

## Myofascial

### Trigger Point Pain

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Acute myospasm or "trismus" is well known to the dental professional, and myalgia (generalized muscle tenderness) is typically expected with temporomandibular joint disorders. However, less well appreciated is the fact that, once acute dental pathology has been ruled out, "pain of muscle origin is the most common cause of suffering about the neck, head and face."

After acute muscle problems, the most common, yet underappreciated muscle disorder that causes chronic or persistent pain in the head, neck, and facial region is myofascial pain (MFP). MFP is the most prevalent cause of unexplained orofacial pain and painful symptoms in temporomandibular disorders. In a retrospective analysis of data collected on 493 consecutive patients referred to the University of Minnesota TMJ and Facial Pain Clinic for "idiopathic or atypical pain", 54.2% had myofascial pain as the primary diagnosis. In another study conducted at this same university orofacial pain clinic, doctors prospectively evaluated 296 consecutive patients with chronic head and neck pain complaints. Myofascial pain was the primary diagnosis in 55.4% of these patients. In contrast, only 21% of these patients had an intrinsic temporomandibular joint disorder as the primary cause of pain. When present, these painful intrinsic joint disorders were almost exclusively due to an inflammation of the TMJ joint capsule or the retrodiscal tissues, not from clicks, pops or crepitus.

What is 'myofascial' pain? Myofascial pain, as defined by Travell and Simons, is a referred pain syndrome associated with focally tender trigger points (TrPs) in skeletal muscle that are characteristically distant from the site of pain. Many physicians and dentists alike insist on calling it myofascial pain and think of it as a myalgia of the facial and masticatory muscles. Others feel that it is a syndrome that involves some internal derangement of the TMJ plus associated local muscle soreness. However, MFP is a distinct entity and has been documented as a common cause of acute and chronic pain in all parts of the body. Despite the muscular origins of the pain, the chief complaint is NOT necessarily located in a muscle and is sometimes associated with autonomic symptoms, easily misleading the diagnostician.

MFP is not limited to dental issues. Almost 30% of patients presenting themselves with a complaint of pain in an internal medicine practice also had MFP as a primary diagnosis. Over 80% of patients enrolled in an inpatient chronic pain program also had MFP as the primary diagnosis. These reports emphasize that MFP is poorly recognized as a cause of chronic pain by almost all health care providers.

The intensity of myofascial pain should not be underestimated. Visual Analog Scale pain ratings for myofascial pain in a general medical practice were as severe or more severe than pain from other causes, another reason recognition and proper management is so critical.

MFP is clearly a very prevalent and potentially disabling disorder. Can you recognize it?

### CLINICAL RESEARCH

In MFP, the presenting pain complaint is almost always a referred symptom, typically, but not exclusively, with a deep, dull aching quality. Examples of the referred pain sites in the head and neck include the teeth, sinuses, cheeks, forehead, temple, ears and TMJ. It is important to understand that the referred pain site is NOT necessarily over another muscle.

Overlapping pain referral patterns from several different trigger points will mimic certain primary headache disorders as well. Careful evaluation of the site of the pain does not yield any findings of pathologic change. In fact, any undiagnosed deep, dull, aching pain may be myofascial in origin or have a contributing myofascial component.

Associated symptoms, due to physiologic sensory, motor, and autonomic effects seen with prolonged pain, are common and may confuse the clinical picture. Associated sensory complaints may include tenderness in the referred pain site, such as tenderness to palpation of the lateral poles of the TMJ without concomitant pain with joint movement, scalp pain on brushing the hair, or abnormal sensitivity of the teeth or gums.

Motor effects include increased EMG activity in the pain reference zone when the pain reference is into another muscle. Although patients rarely complain about this specifically, this referred motor activity frequently results in the development of "satellite" myofascial trigger points in the secondary muscle creating additional referred pain and further confusing the clinical picture.

Autonomic changes such as pallor, sweating, lacrimation, runny nose, ptosis, increased salivation, nausea and vomiting, as well as tinnitus have also been reported.

Aggravating factors include stress, cold weather, immobility, and overuse of the involved muscles. Alleviating factors include hot baths, rest, warm weather, and massage.

