# Myofascial Pain: Current Concepts

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## REGIONAL MYOFASCIAL PAIN SYNDROME

Myofascial pain is a muscular syndrome that is regional in nature, i.e., localized to particular sets of functional muscle groups in a particular region of the body. Classically, myofascial pain is observationally defined by the presence of trigger points in the bellies of muscles. Trigger points are palpable, hypersensitive, taut bands that generate twitch and referred pain upon palpation. The referred pain must, at least in part, replicate the patient's pain complaint. Trigger points come in two varieties. "Active" trigger points are painful both at rest and with activity. "Latent" trigger points are painful only when palpated. "Taut bands" are palpable rope-like hardening of a group of tense muscle fibers that may possibly contain a myofascial trigger point. Characteristics of involved muscles include: (a) chronic stiffness, (b) palpable hard or spasmodic texture, (c) easy fatigability (muscular not systemic fatigue), and (d) an association with headache (chronic tension type headache or migraine). Myofascial pain syndrome is quite common with a higher prevalence in women than men (2:1), and its prevalence increases with age.[1–3]

Several pathophysiologic mechanisms have been proposed for the development of myofascial pain: (a) sustained muscle overload or repetitive strain cause fatigue, local ischemia, and chronic release of algesic peptides after injury; (b) an abnormality of the neuromuscular junction for recovery of calcium with resultant energy deficit causes a lower activation threshold at the neuromuscular junction; (c) dysfunctional biomechanical relationships among functional muscle groups result in deconditioning, atrophic changes, and functional loss; and (d) peripheral or central sensitization. These mechanisms attempt to define myofascial pain as a primary muscle pathologic process (mechanisms 1 through 3, and they may be interrelated) or merely a secondary muscular manifestation of another pathologic process occurring elsewhere (mechanism 4). The common etiologic thread among all the proposed mechanisms is that myofascial pain is directly and causally related to soft tissue injury or secondarily related to biomechanical adaptation to injury.

## FIBROMYALGIA

In contrast, fibromyalgia[4] is a systemic disease that is also observationally defined. Fibromyalgia is defined by the presence of tender points. Tender points are discreet areas of focal muscle tenderness that are elicited upon palpation and are localized over muscle, bone, tendon, and fat. The tender points are found at muscle-tendon junctions and characteristically do not reside in muscle bellies, as do trigger points. Palpation of tender points does not cause referred pain. Patients with fibromyalgia may also have trigger points. The American College of Rheumatologists has published diagnostic criteria for establishing the diagnosis that include chronic widespread pain for greater than 3 months with mechanical allodynia in at least 11 of 18 tender points in defined anatomic locations. Similar to myofascial pain, there is a higher prevalence in women than men (although the female to male predominance is 10:1 in. fibromyalgia). Comorbid conditions in fibromyalgia include sleep disturbances, neuroendocrine abnormalities, and immune system dysfunction, consistent with the systemic nature of the disease. Table 1 outlines T1
the distinctive characteristics of regional myofascial pain and fibromyalgia. The rest of the discussion will focus only on regional myofascial pain.

## EVALUATION OF BIOMECHANICAL ABNORMALITIES

When chronically present, trigger points do not occur in isolation but are rather the result of an interplay of etiologic factors. It is almost the lore of the anesthesiologist that "dry" needling[5] or injection[6] should have a long-term, i.e., curative, rather than palliative effect. Such a conceptualization may be too simplistic and may ignore the presence of biomechanical muscular relationships that may generate and perpetuate myofascial pain. The evaluation and treatment of myofascial pain syndrome should not merely denote trigger points and their location for injection but rather direct therapeutic maneuvers towards normalization of posture and biomechanics.[7]

Correct posture and normality of biomechanical relationships is most easily assessed by examination of an anatomic "plumb line" extending from the tragus of the ear, to the coracoid process of the shoulder, to the greater trochanteric of the femur, to the lateral malleolus of the ankle. Any deviation from this "plumb line" can potentially generate a myofascial pain syndrome as biomechanical relationships among functional muscle groups are altered.

When patients present with myofascial pain syndrome of the shoulders and the neck, measurement of the tragus-coracoid line should always be performed.

