Myofascial Pain Syndrome in the Craniomandibular Region

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Craniomandibular disorders (CMD) are characterized by a combination of symptoms that may include pain, tenderness and dysfunction of the temporomandibular joint, the mouth and the occlusal contacts, the cervical spine, and the muscles of mastication. Patients may present with local dentoalveolar pain; muscle pain; head, facial and neck pain; sounds during condylar movements; deviations and limitations of mandibular movements; altered occlusal relations; parafunctions and poor oral habits; and functional limitations of mastication. Craniofacial pain conditions have special emotional and psychological meaning. The face, the mouth, speech, and other oral functions are essential for nearly all human interactions; craniofacial pain conditions interfere with such functions and with the ability to communicate.

Approximately 10% of the general population experience craniomandibular pain (1). The prevalence is estimated to range from 0% to 10% for males and from 2% to 18% for females. The prevalence in children and adolescents is estimated to range between 2% to 6% (2). Pain complaints range from acute and transient conditions such as toothaches to chronic ailments, such as trigeminal neuralgia and temporomandibular disorders. While in the majority of pain patients, pain decreases over time with or without treatment, for a small percentage the pain complaint persists. Similarly to other musculoskeletal pain problems, between 5% and 15% of all CMD patients become chronic pain patients (3-5). Patients with persistent craniomandibular pain without objective clinical or radiographic findings are especially challenging not only to the general dentist, but also to orthodontists, oral surgeons, TMJ specialists, and other clinicians. Some patients may be considered good candidates for splint therapy. Others are referred to oral surgeons when common dental procedures fail to offer relief. Some frustrated pain patients may even request surgical treatment in an effort to eliminate chronic craniomandibular pain (6).

It is generally accepted that CMD have a multifactorial etiology (7). Although many diseases, such as dental disease, infections and tumors, can be associated with pain, most chronic pain problems are thought to be musculoskeletal in nature. Different perspectives of the primary source of pain have passed the review. Some researchers and clinicians have emphasized joint dysfunction (8, 9); others have focused more on muscular problems (10-12). The relation between occlusion and CMD has been the subject of several investigations (13-16). Multiple studies have considered the impact of stress on CMD and in particular the impact of stress on pain and tenderness of the masticatory muscles (17-19). With the advancement of the pain sciences, more and more emphasis is placed on peripheral and central nervous system sensitization (20-23).

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Functional relations with spine dysfunction need to be considered in the management of patients with craniofacial pain syndromes (24, 25). Rocabado has developed a pragmatic approach incorporating the intricate relationships between the cervical spine and mandible and temporomandibular function. He has demonstrated that centric position can only be achieved when there is a balance between the position and movement patterns of the subcranial region, the mid and lower cervical spine, the hyoid, and the mandible (26, 27). A detailed biomechanical assessment is important. Rocabado’s “pain map” is an excellent tool to determine which joint structures cause or may contribute to the pain complaint (see Rocabado’s chapter in this book for a more detailed description of his approach).

In addition to a biomechanical joint assessment, clinicians need to include a detailed evaluation of the muscles in the cervical and craniofacial region. Numerous studies of craniofacial muscle pain and dysfunction have incorporated the “research diagnostic criteria for temporomandibular disorders” by Dworkin and LeResche, which include a section on muscle dysfunction (28). The research criteria were developed to classify and quantify both the physical and psychosocial components of temporomandibular dysfunction. Although the criteria are widely used in dental research, they are remarkable simplistic where it involves muscle dysfunction. The section “Axis I, Group I, Muscle Disorders” of the criteria includes only two options to describe muscle dysfunction: tender muscles with or without limited mouth opening.

