TrP injections may be effective.

TrP injections have also been shown to reduce pain, increase range of motion, increase exercise tolerance, and increase circulation of muscles.[50–52] The pain relief may last from the duration of the anesthetic to many months, depending on the chronicity and severity of TrPs, and the degree of reducing perpetuating factors. Because the critical factor in relief seems to be the mechanical disruption of the TrP by the needle, precision in needling of the exact TrP and the intensity of pain during needling seem to be the major factors in TrP inactivation. TrP injections with local anesthetic are generally more effective and comfortable than dry needling or injecting other substances, such as saline, although acupuncture may be helpful for patients with chronic TrPs in multiple muscles. The effect of needling can be complemented with the use of local anesthetics in concentrations less than those required for a nerve conduction block, which can markedly lengthen the relative refractory period of peripheral nerves and limit the maximum frequency of impulse conduction. Local anesthetics can be chosen for their duration, safety, and versatility. Three percent chloroprocaine (short acting) and 5% procaine (medium acting) without vasoconstrictors are suggested.

Intraoral appliance therapy

Several systematic reviews of RCTs have found that stabilization appliances, when adjusted to be comfortable and used daily, have good evidence of efficacy in the treatment of masticatory MFP compared with nonoccluding appliances and no treatment. They are also at least equally effective in reducing MFP compared with physical therapy, behavioral therapies, and pharmacologic treatment. Other types of appliances, including soft stabilization appliances, anterior positioning appliances, and anterior bite appliances, have some RCT evidence of efficacy in reducing pain from TMJ disorders. However, the potential for adverse events with these appliances is higher and close monitoring is suggested in their use. There are no studies that suggest maxillary or mandibular appliances have more efficacy than the other; this depends on patient and clinician preference and comfort.

Hard acrylic full-coverage intraoral appliances are intended to reduce MFP and dysfunction by producing orthopedically comfortable jaw positions, reducing masticatory muscle activity and TMJ loading, and increasing patients' awareness of oral parafunctional habits. They also can prevent tooth wear and periodontal trauma. In most cases, stabilization appliances are comfortable to wear unless they are bulky, tight, or ill-fitting, so they need to be well adjusted to facilitate patient comfort, stability, and compliance. Although the percentage of adverse events was not provided in most of these studies, patients should be monitored regularly for evidence of mucosal ulceration or inflammation, tooth pain, mouth odors, speech difficulties, dental caries, tooth mobility, and occlusal changes. Soft, resilient, full-coverage appliances may be less expensive than hard stabilization appliances but need to be adjusted similarly to hard appliances to allow comfort and efficacy.[51,52]

Pharmacotherapy

Pharmacotherapy is a useful adjunct to initial treatment of MFP and other muscle disorders. The most commonly used medications for pain are classified as nonnarcotic analgesics (nonsteroidal antiinflammatories), narcotic analgesics, muscle relaxants, tranquilizers (ataractics), sedatives, and antidepressants. Analgesics are used to allay pain; muscle relaxants and tranquilizers are used for anxiety, fear, and muscle tension; sedatives for enhancing sleep; and antidepressants for pain, depression, and enhancing sleep.[85] Randomized clinical trials on nonsteroidal antiinflammatory drugs (NSAIDs) such as ibuprofen or piroxicam suggest that, for myalgia, short-term use of these medications for analgesic and/or antiinflammatory effects can be considered as a supplement to overall management.[114] Chronic, long-term use requires caution because of the long-term systemic and gastrointestinal effects. However, cyclooxygenase-2 inhibitors (Rofecoxib, Vioxx) have recently become available and these may prove to be safer NSAIDs for long-term use with less gastrointestinal toxicity. If some

therapeutic result is not apparent after 7 to 10 days or if the patient develops any side effects, especially gastrointestinal symptoms, the medication should be discontinued.

For MFP, especially with limited range of motion, benzodiazepines, including diazepam and clonazepam, have been shown to be effective.[115] Experience suggests that these are best used before bedtime to minimize sedation while awake. Cyclobenzaprine (Flexeril) has been shown, in clinical trials of myalgia, to be efficacious in reducing pain and improving sleep.[112,114,115] These medications, with or without NSAIDs, can also be considered for a 2-week to 4-week trial with minimal dependency potential. However, long-term use has not been adequately tested.

For chronic pain conditions that are resistant to interventions, short-term use of opioids can be considered but this is discouraged as a long-term solution because of the adverse events, increasing pain, and abuse potential. Tramadol has been shown to be effective in FBM.[112] However, there are no RCTs evaluating the appropriateness of opioids in the long-term treatment of chronic MFP pain. At this time, chronic opioid use is mainly indicated for patients with chronic, intractable, severe pain conditions that are refractory to all other reasonable treatments. Despite the advantages of medications for pain disorders, there exists an opportunity for problems to be caused by their misuse. The problems that can occur from use of medications include chemical dependency, behavioral reinforcement of continuing pain, inhibition of endogenous pain relief mechanisms, side effects, and adverse effects from the use of polypharmaceuticals. For this reason, use of medication should proceed with caution with MFP.