**Table 1.** Differences Between Myofascial Pain and Fibromyalgia

| | Myofascial pain | Fibromyalgia |
|---|---|---|
| Gender (female:male) | 2:1 | 10:1 |
| Defined by trigger points | (+) | (−) |
| Defined by tender points | (−) | (+) |
| Localization | Muscle belly | Muscle-tendon junction |
| Distribution | Regional | Widespread |
| Systemic fatigue | (−) | (+) |
| Neuroendocrine abnormalities | (−) | (+) |
| Sleep disturbance | (−) | (+) |

Notation should be made of the presence of the tragus forward of the coracoid or *vice versa* (and measured simply in fingerbreadths). Patients with "rounding of the shoulders" will present with deviation of the tragus-coracoid line with the coracoid forward, suggesting weakening of muscles responsible for scapular stabilization. The weakening of the scapular stabilizer muscles (rhomboids, lower trapezius, infraspinatus, and supraspinatus) with shortening of the pectoral muscles causes internal rotation of the shoulder girdles. On the other hand, patients can develop a forward head posture, sometimes referred to as "propulsion" or "forward head syndrome." Forward head syndrome is quite common in patients with myofascial pain of the shoulders and the neck. Cervical paraspinal muscles are shortened and cervical protraction and capital extension occurs. The upper trapezius muscles and levator scapulae muscles are elevated and shortened. The pectoral muscles are shortened and painful. Scapular stabilization issues and propulsion commonly occur together in the same patient. It is simplistic to think that trigger point injections themselves may have long-term therapeutic benefits unless underlying biomechanical relationships are addressed and normalized.

## POSSIBLE RELATION OF CERVICOTHORACIC MYOFASCIAL PAIN TO SPASMODIC TORTICOLLIS (OR CERVICAL DYSTONIA)

For the purposes of our discussion the terms "spasmodic torticollis" or "cervical dystonia" will represent the same disease process and be used interchangeably. Regardless of etiology, cervical dystonia is a syndrome of sustained involuntary neck muscle contractions quite often associated with painful muscles causing: (a) abnormal head or shoulder posture, (b) disturbed voluntary control of head movement, and (c) involuntary movements. Pain from continuous muscle contractions, cervical radiculopathy, cervical spondylosis, cervical facet arthropathy, and mechanical traction on musculoskeletal structures including ligaments and muscles is often associated with spasmodic torticollis. The prevalence of trigger points in spasmodic torticollis is unknown.

**Table 2.** Clinical Overlap Features

| Variable | Spasmodic torticollis | Myofascial pain |
|---|---|---|
| Abnormal Posture | (+) | (+) |
| Limitation in range of motion | (+) | (+) |
| Pain: "aching," "pulling," "burning" and "tightness" | (+) | (+) |
| Hypertrophy of muscle | (+) | (?) |
| Trigger points | ? | (+) |
| Tender points and taut bands | (+) | (+) |

Is it possible that certain patients with myofascial pain may have characteristics that ostensibly (and perhaps not pathophysiologically) "overlap" with spasmodic torticollis? As mentioned previously, myofascial pain of the head and neck can be associated with abnormal head or shoulder posture, meeting one of the potential criteria for diagnosis of cervical dystonia. The most common qualities of pain described in patients with cervical dystonia[8] include "aching" (48.3%), "pulling" (34.3%), and "burning " and "tightness" (9.4% each), reminiscent of the descriptors used in myofascial pain. The incidence of arm pain (potentially indicative of radiculopathy) in patients with spasmodic torticollis was 14.7% according to the study of Tarsy and First.[8] There is a high incidence of pain referred to the hand in patients with cervical myofascial syndrome. (Myofascial pain defies dermatomal anatomic boundaries found with radiculopathies and characteristically refers to all the digits of the hand.) According to the study by Galvez-Jiminez et al., localization of headache in cervical dystonia was: frontal region (43%), temporalis muscle area (68%), occiput (61%), cervical region (71%), and shoulder (18%).[9] The percentages and localization of headache are very reminiscent of the prevalence and location of headache in patients with cervical myofascial pain.