The inclusion of the word “diagnostic” in the title of Dworkin and LeResche’s classification criteria may be interpreted that the criteria can be used in clinical practice. However, research criteria do not necessarily provide a mechanism for clinical diagnosis. For example, MTrPs, myositis, and other less common conditions affecting muscles, such as myoadenylate deaminase deficiency or fascioliensis cannot be diagnosed using the research criteria. Therefore, Dworkin and LeResche’s criteria do not provide any starting points to treat patients with craniofacial muscle dysfunction in clinical settings, even though they have been validated for research purposes (29-32).

**Myofascial Pain Syndrome**

In the dental literature, muscle pain with or without limits in mouth opening is commonly referred to as “myofascial pain dysfunction syndrome,” a somewhat unfortunate term, as it is often confused and used interchangeably with the more practical and better defined concept of “myofascial pain syndrome” (MPS) described by Simons, Travell and Simons (33). MPS is the main subject of this chapter and can be described as the sensory, motor, and autonomic symptoms caused by myofascial trigger points. A myofascial trigger point (MTrP) is clinically defined as a hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. The spot is painful on compression and can give rise to characteristic referred pain, referred tenderness, motor dysfunction, and autonomic phenomena (33). Although there are many peer-reviewed studies and reports in the international medical literature supporting the importance of MTrPs in clinical practice, many of these reports are not included in major medical library indices. Fortunately, several prominent researchers are now investigating MTrPs and the results of many of these studies support the theoretical foundations and clinical applications. Nevertheless, there is a substantial lack of basic scientific studies; in general, MTrPs are underexplored by research investigators (34).

A better understanding and working knowledge of MTrPs and MPS will provide dentists, orthodontists, oral surgeons, and other clinicians with an effective approach to relieve human suffering, and contribute significantly to their patients’ quality of life. If MTrPs are not considered in the differential diagnosis, a common cause of patients’ pain complaints will be overlooked (34, 35). MPS should be considered with any pain syndrome in the head, neck, face, and TMJ area (12, 33, 36). Dentists need to be aware that apparent dental or TMJ pain does not necessarily have a dental or joint origin (37).

Myofascial pain tends to be dull, poorly localized, and deep, in contrast to the precise location of dental pain and cutaneous pain. Muscles can refer pain to other deep somatic structures, such as fascia, joints, viscera, and other muscles (38). Clinically, referred pain is confusing to many clinicians, as frequently, patients complain more of pain in the referred pain zone and not necessarily of pain in the immediate area of a MTrP. Signs and symptoms suggestive of non-odontogenic pain include an inadequate local dental cause for the pain; a recurrence of pain in spite of reasonable dental therapy of the tooth or TMJ; poor lasting pain relief after local anesthetic blocking; positive findings with Rocabado’s pain map; postural abnormalities such as forward head posture; and other pain problems, such as chronic and recurrent headaches and widespread chronic pain conditions.

Patients with MPS usually have a history of acute or chronic muscle overload. In dental practice, MPS is commonly seen in patients with a history of
bruxism or clenching (12, 39-41). A common iatrogenic cause of MPS occurs when patients are required to keep their mouths open for prolonged periods of time during dental procedures (42). Patients with MPS in the head-neck region often are unable to relax their muscles in between contractions, i.e., the masseter, sternocleidomastoid and trapezius muscles. The muscles are in continuous contracture, which can result in muscle ischemia, fatigue and pain (23, 43, 44).

**Historical Overview of Myofascial Pain Syndrome**

MTrPs are the principal characteristic of MPS. During the past nearly 200 years, numerous authors have described MTrPs in the English, German, Dutch, and French medical literature, illustrating that musculoskeletal pain due to MTrPs is very common (45, 46). Already in 1816, British physician Balfour described MTrPs as “nodular tumours and thickenings which were painful to the touch, and from which pains shot to neighbouring parts.” Balfour speculated that the nodules were due to inflammation of the fibrous connective tissue in muscle (47). Historically, multiple terms have been used to describe muscle pain syndromes, many of which would have been diagnosed as MPS using the current definition, including fibrositis, myofascitis, muscular rheumatism, rheumatic myositis, muscle hardening, myogelosis, myalgia, myofascial pain, and even fibromyalgia (45).

