Cervical Myofascial Pain and Headache

Joanne Borg-Stein, MD

Address
Spaulding-Wellesley Rehabilitation Center,
65 Walnut Street, Wellesley, MA 02481, USA.
E-mail: jborgstein@partners.org

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Myofascial pain is a common cause of regional chronic pain. Myofascial trigger points can refer pain to the head and face in the cervical region, thus contributing to cervicogenic headache. When identified properly, cervical myofascial pain is a treatable component of headache management. This article reviews current literature on the pathophysiology, diagnosis, and management of cervical myofascial pain.

Introduction

Myofascial pain is traditionally defined as pain that derives from myofascial trigger points (MTrPs), 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 [1••]. Myofascial pain is highly prevalent among patients with regional pain complaints; 85% to 93% of patients in pain management centers have myofascial pain [2].

More broadly, many clinicians and investigators refer to muscle pain, sensitization, tenderness, and muscle tension. This is relevant in the commonly described pericranial muscle activity of tension-type headache [3]. Although there is clinical overlap among these syndromes, it is useful to focus attention on the clinical entity of cervical myofascial pain and its relationship to headache. With this construct, headache specialists can offer specific pharmacologic, manual, injection, and exercise treatments to reduce the burden of pain.

Epidemiology

Myofascial pain is very common in adult patients with musculoskeletal pain; it may affect 37% of men and 65% of women with pain [4]. There is a prevalence of latent trigger points in the neck and shoulder girdle of young adults who are asymptomatic (45% to 55%) [5]. 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; it is not associated with spontaneous pain. Active trigger points are associated with a clinical pain complaint.

Clinical Characteristics

Patient presentation and symptoms

Patients with myofascial pain have chronic regional pain. Their pain may be of insidious onset or may occur after a specific local trauma. Classically, the complaint is one of a deep regional ache that is often accompanied by a sensation of pulling or tightness. The intensity can vary from mild to severe [6]. Each MTrP has a characteristic pain referral pattern; therefore, the distribution of pain can help prompt a search for the muscle of origin of the trigger point [2,7]. Frequently, associated autonomic dysfunction may occur, including abnormal sweating, lacrimation, dermal flushing, and vasomotor and temperature changes [8]. Cervical myofascial pain may be associated with neuro-otologic symptoms including imbalance, dizziness, and tinnitus. Functional complaints include impaired muscle coordination, stiff joints, muscle fatigue, and weakness. Other associated neurologic symptoms may include paresthesias, numbness, blurred vision, twitches, and trembling [8]. Later stages can be compounded by sleep disturbance, mood changes, and stress [9,10].

The pain and symptoms of myofascial pain syndrome should be differentiated from fibromylagia syndrome, which is a widespread chronic pain disorder characterized by widespread muscle pain, fatigue, sleep disturbance, and 18 paired tender points in a widespread distribution [11••].

Physical examination

For a patient with headache and myofascial pain, the physical examination must begin with a careful medical and neurologic examination. A thorough musculoskeletal examination of the spine and shoulders should then be performed. The clinician analyzes posture, biomechanics, joint function, muscle strength, and imbalances to identify underlying correctable factors that may contribute to the development of regional pain.

Following this, the physician identifies active MTrPs in one or several muscles that may contribute to cervical, head, and possibly facial pain. Four of the most common muscles that harbor MTrPs in patients with headache include the temporalis, trapezius (upper), splenius capitus, and sternocleidomastoid (Fig. 1) [12].

The trigger point should be identified by gentle palpation across the direction of the muscle fibers. The