Training to reduce risk factors

One of the common causes of failure in managing MFP and other muscle disorders is failure to recognize and subsequently control risk factors that may perpetuate muscle restriction and tension. As noted earlier, postural factors, whether behavioral or biological, perpetuate muscle pain if not corrected. In general, a muscle is more predisposed to developing problems if it is held in sustained contraction in the normal position and, especially, if it is in an abnormally shortened position. Such a situation exists with structural problems such as loss of posterior teeth, an excessive lordosis of the cervical spine, a unilateral short leg, or a small hemipelvis. An occlusal imbalance can be corrected with an occlusal stabilization splint, also termed a flat-plane or full-coverage splint. Other postural factors that can be corrected include a foot lift for a unilateral leg length discrepancy, a pelvic lift for a small hemipelvis, and proper height of arm rests in chairs for short upper arms.

Behavioral risk factors causing sustained muscle tension can also occur with habits such as a receptionist cradling a phone between the head and shoulder for hours each day, a student typing on a computer for hours at a time, or day or nighttime oral parafunctional habits. Correcting poor habits through education, training, and long-term reinforcement is essential to preventing MFP from returning. Biofeedback, meditation, stress management, psychotherapy, antianxiety medications, antidepressants, and even placebos have been reported to be effective in treating MFP and other muscle disorders.[92–95] Many of these treatments are directed toward reducing muscle tension–producing habits such as bruxism or bracing of muscles. Teaching control of habits is a difficult process because of the relationship that muscle tension may have to psychosocial factors. Simply telling patients to stop the habits may be helpful with some but with others may result in noncompliance, failure, and frustration. An integrated transformative care approach involving education, increased awareness, and patient training such as cognitive behavior therapy, biofeedback, and mindfulness-based stress reduction has the best potential for success.

Interdisciplinary team management

Although each clinician may have limited success in managing whole patients alone, a team approach can address different aspects of the problem with different health professionals in order to enhance the overall potential for success.[94,116–120] Although these programs provide a broader framework for treating complex patients, they have added another dimension to the skills needed by clinicians: working as part of a coordinated team. Failure to adequately integrate care may result in poor communication, fragmented care, distrustful relationships, and eventually confusion and failure in management. However, team coordination can be facilitated by a well-defined evaluation and management system that clearly integrates team members. **Fig. 3** provides a patient flow from evaluation to assessment to treatment to follow-up.

A prerequisite to a team approach is an inclusive medical model and conceptual framework that places the physical, behavioral, and psychosocial aspects of illness on an equal and integrated basis.[12,14–17] With an inclusive theory of human systems and their relationship to illness, patients can be assessed as whole persons by different