In 1938, British rheumatologist Kellgren published a seminal paper describing specific referred pain patterns of many muscles and spinal ligaments following injections of hypertonic saline. In 1952, Travell wrote the first of many articles introducing the myofascial genesis of pain illustrated by specific referred pain patterns of over 30 muscles (48). Travell (1901 - 1997) has been referred to as the pioneer in the treatment of musculoskeletal pain through the recognition of MTrPs. She coined the term “myofascial pain syndrome” to describe pain as a result of trigger points in muscle, tendon, skin, fascia, and ligaments (the term “trigger point” was introduced by Steindler in 1940 (49)). Several of Travell’s subsequent papers included referred pain patterns relevant for head, neck, facial, and craniofacial pain (50-52). Travell and Simons authored the classic two-volume text “Myofascial Pain and Dysfunction: The Trigger Point Manual.” The first volume was published in 1983 for the upper half of the body followed by the second volume in 1992 for the lower half (53, 54). The second edition of the first volume, authored by Simons, Travell and Simons, was released in 1999 and includes contributions by experts in the growing field of myofascial pain and dysfunction (33).

The second edition summarizes significant progress in the understanding of the pathophysiological basis of the clinical presentations associated with MTrPs. The manuals have been translated into six different languages, including Spanish. Several other MTrP manuals have been published in Switzerland, the United States and the UK (46, 55, 56).

Several assessment and treatment approaches have emerged independently of each other both in Europe and in the United States, including myofascial trigger point therapy (USA), neuromuscular technique or NMT (UK), neuromuscular therapy, also abbreviated as NMT (USA), and manual trigger point therapy (Switzerland). It is not a coincidence that these approaches share many similarities and have common goals and objectives (57). Recent insights in the nature, etiology and neuromusiology of MTrPs and their associated symptoms have propelled the interest in the diagnosis and treatment of persons with MPS worldwide (58).

**Diagnostic Criteria**

A diagnosis of MPS can be made only by palpation. There are no laboratory tests, imaging studies, or standardized diagnostic criteria to make an initial diagnosis of MPS. While this may suggest, that a diagnosis of MPS cannot be established, clinical evidence combined with multiple scientific studies and case reports support the practice of evaluating patients for the presence of MTrPs. In a survey of physician members of the American Pain Society, 85% of 493 medical pain specialists, representing fourteen medical specialties, agreed that MPS is a distinct syndrome (59). MPS may actually be the most overlooked diagnosis in chronic pain patients (60). The diagnosis depends on the clinician’s skills, training and experience in taking a patient’s history, performing a comprehensive examination, and assessing patients for MTrPs. Clinicians should not assume knowing how to identify MTrPs without specific training (61, 62). Unfortunately, few medical, dental and allied health school curricula include education and training in the assessment and treatment of MTrPs. Most clinicians trained in the diagnosis and treatment of MTrPs have been trained through post-graduate continuing education programs, such as the Interessengemeinschaft für Myofaszielle Triggerpunkttherapie (Society for Myofascial Trigger Point Therapy) in Switzerland (www.imtt.ch) and the Janet G. Travell, MD Seminar Series™ in the US (www.painpoints.com). The International Myofascial Society has established a multidisciplinary international committee to design a study model for validation of diagnostic criteria. The
committee aims to establish reliable methods for diagnosis of MPS, determine the interrater reliability of MTrP examination, and determine the sensitivity and specificity with which classification criteria can distinguish patients with MPS from healthy control subjects (63).