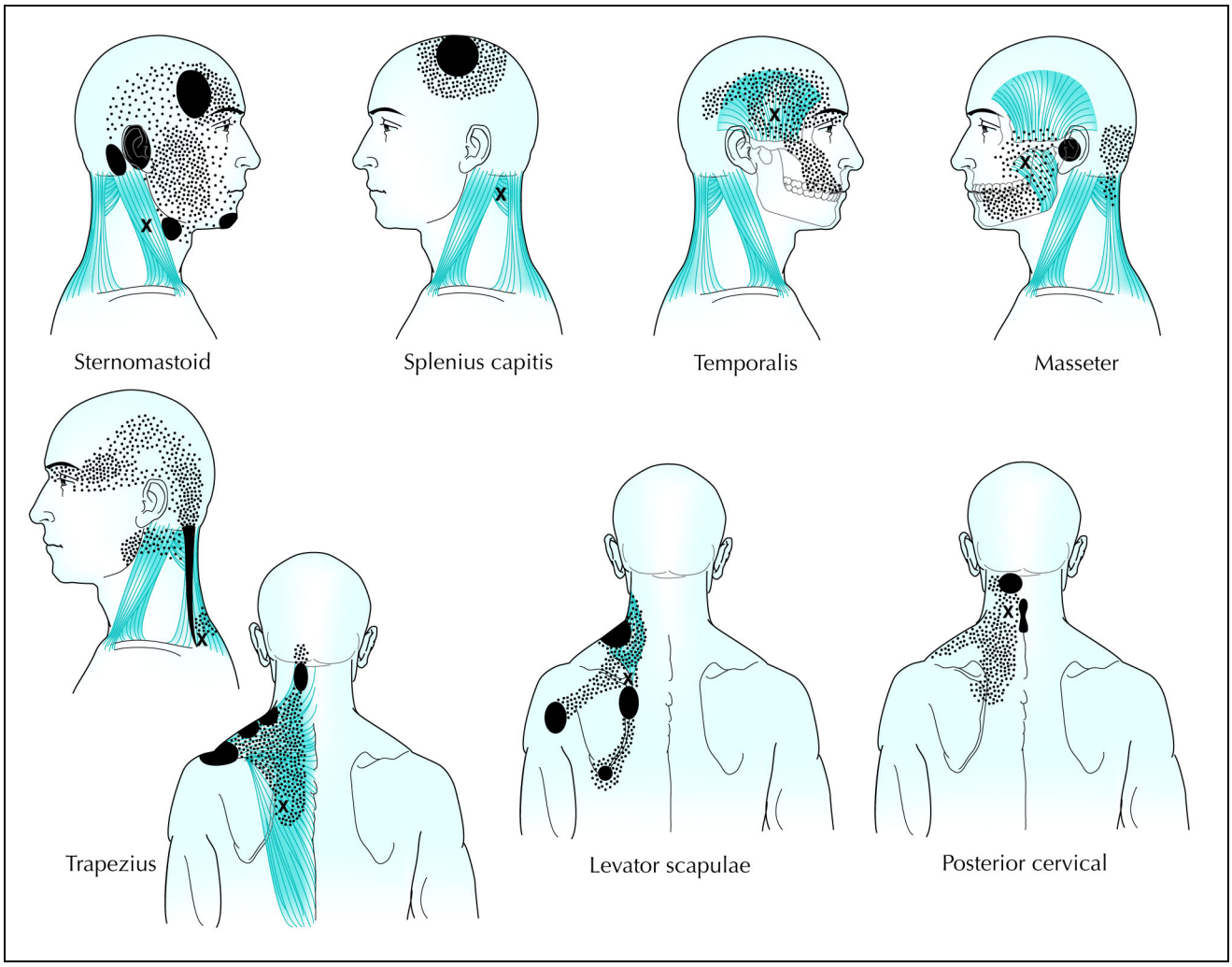

Figure 1. Myofascial pain patterns showing the trigger point (X) and its pain referral pattern (solid black stripping). (*Adapted from* Simons [12].)

examiner appreciates a "rope-like" nodularity to the taut band of muscle. Palpation of the area is painful and reproduces the patient's local and referred pain pattern [11••]. The ability to identify MTrPs depends on the palpation skills and training of the examiner and the depth and size of the muscle [13]. The patient will often have a restricted ROM of the muscle that contains the trigger point, thus providing a clue to the clinician as to where the trigger points are located. [14]

Differential Diagnosis

The differential diagnosis of myofascial pain is broad; it includes overlapping causes of regional musculoskeletal pain and headache. The following questions may be useful in distinguishing the contribution of myofascial pain to the patient's symptoms. Is there regional myofascial pain with trigger points? Is the myofascial pain the primary pain generator or are there other coexisting or underlying structural diagnoses? Is there a nutritional, metabolic, endocrinologic, psychologic, or inflammatory disorder that may be contributing to the regional pain? Is there widespread pain and other associated symptoms?