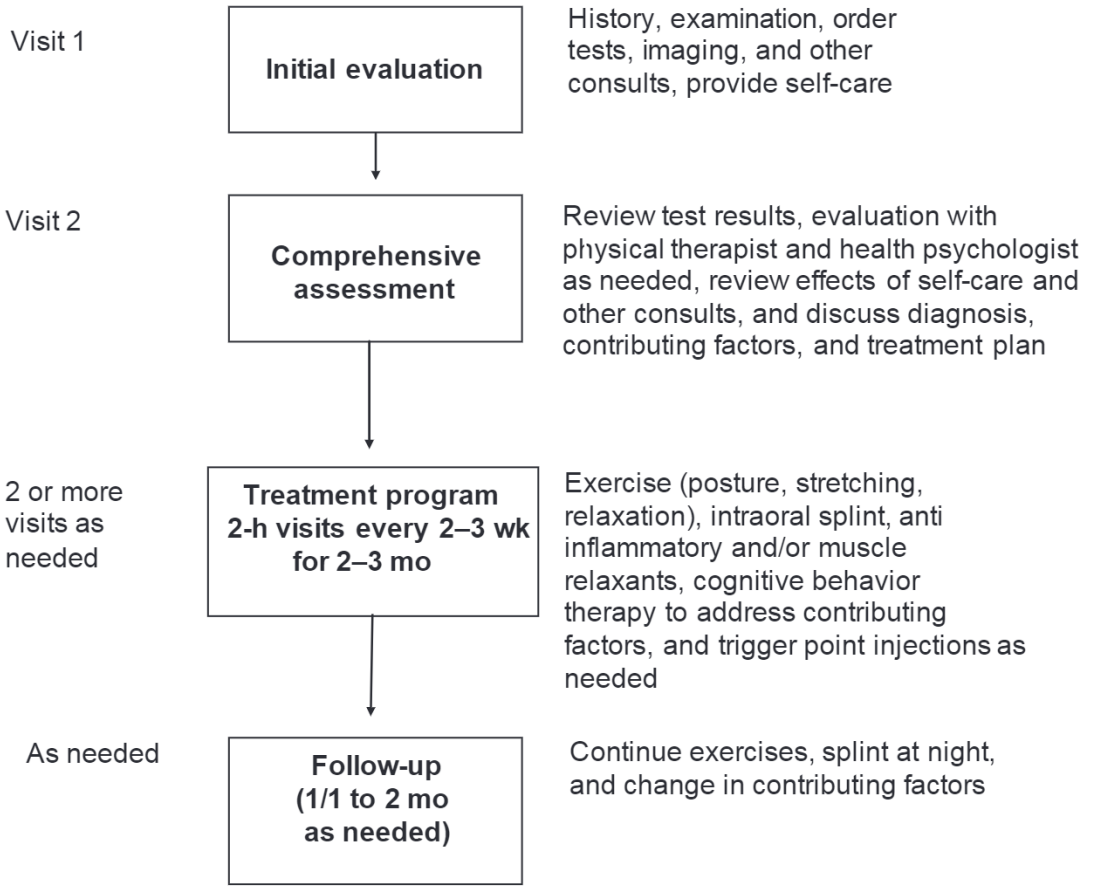

Fig. 3. Patient flow from evaluation to assessment to treatment includes many components. The key to successful management lies in matching the patient's needs with the unique combination of active treatment, education on contributing factors, and self-care appropriate for that patient.

clinicians from diverse backgrounds. Although each clinician understands a different part of the patient's problem, these can be integrated with other clinicians' perspectives to see how each part is interrelated in the whole patient. For example, a physician or dentist evaluates the physical diagnosis, a physical therapist evaluates poor postural habits, and a psychologist evaluates behavioral problems or social stressors. Each factor becomes part of the problem list to be addressed in the treatment plan. In the process, the synergism of each factor in the cause of the disorder can become apparent to clinicians. For example, social stressors can lead to anxiety, anxiety can lead to poor posture and muscle tension, and the poor posture and muscle tension can lead to MFP syndrome; the pain then contributes to more anxiety, and a cycle continues. Likewise, a reduction of each factor works synergistically to improve the whole problem. Treatment of only 1 factor may improve the problem, but relief may be partial or temporary. Treatment of all factors simultaneously can have a cumulative effect that is greater than the effects of treating each factor individually.

As noted, the problem list for a patient with a specific chronic illness includes both a physical diagnosis and a list of risk factors and protective factors. In establishing the problem list, the clinician needs to determine whether the patient is complex and requires a team approach. Recommended criteria for determining complexity include any of the following: multiple diagnoses, persistent pain longer than 6 months in duration, significant emotional problems (depression, anxiety), frequent use of health care services or medication, daily oral parafunctional habits, and significant lifestyle disturbances. The use of a screening instrument can readily elicit the degree of complexity of a case at initial evaluation (see www.preventingchronicpain.org). The more complex the case is, the greater the need for a

team approach. The decision to use a team must be made at the time of evaluation and not part way through a failing singular treatment plan. If a team is needed, the broad understanding of the patient is then used to design a long-term management program that both treats the physical diagnosis and helps reduce these contributing factors.

The primary goals of the program include reducing the pain and dysfunction while helping the patient return to normal daily activities without the need for future health care. The dentist or physician is responsible for establishing the physical diagnosis, providing short-term treatment, and monitoring patient progress. The health psychologist is responsible for providing training on reducing risk factors and enhancing protective factors; diagnosing, managing, or referring for primary psychological disturbances. A health coach can also provide support to the patient and family in making changes. The physical therapist is responsible for providing support, training, and a management program specifically designed to improve protective factors, such as an exercise and posture program. Depending on the therapist's background and the patient's needs, this person may also provide special care, such as physical therapy modalities or occupational therapy. Each clinician is also responsible for establishing a trusting, supportive relationship with the patient while reaffirming the self-care philosophy of the program, reinforcing change, and ensuring compliance. The patient is viewed as responsible for making the changes (see **Table 4**).

SUMMARY

MFP is a regional muscle pain disorder characterized by localized muscle tenderness and pain and is the most common cause of persistent regional pain. FBM is a widespread pain disorder characterized by sleep disturbance, fatigue, cognitive symptoms, and often psychological distress. The affected muscles in both disorders may also have increased fatigability, stiffness, subjective weakness, pain in movement, and slightly restricted range of motion that is unrelated to joint restriction. They are frequently overlooked as diagnoses because they are often accompanied by signs and symptoms in addition to pain, coincidental disorders, and behavioral and psychosocial problems. As these disorders persist, chronic pain characteristics often precede or follow their development.

Management of both disorders includes exercise, therapy to the TrPs, training the patient in reducing risk factors and strengthening protective factors, and selective treatments. The difficulty in management lies in the need to integrate training with treatments to educate, engage, and empower patients to reduce the lifestyle risk factors that contribute to their persistence. This requirement dictates the critical need to match the level of complexity of the management program with the complexity of the patient; the more risk factors and training needed, the more complex the care team. Failure to address the entire problem through a team approach if needed may lead to failure to resolve the pain and the perpetuation of chronic pain.

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