Simons, Travell and Simons defined a MTrP as a localized spot of tenderness in a nodule of a palpable taut band of contractured muscle fibers (33). Taut bands are examined by gently palpating a muscle perpendicular to the direction of the muscle fibers. Taut bands need to be differentiated from more generalized muscle spasms, that can be defined as electromyographic activity as a result of increased neuromuscular tone of the entire muscle (64, 65). A taut band feels like a rope or string of contractured fibers, that may extend from one end of the muscle to the other end. There are two basic palpation techniques for the proper identification of MTrPs. A flat palpation technique is used for example with palpation of the temporalis and masseter muscles (Figure 1). A pincher palpation technique is used for example with palpation of the sternocleidomastoid and the upper trapezius muscles (Figure 2).

Figure 1 – a. Flat palpation of the temporalis muscle

Figure 1 – b Flat palpation of the masseter muscle

Figure 1 – c. Intra-oral palpation of the masseter muscle

Figure 2 – a. Pincher palpation of the sternocleidomastoid muscle

Figure 2 – b. Pincher palpation of the trapezius muscle

Two recent studies have established excellent overall interrater reliability of the clinical examination used to establish the presence of MTrPs (62, 66). To make a diagnosis of MPS, the minimum essential features that need to be present are a taut band, an exquisitely tender spot or nodule in the taut band, and
the patient’s recognition of the pain complaint with pressure on the tender nodule (62). Simons, Travell and Simons add a painful limit to stretch range of motion as the fourth essential criterion (33). Taut bands and MTrPs are found in asymptomatic individuals and are only considered clinically relevant when the patient recognizes the elicited pain or when the functional limitations imposed by the taut band contribute to mechanical dysfunction secondary to muscle shortening (61, 67). The presence of a local twitch response, referred pain, and the electromyographic demonstration of endplate noise increase the certainty and specificity of the diagnosis.

Local Twitch Response

A local twitch response (LTR) is elicited by either strong digital palpation or needling/injection of a taut band or MTrP, that results in an involuntary brief burst of motor action potentials propagated only by fibers of that taut band or by fibers of a taut band from another muscle (68–70). A LTR is a spinal cord reflex, mediated through the spinal cord without supraspinal influences (69). During the physical examination, a LTR confirms the presence of a MTrP. Although the LTR can occur during physical examination of MTrPs, eliciting a LTR is not essential for making a diagnosis of MPS. Eliciting a LTR manually or with needling can be difficult and rather painful for patients, which suggests that a LTR perhaps originates from stimulation of sensitized nociceptors in the MTrP region rather than exclusively from mechanical stimulation (38). Eliciting a LTR during needling procedures is critical for the successful treatment of a MTrP (61, 71). Close proximity to the MTrP is essential (68). Gerwin and Duranleau were able to objectively visualize the LTR using diagnostic sonography, following stimulation of a MTrP by insertion of a hypodermic needle (72). High resolution sonography was however, not sensitive enough to visualize the actual MTrP (73).

Referred Pain

Since Kellgren’s early studies on referred pain from muscle, multiple clinical reports have been published on referred pain from MTrPs (74). Referred pain is challenging for clinicians, as the patient’s perception of localization of pain can be very different from the original source of pain (75). To better appreciate the clinical implications of referred pain from MTrPs, a more extended review is included in this chapter. Although Asina and colleagues did not specifically mention MTrPs, they described many of the symptoms commonly associated with MTrPs, such as increased myofascial tenderness, hardness, referred pain causing headaches, and central sensitization as a result of persistent nociceptive input from pericranial and extracranial muscles (76). Many case reports describe that MTrPs can cause or contribute to persistent headaches, facial pain, and temporomandibular pain (50, 77–80). Carlson demonstrated the clinical significance of referred pain by eliminating pain of the masseter muscle by treating MTrPs in the trapezius muscle (81).