The differential diagnosis of cervical myofascial pain and headache should include (but is not limited to) the following:

1. Other headache types: chronic tension-type headaches with pericranial tenderness, headaches caused by joint dysfunction involving the upper cervical synovial joints, occipital neuralgia, and head pain associated with temporomandibular disorders.
2. Inflammatory disorders: polymyositis, polymyalgia rheumatica, temporal arteritis, and rheumatoid arthritis.
3. Neurologic disorders: radiculopathy and entrapment neuropathy.
4. Discogenic disorders: degenerative disc disease, annular tears, protrusion, and herniation.
5. Mechanical stresses: postural dysfunction and poor ergonomic worksite.

6. Endocrine: hypothyroidism
7. Psychologic disorders: depression, anxiety, and disordered sleep.
8. Fibromyalgia or widespread chronic pain [15].

Pathophysiology of Myofascial Pain

Current concepts of chronic myofascial pain generally incorporate a complex interplay between peripheral nociception and central sensitization [16]. In this review, current pathophysiologic concepts are organized by neuroanatomic location so that they are easier to understand; however, physicians should appreciate that these processes are interrelated and should be considered in an integrated fashion.

Motor endplate

The concept of a pathologic increase in the release of acetylcholine (Ach) by the nerve terminal of an abnormal motor endplate under resting conditions has been supported by electrodiagnostic evidence [17–19]. This abnormality is considered to be the primary dysfunction in the "integrated hypothesis," which was proposed by Simons *et al.* [1••,11••] and which postulates a positive feedback loop (Fig. 2). An increase in endplate noise (EPN) has been reported more frequently in MTrPs than in the endplate zone outside of the MTrP [18,19]. An increase in EPN has been seen in response to many types of mechanical stimulation of the endplate structure; it may not be specific to myofascial pain [20].

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 *et al.* [1••], is diagrammed in Figure 3. There is histologic support for this hypothesis in canine and human cross-sections of MTrPs [21,22].

A consequence of a chronically sustained sarcomere shortening may be increased local energy consumption and reduction of local circulation, a combination that produces local ischemia and hypoxia [23]. 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 account for local MTrP tenderness [11••].

Central mechanisms: spinal and supraspinal

The referred pain resulting from MTrPs stems from central convergence and facilitation. Convergent connections from deep muscle afferent nociceptors to dorsal horn neurons are facilitated and amplified in the spinal cord under pathologic conditions. Referral to adjacent myotomes results from the spreading of central sensitization to adjacent spinal segments [24,25]. This pattern results in referred pain and in expansion of the region of pain beyond the initial nociceptive region.

At the level of the central nervous system, neuroplastic changes occur in the second-order dorsal horn neurons and in trigeminal nucleus structures, which produce a long-lasting increase in the excitability or nociceptor pathways. This central sensitization involves neurotransmitters such as substance P, *N*-methyl-D-aspartate, glutamate, and nitric oxide [16,26]. In addition, there may be impairments in supraspinal, inhibitory, descending pain-control pathways that release inhibitory neurotransmitters such as gamma-aminobutyric acid, serotonin, and norepinephrine [27].

Autonomic nervous system

Under pathologic conditions, the neurovasoreactive substances, such as bradykinin, substance P, serotonin, and histamine, stimulate activity of the local autonomic nervous system fibers to release more Ach. This completes the positive feedback loop as diagrammed in Figure 1. There seems to be a relationship between autonomic activation and increased motor endplate activity [28,29].

The sympathetic nervous system presumably plays a role in the commonly described findings of painful skin rolling, hypersensitivity to touch, temperature and blood flow changes, abnormal sweating, reactive hyperemia, dermatographia, and altered pilomotor responses that are associated with myofascial pain [30].