Thus, there is much circumstantial evidence to suggest an overlap of clinical characteristics between patients with spasmodic torticollis and certain forms of cervicothoracic myofascial pain. These findings are summarized in Table 2. If the injection of botulinum toxin is T2
effective in the treatment of cervical dystonia, might this suggest potential efficacy in patients with cervicothoracic myofascial pain and a postural abnormality?

## TREATMENT OPTIONS FOR MYOFASCIAL PAIN

Traditional therapies for the treatment of myofascial pain have included pharmacotherapy (nonsteroidal antiinflammatory drugs, steroids, tricyclic antidepressants, vasodilators, oral skeletal muscle relaxants), injection therapy ("dry" needling or trigger point injection of local anesthetic with and without corticosteroid), physical therapy, and behavioral modification. Such traditional therapies result in long-term benefit that is transient, variable, often incomplete, or nonexistent.[10,11]

## ANOTHER OPTION: INJECTION OF BOTULINUM TOXINS?

Botulinum neurotoxin is produced by the spore-forming bacterium *Clostridium botulinum*. There are seven distinct serotypes of the neurotoxin (A, B, C1, D, E, F, G). Serotype A is the most potent. Botulinum toxin has a molecular weight of approximately 150,000 Daltons and is a dichain polypeptide. The 100,000-Dalton heavy chain (allows internalization of the light chain) is linked by a disulfide bond to the 50,000-Dalton light chain. A number of SNARE proteins (synaptobrevin, SNAP-25, and Syntaxin) allow synaptic vesicles containing acetylcholine to bind to pre-synaptic membranes and fuse, releasing neurotransmitter into the synaptic cleft. Different toxin serotypes bind to distinct SNARE proteins and have unique cleavage sights. Cleavage of the SNARE protein prevents binding of vesicles containing acetylcholine with the pre-synaptic membrane, thereby blocking acetylcholine release at the neuromuscular junction. The light chain of botulinum toxin serotype A binds to SNAP-25, while the light chain of botulinum serotype B binds to synaptobrevin.[12]

Botulinum toxin serotype A has been shown to inhibit the release of a number of nociceptive neurotransmitter peptides via an identical SNAP-25 cleavage mechanism, preventing fusion of synaptic vesicles to the pre-synaptic membrane. These peripheral peptides include glutamate, substance P, and calcitonin gene-related peptide.[13,14] Moreover, botulinum toxin has been shown to have an antinociceptive effect by inhibition of peripheral and central sensitization.[15] Thus, any potential analgesic effects from botulinum toxin in the treatment of myofascial pain could result from chemodenervation (muscle relaxation) and antinociception.

There are currently two commercial preparations of botulinum toxin available in the United States for clinical use: botulinum toxin type A (BOTOX®; Allergan, Inc.) and botulinum toxin type B (Myobloc®; Solstice Pharmaceuticals). (A third preparation [another serotype A] is anticipating entry to the U.S. marketplace.) Botulinum toxin is currently approved in the United States for treating cervical dystonia, strabismus, laryngeal spasm associated with dystonia, and glabelar lines.

## PREVIOUS STUDIES

The literature is contradictory with respect to the efficacy of botulinum toxin in the treatment of myofascial pain. Early studies were powered with too small a number of patients to be deemed more than probes.[16,17] Alo et al.[18] and Lang[10] performed uncontrolled open-label studies, which did suggest efficacy. Freund and Schwartz performed a double-blind, randomized, placebo-controlled trial of direct trigger point injection in patients with chronic whiplash injuries, showing reduction in pain and improved cervical range of motion.[19] Wheeler et al. performed two double-blind, randomized, placebo-controlled trials of direct trigger point injection without positive results.[11,20] Ferrante et al.[7] using a double-blind, randomized, placebo-controlled design demonstrated that direct injection of botulinum toxin into trigger points was ineffective in the treatment of cervicothoracic myofascial pain.

## CONCLUSION

Could botulinum toxin be effectively used to treat myofascial pain? Previous studies suffered from a number of problems with methodology. Future studies must address the effects of dosing, volume, postural abnormalities, choice of muscles to inject, injection site, and injection technique.