Many scientific studies and review articles have focused on muscle referred pain (12, 23, 44, 82–93). Referred pain is not specific to MTrPs, but it is more common and much easier to elicit over MTrPs than over other structures (85). Hong found that all subjects reported referred pain with pressure directly over active MTrPs, but less than 50% of subjects reported referred pain with pressure over latent MTrPs (85). Compared to active MTrPs, latent MTrPs do not produce spontaneous pain; they require more pressure to elicit localized and referred pain (33). Normal muscle tissue and other body tissues, including skin, zygapophyseal joints, and internal organs may also refer pain to distant regions with prolonged and sufficient mechanical pressure (67, 89, 94–99). In the orofacial region, it is relevant that the teeth can also refer pain to other areas, including the TMJ and the sinuses (75).

By mechanically stimulating an active MTrP, patients often report the development of referred pain in fairly consistent patterns, either immediately or after a brief 10-15 second delay. Mechanical stimulation may consist of manual pressure, needling of the MTrP, movement of the involved body region, or even prolonged postural strains, such as forward head posture or prolonged mouth opening. Usually, pain in reference zones is described as deep tissue pain of a dull and aching nature. However, some patients report burning or tingling sensations or other paresthesia (89, 100). In that sense, the term “referred sensation” reflects the clinical condition sometimes more accurately than the established term “referred pain.” Common referred pain patterns for nearly all skeletal muscles are documented by Travell and Simons (33, 53, 54). Dejung and colleagues have modified several referred pain patterns in their recent publication on MTrPs (55).
Neurobiology of Referred Pain

In recent years, much new information has become available about the neurobiology of muscle referred pain (23, 38). Although the majority of data has been obtained from animal experiments, it is probably applicable to patients. Mense and colleagues have demonstrated that skeletal muscles have different types of nociceptors (101-103). Under normal conditions, skeletal muscle nociceptors require high intensities of noceptive stimulation involving high-threshold mechanosensitive neurons, that do not respond to moderate local pressure, contractions or muscle stretches (65). Muscle nociceptors are activated by endogenous pain-producing substances such as bradykinin or serotonin. At least one of the muscle nociceptors is specifically sensitive to ischemic conditions (104).

Local ischemia is associated with MTrPs. Looking at MTrPs from a biomechanical perspective, it is conceivable that at the cytoskeletal level, myosin filaments literally get stuck in the Z band of the sarcomere. During sarcomere contractions, titin filaments are folded into a gel-like structure at the Z band. In MTrPs, gel-like titin appears to prevent the myosin filaments from detaching. The myosin filaments may actually damage the regular motor assembly and prevent the sarcomere from restoring its resting length (105). The contracted sarcomeres are thought to reduce the local circulation by compressing the capillary blood supply and thus cause a significant lack of local oxygen. One study has shown that the oxygen saturation in the center of a MTrP is less than 5% of normal (106). Histological studies also suggest that MTrPs are ischemic: ragged-red and moth-eaten fibers have been identified in myoglobin and MTrPs (107-110). Moth-eaten fibers indicate a change in the distribution of mitochondria or the sarcotubular system, while ragged-red fibers reflect an accumulation of mitochondria (111). Both are usually associated with muscle ischemia, denervation, or strenuous exercise (112). Simons suggested that myoglobin and MTrPs may represent the same phenomenon (113).

Local hypoxia is associated with the release of endogenous inflammatory substances, such as prostaglandin, bradykinin, serotonin, capsaicin, histamine, and interleukins. Activation and sensitization of muscle nociceptors leads to inflammatory hyperalgesia, an activation of high threshold nociceptors associated with C fibers, and an increased production of bradykinin. Furthermore, bradykinin stimulates the release of tumor necrosis factor (TNF-α), which in turn activates the production of the interleukins IL-1β, IL-6 and IL-8. Especially IL-8 can cause hyperalgesia that is independent from prostaglandin mechanisms. Via a positive feedback loop, IL-1 beta can also induce the further release of bradykinin (114). Bradykinin triggers the release of calcitonin gene related peptide (CGRP), which reduces the effectiveness of acetylcholinesterase (115-117). The combination of endogenous substances reinforces the observed hyperalgesia. Injections of bradykinin and serotonin into the temporal muscle of healthy volunteers caused more severe pain than either bradykinin or serotonin by itself (118). Following muscle trauma multiple substances are released simultaneously, which appears to occur in MTrPs as well. In their studies of the neurochemical milieu of MTrPs, Shah and colleagues at the U.S. National Institute of Health have confirmed increased concentrations of bradykinin, substance P, TNF-α, interleukins, CGRP, and norepinephrine in active MTrPs when compared with latent MTrPs and healthy control subjects (119). In addition, the pH of the MTrP site was significantly lower in the active MTrP group.