Neuroanatomic Basis of Cervical Myofascial Pain and Headache

There are good neuroanatomic and neurophysiologic studies in animal models that establish the convergence of cervical sensory and muscle afferent input onto trigeminal subnucleus caudalis nociceptive and non-nociceptive neurons. Stimulation of the supraorbital nerve and the infraorbital nerve elicits responses in splenius and trapezius motor neurons [31].

There are neurons in the spinal cord that respond to electrical stimulation of the trigeminal nerve and of the cervical nerves. In particular, overlap between terminals of the upper three cervical segments and terminals of the trigeminal nerve provide the neuranatomic basis for cervical myofascial pain, causing headache [32]. Some of the muscles commonly involved include the sternocleidomastoid (supplied by C1, C2), the trapezius (supplied by C1, C2), the splenius capitus and the cervicus (supplied by C2, C3), and the semispinalis capitus and the cervicus (supplied by C3, C3).

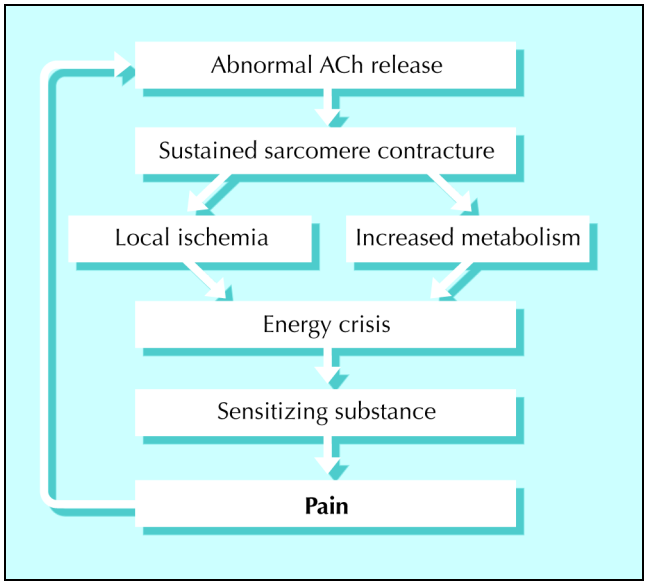

Figure 2. Positive feedback cycle that summarizes the integrated hypothesis. An increase in the release of acetylcholine (ACh) at the motor endplate resulting from mechanical trauma or chemical stimulation of the nerve terminal induces sustained sarcomere contraction. This occurrence results in localized ischemia, which results in the release of substances that sensitize nociceptors, produce pain, and induce release of neurovasoreactive chemicals. These chemicals lead to increases in acetylcholine release, sustaining the cycle. (*Adapted from* Simons *et al.* [1••]; with permission.)

Treatment of Myofascial Pain

Pharmacologic treatment of myofascial pain

Given the considerable clinical overlap among myofascial pain, fibromylagia, regional soft tissue pain, and tension headache, agents that are beneficial in the treatment of an associated syndrome may be useful in treating myofascial pain. In the absence of controlled data that specifically examine drug efficacy in myofascial pain, experienced headache 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 studies [33–35] have found NSAIDs to have a small supplemental benefit in the management of fibromyalgia pain. NSAIDs may also play a role in the treatment of patients who have concomitant cervical osteoarthritis.

Tramadol

Tramadol is a combination of a weak mυ opioid agonist and serves as an inhibitor of the reuptake of serotonin and norepinephrine in the dorsal horn. There are no published controlled trials of tramadol as a treatment for myofascial pain; however, several studies support its efficacy in the treatment of fibromyalgia, chronic back pain, and osteoarthritis, all of which are commonly associated with myofascial pain [36–40].