Still, there appears to be accumulating evidence that patients with cervical myofascial pain, headache, and cervical dystonia may have common clinical features. The use of botulinum toxin in patients with cervical myofascial pain should be limited to those individuals with overlap features of spasmodic torticollis and must be coupled with aggressive rehabilitation to restore biomechanical abnormalities.

### REFERENCES


1. Davidoff RA. Trigger points and myofascial pain: toward understanding how they affect headaches. Cephalgia 1988;18:436–8
2. Borg-Stein J, Simons DG. Focused review: myofascial pain. Arch Phys Med Rehabil 2002;83(suppl 1):540–7
3. Simons DG. Muscular pain syndromes. In: Fricton Jr., Award EA, eds. Advances in pain research and therapy. Vol. 17. New York: Raven Press, 1990:1–41
4. Staud R. Fibromyalgia. In: Von Roenn JH, Paice JA, Preodor ME, eds. Current diagnosis and treatment of pain. New York: Lange, 2006:227–35
5. Chen JT, Chung KC, Hou CR, Kuan TS, Chen SM, Hong CZ. Inhibitory effect of dry needling on the spontaneous electrical activity recorded from myofascial trigger spots of rabbit skeletal muscle. Am J Phys Rehabil 2001;10:729–35
6. Hong CZ. Lidocaine injections versus dry needling to myofascial trigger point: the importance of the local twitch response. Am J Phys Rehabil 1994;73:256–63
7. Ferrante FM, Bearn L, Rothrock R, King L. Evidence against trigger point technique for the treatment of cervicothoracic myofascial pain with botulinum toxin type A. Anesthesiology 2005;103:377–83
8. Tarsy D, First ER. Painful cervical dystonia: clinical features and response to treatments with botulinum toxin. Mov Disord 1999;14:1043–5
9. Galvez-Jiminez N, Lampuri C, Patino-Picirrillo R, Hargreave MJA, Hanson MR. In: Fahn S, Hallett M, DeLong MR, eds. Dytsonia and headaches: clinical features and response to botulinum toxin. Advances in Neurology. Vol. 94. Dystonia 4. Philadelphia: Lippincott Williams & Wilkins, 2004:329–36
10. Lang AM. A pilot study of Botulinum toxin type A (Botox®), administered using a novel injection technique, for the treatment of myofascial pain. Am J Pain Manage 2000;10:108–12
11. Wheeler AH, Goolkasian P, Gretz SS. A randomized, double-blind, prospective pilot study of botulinum injection for refractory, unilateral, cervicothoracic, paraspinal myofascial pain. Spine 1998;23:1662–7
12. Montecucco C, Schiavo G. Structure and function of tetanus and botulinum neurotoxins. Q Rev Biophys 1995;28:423–72
13. Humm AM, Pabst C, Lauterburg T, Burgunder JM. Enkephalin and aFGF are differentially regulated in rat spinal motoneurons after chemodenervation with Botulinum toxin. Exp Neurol 2000;161:361–72

14. Puffer EB, Lomneth RB, Sarkar HK, Singh BR. Differential roles of developmentally distinct SNAP-25 isoforms in the neurotransmitter release process. Biochemistry 2001;40:9374–8
15. Cui M, Khanijou S, Rubino J, Aoki KR. Subcutaneous administration of botulinum toxin A reduces formalin-induced pain. Pain 2004;107:125–33
16. Cheshire WP, Abashian SW, Mann JD. Botulinum toxin in the treatment of myofascial pain syndrome. Pain 1994;59:65–9
17. Acquadro MA, Borodic GE. Treatment of myofascial pain with botulinum A toxin. Anesthesiology 1994;80:705–6
18. Alo KM, Yland MJ, Kramer DL, Charnov JH, Redko V. Botulinum toxin in the treatment of myofascial pain. Pain Clin 1997;10:107–16
19. Freund BJ, Schwartz M. Treatment of whiplash associated neck pain with botulinum toxin-A: a pilot study. J Rheumatol 2000;27:481–4
20. Wheeler AH, Goolkasian P, Gretz SS. Botulinum toxin A for the treatment of chronic neck pain. Pain 2001;94:255–60

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