The endogenous substances bind to specific receptor molecules in the membrane of nociceptive nerve endings, including the so-called purinergic and vanilloid (VR-1) receptors. Purinergic receptors bind adenosine triphosphate and stimulate nociceptors accordingly. VR-1 receptors are especially sensitive under conditions of lowered tissue pH and muscle ischemia. Mense suggested that pain during tooth clenching, bruxism, and tension-type headaches is mediated by the VR-1 receptor molecule (44). Bradykinin, serotonin and prostaglandin interact at many levels at the vanilloid receptors (120).

Referred pain is generated by central mechanisms dependent on peripheral input from the referred pain area. MTrPs are a source of ongoing peripheral nociceptive input. Long-term nociceptive input into the spinal cord or brain stem causes changes in function and connectivity of sensory dorsal horn neurons or neurons in the trigeminal nucleus caudalis respectively (21, 44, 121, 122). The ongoing afferent barrage into the dorsal horn and the trigeminal nucleus results in central sensitization, the unmasking of sleeping receptors in the dorsal horn, referred pain, and hyperexcitability (121, 123-125). There is considerable evidence that both neurokinin and N-methyl-D-aspartate (NMDA) receptors are involved in triggering hyperalgesia and the MTrP-induced hyperexcitability of dorsal horn cells and brainstem nociceptive neurons (21, 44, 126).
In MTrPs, low-threshold mechanosensitive neurons may also get activated (127). Strong or long-lasting nociceptive input from damaged muscle tissue or from MTrPs results in spatial summation in the dorsal horn or brain stem and the appearance of new receptive fields, which means that input from previously ineffective regions can now stimulate the neurons. In other words, the population of dorsal horn sensory neurons responding to the MTrP afferent input grows larger. As interneurons are located over various segments, the expansion may reach sensory neurons that supply peripheral areas outside the initial MTrP area. The expansion of pain areas is commonly seen in patients with headaches, whiplash, and other diagnoses (128, 129). There is no nociceptor activity in the referred pain area (44).

Masseter MTrPs can trigger maxillary and mandibular tooth pain, TMJ pain, facial pain, including pain commonly associated with sinusitis, deep ear pain, tinnitus, and frontal and temporal headaches (Figure 3).

Figure 3 – a. Referred pain pattern of the deep masseter muscle

Figure 3 – b. Referred pain pattern of the superficial masseter muscle

Temporalis MTrPs can induce maxillary tooth pain and temporal headaches (Figure 4).

Figure 4 – Referred pain pattern of the temporalis muscle

Medial pterygoid MTrPs result in pain in the mouth, tongue, pharynx, hard palate, and behind the temporomandibular joint (Figure 5).
Lateral pterygoid MTrPs cause TMJ pain and pain in the maxillary sinus (Figure 6).