Antidepressants

Tricyclic antidepressants (*eg,* amitriptyline) are shown to be effective in the treatment of chronic tension-type headache, fibromyalgia, acute low back pain, and intractable pain syndromes with muscle spasm [41–44]. Selective serotonin reuptake inhibitors have not been specifically studied for myofascial pain, although efficacy has been documented in fibromyalgia for improving pain, sleep, and providing a global sense of well-being. [41,45]

α-2 adrenergic agonists

The two major α-2 adrenergic agonists available for clinical use are clonidine and tizanidine. Tizanidine centrally acts at the level of the spinal cord to inhibit spinal polysynaptic pathways and to reduce the release of aspartate, glutamate, and substance P [46,47]. Additionally, tizanidine has supraspinal effects that increase nociceptive thresholds and inhibit the responses of spinal neurons [48]. In one study of tizanidine, a mixed population of patients with myofascial pain syndrome or fibromyalgia were observed; tizanidine treatment reduced pain [49].

Anticonvulsants

There are no controlled trials focusing on anticonvulsants in the treatment of myofascial pain. One open-label study of gabapentin, used in the treatment of chronic daily headache, found possible efficacy [50]. Administration of gabapentin and topiramate may be considered in the treatment of coexistent migraine or neuropathic pain [14].

Botulinum toxin

Botulinum toxin type A appears to be emerging as a promising but expensive new agent used to treat chronic myofascial pain syndromes. In two recent studies of myofascial pain [51,52], botulinum toxin injections provided greater relief of pain symptoms compared with placebo. In another study, Wheeler *et al.* [53] was unable to demonstrate a statistically significant difference compared with placebo. There may be a peripheral and central mechanism that explains the apparent efficacy of botulinum toxin in the treatment of chronic myofascial pain [54].

Nonpharmacologic treatment of myofascial pain

Postural, mechanical, and ergonomic modifications

Cervical muscle dysfunction of insidious onset may be caused and perpetuated by abnormal postures, especially those related to poor ergonomics at home and in the workplace. Correction of awkward postures and ergonomic management is often an effective component of treatment [55].

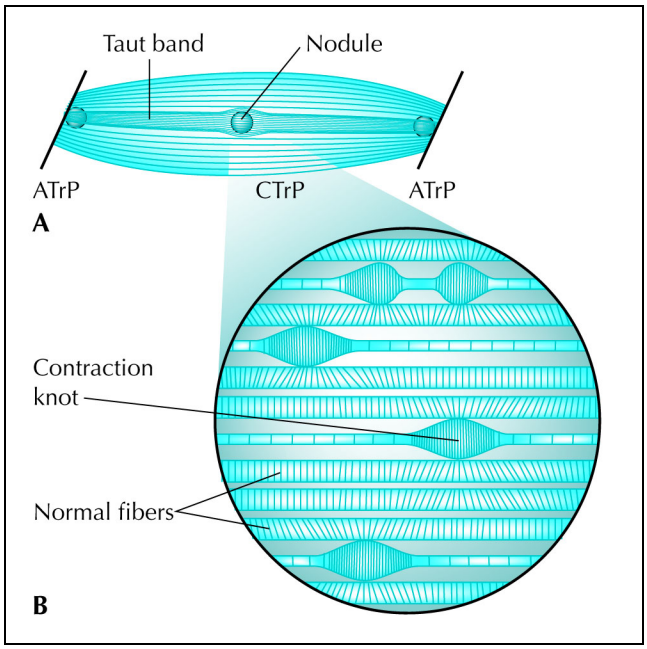

Figure 3. Trigger point complex. **A,** The band of dark muscle fibers running the length of the muscle represents the increased tension of palpable taut band fibers. The nodular region in the middle of that band represents a central trigger point (CTrP) that is exquisitely tender and may be palpable as a nodule in the taut band. The regions at each end of the taut band represent attachment trigger points (ATrPs). ATrPs result in an inflammatory reaction (enthesopathy) at the musculotendinous junctions of the taut band muscle fibers as a result of the sustained tension in those fibers. **B**, The microscopic view of the CTrP shows several swollen contraction knots, which represent the result of the acetylcholine-induced maximal sarcomere contraction in the region of an endplate. The dense region of contracted sarcomeres increases tension in the fiber and causes compensatory stretching of the remaining sarcomeres of the fiber, which increases their resting tension. Involved fibers have an uneven distribution of fiber length.

Stress reduction

Stress reduction techniques, including cognitive-behavioral programs, meditation, progressive relaxation training, and biofeedback, should be incorporated into chronic pain rehabilitation programs [56].