### EXAMINATION

With MFP the patient's pain complaint is typically a referred symptom. Therefore, it is usually distant from the muscle containing the guilty TrP. Multiple TrPs can produce overlapping areas of referred pain. Familiarity with the typical referral patterns allows the clinician to use the location of the pain in reverse to identify possible etiologic TrPs (Figs. 1-9). Systematic fingertip examination of suspected muscles looking for taut bands and focal tenderness, is required.

Effective TrP palpation is a skill that must be learned and practiced. Most muscles lend themselves to flat palpation using the tip of the index finger. Some muscles can be palpated between the index finger and thumb for pincer-type palpation. In the head and neck region, the masseter, sternocleidomastoid and upper trapezius muscles lend themselves to pincer palpation. Once a suspected TrP is found, 2 to 4 kg/cm<sup>2</sup> of pressure should be applied for 6 to 10 seconds to allow the referred pain pattern, if any, to develop. The examination may replicate the patient's pain so precisely that there is no doubt about the diagnosis. If uncertainty exists, specific TrP therapies, such as "spray and stretch" or TrP injections, described below, may be used diagnostically.

All head and neck muscles should be routinely examined in patients with a persistent pain complaint, keeping in mind that cervical muscle myofascial TrPs (e.g. upper trapezius or sternocleidomastoid) are almost always the "key" trigger points, their secondary referred motor effects feeding and perpetuating "satellite" trigger points in the masticatory muscles (e.g. the masseter and temporalis).

The location of TrPs and their associated referred pain patterns are predictable and reproducible from patient to patient. A meticulous discussion of MFP, as well as a complete compendium of the pain referral patterns for most muscles of the body, has been brilliantly detailed by Travell and Simons.

#### CAUSES

Myofascial TrPs may be "primary" or "secondary".

When primary, there is usually a history that includes a clearly identifiable musculoskeletal injury or "macrotrauma" such as a fall, sports injury, motor vehicle accident or even prolonged jaw opening at the dental office. Alternatively, myofascial TrPs also develop with chronic muscle overuse or "microtrauma" due more insidious factors such as poor posture and body mechanics, or repetitive motion activities.

Secondary myofascial TrPs develop in response to any prolonged painful process or disease such as inflammatory disorders of the TMJ, chronic ear infections, persistent toothaches, migraine, cancer or any other chronic painful condition. The primary noxious stimulus (for example, a third molar infection) causes a protective spinal reflex response (trismus). Initially the increased motor activity is dependent on the primary pain source, but if the primary pain problem persists long enough, the motor activity becomes an independent, self-perpetuating primary pain source in the muscle (a trigger point) that persists even after the primary pain source (the infected third molar) is removed. The pain from this secondary myofascial trigger point typically refers back to the same site as the initial primary pain causing the clinician to believe that there is persistent pathology, often resulting in additional unnecessary treatments.

Secondary myofascial pain needs to be identified and treated to reduce unnecessary treatments and also to reduce pain and improve response to other therapies when the initiating source is also a chronic pain such as migraine or post herpetic neuralgia.

Trigger points may be "active" or "latent". They are considered active when the referred pain pattern and associated symptoms are clinically present and latent when they are not clinically present but can be elicited with palpation. Trigger points will vacillate between active and latent states depending on the amount of psychological stress the individual is under and the amount of muscle overload being placed on the affected muscle.

#### PATHOPHYSIOLOGY

With some training, myofascial TrPs are relatively easy to palpate. Despite the comparative ease of clinical identification of TrPs, questions still exist about their structure and exact pathophysiology, but progress has been made. In 1993, careful monopolar needle EMG evaluation revealed spontaneous electrical activity or "SEA" at the TrP sites, while EMG evaluation of the muscle surrounding the TrP was normal. This objective laboratory finding opened the door to establishing myofascial pain as a clinical entity with an electromyographical marker. SEA is significantly higher in subjects with clinical pain due to active TrPs than in subjects without clinical pain who have latent or no TrPs. SEA can be recorded only if the needle is precisely placed in the nidus of the TrP; movement of the needle tip as little as 1 mm is enough to lose the signal.

In 2005, Shah and his colleagues, using a unique in vivo microanalytical technique, demonstrated that concentrations of protons, bradykinin, calcitonin gene-related peptide, substance P, tumor necrosis factor-alpha, interleukin-1beta, serotonin, and norepinephrine are significantly higher in active TrP sites than in latent TrP sites or in normal muscle. In addition, pH was significantly lower in the active TrP sites than the other two groups.