MTrPs in the posterior belly of the digastric muscle refer pain to the upper part of the sternocleidomastoid muscle, the front of the throat, and sometimes into the occiput. MTrPs in the anterior belly of the digastric muscle refer pain to the mandibular incisors, the alveolar ridge, and the tongue (Figure 7).
Electromyographic Findings

Hubbard and Berkhoff recorded both low-amplitude continuous action potentials (10-50 microvolts), which they labeled “spontaneous electrical activity,” and intermittent spikes (100-600 microvolts) from active MTrPs (130). The electrical activity was only present in sites of active MTrPs and was absent in adjacent non-tender muscle tissue. Subsequent studies indicated that the low-amplitude noise-like potentials represented grossly abnormal endplate activity (EPN) as compared to the normal miniature endplate potentials (MEPP) commonly reported by electromyographers (131-134). In the EMG literature, abnormal EPN is assumed to be associated with normal motor endplates and normal disease-free muscles (135). However, the physiology literature indicates that EPN represents an abnormal concentration of acetylcholine up to three times normal levels (132, 134, 136, 137). Recent studies by Shah and colleagues confirmed high concentrations of acetylcholine at MTrP sites (119, 138).

EPN is not exclusive to MTrPs. Already in 1956, Liley reported that a normal endplate could exhibit abnormal noise-like discharge patterns by mechanically stressing the endplate (139). Simons suggested that Liley’s observation may help to account for the activation of MTrPs by mechanical overload or mechanical trauma (34). In 1997, Maselli confirmed that damage to the endplate caused by the tip of the advancing EMG needle could convert normal miniature endplate potentials (MEPP) to EPN, because of a massive release of acetylcholine (140). Brown and Varkey suggested that the spikes, that Hubbard and Berkhof had observed in MTrPs, could also arise from mechanical stimulation of the endplate, however, Wiederholt had already provided evidence that the spikes indeed originated in the neuromuscular junction (130, 135, 141). In Maselli’s words, the origin of the spikes “corresponds to single muscle fiber action potentials postsynaptically activated by suprathreshold end-plate noise” (140). By using a very slow insertion technique as described by Simons and colleagues, EPN from MTrP can be reliably observed (132, 134).
Integrated Trigger Point Hypothesis

Combining all available supporting evidence of the existence of MTrPs, Simons has recently proposed a new “integrated trigger point hypothesis” (33). The integrated trigger point hypothesis has evolved through several steps of progress since its first introduction as the “energy crisis hypothesis” in 1981 (142). The hypothesis builds on the finding that excessive amounts of acetylcholine from the motor nerve terminal cause miniature motor endplates potentials that produce the endplate noise observed with needle EMG of MTrPs. The excessive acetylcholine maintains a sustained depolarization of the postjunctional membrane, which in turn results in an excessive release of calcium from the sarcoplasmic reticulum and sustained sarcomeric contractations. Shenoi and Nagler suggested that an impaired re-uptake of calcium into the sarcoplasmic reticulum induced by calcium channel blockers may cause MTrPs (143).

According to the integrated trigger point hypothesis, the sarcomeric contractations cause local hypoxia, reducing the available energy supply. The combined decreased energy supply and increased metabolic demand possibly may explain the common finding of abnormal mitochondria in the nerve terminal, referred to as “ragged red fibers” (Henriksson et al. 1993; Henriksson 1999). The local energy crisis would also impair the calcium pump, which would provide another mechanism of the sustained contractures. The calcium pump is responsible for returning intracellular calcium into the sarcoplasmic reticulum against a concentration gradient, which requires a functional energy supply.

As reviewed, hypoxia also stimulates the release of endogenous substances, resulting in hyperalgesia, central sensitization, and stimulation of the autonomic nervous system, which has been shown to increase endplate potentials. For example, an increase in psychological arousal resulted in an immediate increase of endplate spike rates (144, 145). Autogenic relaxation and the administration of the sympathetic blocking agents phentolamine and phenoxybenzamine inhibited the autonomic activation (146-148). Induced autonomic nerve activity would explain autonomic phenomena commonly seen with MPS, and further contribute to the abnormal release of acetylcholine, possibly by increasing the permeability of calcium channels in the cell membrane of the nerve terminal (149, 150). A recent study examined the effects of MTrP massage therapy on the cardiac autonomic tone in healthy subjects. The researchers observed that following MTrP therapy, there was a significant decrease in heart rate, and systolic and diastolic blood pressure, indicating a significant increase in parasympathetic activity (151).