Electromyographic biofeedback and meditation-based stress reduction programs have been effective in the treatment of fibromyalgia [57,58].

Acupuncture

A growing body of evidence supports the efficacy of acupuncture in the treatment of myofascial pain and fibromyalgia. The limited amount of high-quality data suggests that traditional acupuncture is more effective for relieving pain, improving global ratings, and reducing morning stiffness in fibromyalgia [59]. The 1997 National Institute of Health Consensus Statement of Acupuncture [60] concluded that "acupuncture may be useful as an adjunct treatment or an acceptable alternative to be included in a comprehensive management program" for the treatment of fibromyalgia, myofascial pain, low back pain, osteoarthritis, and lateral epicondylitis. Birch and Jamison [61] found relevant acupuncture (over points relevant to myofascial neck pain) to be superior to NSAID treatment and irrelevant acupuncture (superficial needling over points not related to neck pain) in a group of 46 patients with chronic myofascial pain. Questions that need to be answered in future randomized 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, transcutaneous electrical nerve stimulation, and ultrasonography

Although studies suggesting efficacy of massage as part of a treatment program for myofascial pain are scant, a recent study by Gam *et al.* [62] demonstrated that massage combined with stretching exercises was more effective than the control group in reducing the number and intensity of MTrPs.

Transcutaneous electric nerve stimulation treatment has shown mixed results in the treatment of myofascial pain [63,64]. As a noninvasive, independent, self-management option for patients, it is reasonable to use this as an adjunct to other active treatments.

In one randomized controlled trial [62], ultrasonography used in combination with massage and exercise had no additional benefit over sham ultrasonography for the treatment of MTrPs.

Exercise for myofascial pain

Stretching exercises form the basis of exercise treatment of myofascial pain. This treatment addresses muscle tightness and shortening that are closely associated with pain in this disorder and permits gradual restoration of normal activity. A slow, sustained stretch throughout the available ROM is the most effective approach.

Patients should be encouraged to remain active, but perform daily activities in a gentle, lightly loaded manner. After pain from MTrPs is reduced and ROM restored, a graded stabilization and strengthening program should be undertaken to maximize the functional outcome. Aerobic exercise should be included in the overall musculoskeletal and cardiovascular fitness program to prevent recurrence [65].

Trigger point injection for cervical myofascial pain and headache

Trigger point injections are useful for areas of recalcitrant myofascial pain. The patient should be informed that this treatment has a limited role in the long-term management of myofascial pain, but will reduce the pain and facilitate an active exercise and self-management program. Three consecutive injections are often recommended in chronic myofascial pain; reassessment after the third injection is necessary to evaluate the efficacy of the injections and to determine if further injections are needed.

Myofascial trigger point injections may employ several medications, including short- or long-acting anesthetics, steroids, botulinum toxin, or dry needling (no medication). Physicians may use a number of techniques when administering injections, including slow search, fast in-fast out, superficial dry needling, intramuscular stimulation, twitch-obtaining intramuscular stimulation, and needling and infiltration with preinjection blocks [66]. The effectiveness of needling depends on the needle-eliciting local twitch responses [67]. Presumably, the needle mechanically disrupts and terminates the dysfunctional activity of the motor endplates that are involved.

These techniques rely on an accurate identification of MTrPs by means of palpation. There is no definitive evidence that proves if one technique is superior to another in long-term outcome.

Conclusions

Myofascial pain is a disorder with a new and emerging level of understanding of its peripheral and central neurophysiologic basis. Cervical myofascial pain and headache can be rationally understood if one considers the neurophysiology of chronic myofascial pain and the neuroanatomic innervation of the cervical musculature.

Although there is much to learn about cervical myofascial pain and its relative contribution to the multifactorial causes of chronic headache, it remains a treatable and manageable type of headache. The most successful approach to long-term management is one that is interdisciplinary and addresses the peripheral component (the trigger point), the central component (altered pharmacology), and the behavioral (stress management) and biomechanical issues (ergonomic).

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- •• Of major importance

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