More recently, they also demonstrated that TrPs can be located and identified with ultrasound and vibration sonoelastography.

Psychological stress, which causes increased sympathetic output, has been shown to increase the SEA recorded from TrPs, whereas the EMG activity of adjacent non-TrP sites remains unchanged. These data parallel the clinical observation that emotional stress activates or aggravates pain from TrPs.

Based on the psychophysical evidence available to date and the observation that myofascial TrPs are frequently located in and around the motor end-plate area of muscles, Simons hypothesized that the myofascial TrP represents an area of isolated sustained muscle contraction due to excess acetylcholine release. This would result in uncontrolled metabolism, and localized ischemia that is initiated by acute eccentric muscle injury or strain. This theory does provide a credible explanation for the palpable nodules and taut muscle bands associated with TrPs. The TrP nodule is described as a group of "contraction knots" in which a number of individual muscle fibers are maximally contracted at the end-plate zone, making them shorter and wider at that point than the non-contracted neighboring fibers. If enough fibers are so activated, a palpable nodule could result. As for the taut band, both ends of these affected muscle fibers would be maximally stretched out and "taut," producing the palpable taut band.

Clinically, data exist documenting that TrPs are truly focally tender areas in muscle; pain with palpation is not due to generalized muscle tenderness. Indeed, tenderness to palpation over non-TrP sites in subjects with MFP does not differ significantly from normals. Also, muscle bands containing TrPs will display a local twitch response, a transient contraction of the muscle band with deep "snapping" digital palpation. This response, best appreciated in more superficial muscle fibers, has also been substantiated experimentally. A rabbit model of the twitch response has documented that, at least in rabbits, the twitch response is a spinally mediated reflex.

The mechanism of referred pain from myofascial TrPs is also under speculation. According to Mense and Vecchiet et al., the convergence-projection and convergence-facilitation models of referred pain do not directly apply to muscle pain because there is little convergence of neurons from deep tissues in the dorsal horn. These authors proposed that convergent connections from other spinal cord segments are "unmasked" or opened by nociceptive input from skeletal muscle and that referral to other myotomes is owing to the release and spread of substance P to adjacent spinal segments. Simons expanded on this theory to specifically explain the referred pain from TrPs.

The following cases are typical scenarios involving myofascial pain that may provide a diagnostic challenge for the general dentist.

#### CASE HISTORY #1

A 47-year-old male presented with a complaint of acute left TMJ pain. He had a history of chronic painless bilateral TMJ osteoarthritis. On examination jaw opening was 41mm with tenderness to palpation over the left TMJ. He was instructed in palliative care including rest, soft diet, gentle hinge axis jaw stretching and seven days of non-steroidal anti-inflammatory medications. He returned a week later saying that the severe symptoms had improved, but he still had "persistent aching of the left jaw" along with a ringing in his left ear, especially with clenching. Active range of motion of the jaw had increased from 41 to 47mm and the left TMJ was now non-tender to palpation. Why does he still have pain? Is it due to the osteoarthritis?

The less astute clinician may still direct his or her energies toward treating the TMJs, especially since there was documented osteoarthritis bilaterally, worse on the left. However, the source of the pain was now from myofascial TrPs and not the joint. Palpation of the masseter muscle, particularly the deep fibers, reproduced the patient's current symptoms, including the ringing in his left ear. Trigger points in this part of the masseter muscle have also been reported to cause unilateral tinnitus and accounted for the high-pitched sound the patient complained of with clenching. Treatment must be directed at rehabilitating the masseter muscle and not at the asymptomatic joint.

#### CASE HISTORY #2

A 40-year-old woman sustained a luxation injury to tooth #9 when her face hit the steering wheel during a motor vehicle accident. The tooth was repositioned and stabilized by her regular dentist, but the pain did not subside. After six months she had a root canal treatment. The pain persisted. A subsequent apicoectomy was equally ineffective in relieving the pain. The tooth was extracted. A deep aching pain persisted. As a result, her oral surgeon prescribed 800mg ibuprofen and Vicodin. She went to see a neurologist who placed her on gabapentin with no improvement. What could possibly be causing her persistent pain?

This is an example of key trigger points in the sternocleidomastoid and upper trapezius muscles from the initial whiplash injury, along with prolonged dental pain, inducing secondary myofascial trigger points in the temporalis muscle. Treatment must be primarily directed at the TrPs in the cervical muscles to reduce TrP activity in the temporalis muscle. Unfortunately,

this patient lost her upper anterior incisor because the dentists she saw were unaware of myofascial pain as a potential cause of this type of persistent pain, and, despite any clear pathology, chose instead to focus on a presumed dental origin for the pain.

#### TREATMENT

Recommended treatment of MFP involves most importantly identification and control of causal and perpetuating factors, patient education, and specific home stretching exercises. Therapeutic techniques such as "spray and stretch," voluntary contract-release, TrP pressure release, and TrP injections are useful adjunctive techniques that usually facilitate the patient's recovery once most perpetuating factors are controlled.

Perpetuating factors most commonly include mechanical factors that place an increased load on the muscles. Teaching patients good posture and body mechanics will go a long way in reducing referred pain from myofascial TrPs, especially in the head and neck region. Cervical muscle TrPs almost always perpetuate TrPs in the masticatory muscles and should always be treated first, regardless of the pain complaint.

Psychological factors, such as stress that has been shown to cause TrP activation or depression that lowers pain thresholds, will contribute to and perpetuate MFP. Sleep disturbance and inactivity are also common perpetuating factors. Simple stress management and relaxation skills are invaluable in controlling the associated increased TrP irritability if this is a problem. Mild depression and sleep disturbance can be treated with low doses of tricyclic antidepressant drugs and a structured exercise/activation program.

Other perpetuating factors include metabolic, endocrine, or nutritional inadequacies that affect muscle metabolism. Patients should be screened for general good health and referred to their physician for management of any systemic abnormalities. In secondary MFP, the primary concomitant painful disorder, such as a true TMJ capsulitis, pulpitis, a primary headache disorder or chronic deafferentation pain, must also be treated or managed.

Spray and stretch is a highly successful technique for the treatment of myofascial TrPs that uses a vapocoolant spray (Gebbauer, Co.; Cleveland, OH) to facilitate muscle stretching. Muscle stretching has been shown to reduce the intensity of referred pain and TrP sensitivity in patients with myofascial pain. Vapocoolant is applied slowly in a systematic pattern over the muscle being stretched and into the pain reference zone. This technique and alternatives to vapocoolant are described in detail in Simons et al.'s text.

Needling of TrPs with or without injection of solution has also been shown to be helpful in reducing TrP activity to allow stretching. Dry needling or injection of "key" myofascial trigger points has been shown to reduce activity and tenderness in related satellite TrPs. Clinically it is essential to carefully palpate and find the exact location of the trigger point and elicit a twitch response with needle penetration. A twitch response means that the trigger point has been impaled and this means that the clinical result will be profound. Without a twitch response, a reproduction of the referred pain pattern is the second most important sign that the trigger point has been found. Just injecting randomly and flooding the area with local anesthetic is NOT effective.

Although dry needling is effective, use of a local anesthetic reduces post-injection soreness. If a local anesthetic is to be injected, 0.5% procaine or 0.5% lidocaine is recommended. Longer-acting amide local anesthetics or local anesthetics containing epinephrine cause muscle damage. Injection must always be followed by stretch. Trigger point injection in the absence of a home program that addresses relevant perpetuating factors will provide only temporary relief. Randomized controlled studies looking at the efficacy of botulinum toxin in comparison to dry needling, saline or other placebo solutions in trigger point injections have shown that overall Botox is no more effective than dry needling or other solutions.

#### SUMMARY

Myofascial trigger point pain is an extremely prevalent cause of persistent pain disorders in all parts of the body, not just the head, neck, and face. While not limited to dental practice, within dental practice it is an extremely common cause of otherwise difficult to diagnose pain. Features include typically, but not exclusively, deep aching pain in any structure, referred from focally tender points in taut bands of skeletal muscle (the trigger points). Diagnosis depends on accurate palpation with 2-4 kg/cm<sup>2</sup> of pressure for 6-10 seconds over the suspected trigger point to allow the referred pain pattern to develop. In the head and neck region, cervical muscle trigger points (key trigger points) often incite and perpetuate trigger points (satellite trigger points) and referred pain from masticatory muscles. Management requires identification and control of as many perpetuating factors as possible (posture, body mechanics, psychological stress or depression, poor sleep or nutrition). Trigger point therapies such as spray and stretch or trigger point injections are best used as adjunctive therapy. **OH**

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