The integrated trigger point hypothesis is summarized in figure 9. The hypothesis is a “work in progress” that is beginning to be subjected to rigorous scientific review and verification.

Figure 9 – Integrated trigger point hypothesis

It is likely that new studies will demonstrate that there are many other factors to consider. For example, preliminary data from current studies by Shah and colleagues provide already new insights in the basic pathophysiology of MTrPs (119). The finding of increased levels of several endogenous substances in the neurochemical milieu of MTrPs, including bradykinin, serotonin, prostaglandin, and CGRP, combined with the finding of a lowered pH may alter the current understanding of motor endplate dysfunction in relation to MTrPs. Both CGRP and a lowered pH can effectively reduce the function of acetylcholinesterase, which could cause an overall increase of available acetylcholine (115-117). At the same time, a lowered pH can activate peripheral nociceptors and trigger capsaicin- sensitive afferents, which in turn can release several of the endogenous
substances from their peripheral endings, including CGRP (152-154). A pH of 6.1 resulted in a threefold increase of the basal CGRP release, which was entirely dependent on the presence of extracellular calcium (155). Bradykinin caused a 50% increase of the basal CGRP release which was also dependent on the presence of extra-cellular calcium (155). Endogenous substances such as bradykinin, serotonin and prostaglandin, clearly interact at multiple levels, including at the vanilloid receptors (120). Although the relevance of these interactions has not been studied specifically in relationship to MTrPs, it is likely that future studies will reveal new aspects of their pathophysiology. Interestingly, Shah and colleagues established an immediate drop in the concentrations of several substances with dry needling of a MTrP (119). If the integrated trigger point hypothesis is basically correct, MTrPs are primarily a muscle disease with secondary but important sensory, motor and autonomic phenomena (156).

Clinical Applications

MPS and MTrPs should be considered in the differential diagnosis of craniofacial pain and temporomandibular joint dysfunction, migraines, tension headaches, radiculopathies, complex regional pain syndrome, whiplash injuries, and most other pain syndromes. To diagnose and manage MPS, clinicians must become well-trained in the identification of MTrPs. Through inactivation of MTrPs, clinicians can facilitate return to function and elimination of pain in both acute and chronic pain patients. Together with the patient, ultimate functional goals need to be discussed. For some patients, eating solid foods may be the final goal. For others, it may be restoring sleep, regaining sexual activity, or returning to work. Treating MTrPs is merely an effective tool to assist patients in regaining lost function (157). There are many different manual techniques, including myofascial release techniques, compression, trigger point compression combined with active contractions of the involved muscle, muscle energy or post-isometric relaxation, connective tissue stretches, and general massage therapy. Needling techniques include superficial and deep dry needling, and trigger point injections (61, 158). For persistent cases, botulinum toxin can be used as it blocks the release of acetylcholine (159, 160). Throughout the treatment process, patients must be educated regarding the etiology, perpetuating factors, and self-management. Patients must learn to modify their behaviors and avoid overloading the muscles without resorting to total inactivity (161).

A sudden onset or a clear remembrance of the onset of pain may indicate an acute activation of MTrPs due to mechanical stress, but it may also indicate a sudden change in the patient’s environment or habits. The mechanical stress may be the result of sudden or abrupt movements, motor vehicle accidents, falls, fractures, joint sprains or dislocations, a direct blow to a muscle, joint, or mandible, excessive exercise or activity, or performing new or unusual activities. A slow insidious onset is usually the result of chronic overloading of tissue, such as habitual chewing gum or tobacco, postural imbalances, poor body mechanics, repetitive movements, and tension as a consequence of psychological or emotional stress (57).

Clinicians should consider both the sensory and mechanical aspects of MTrPs. The physical examination must include a thorough evaluation of MTrPs relevant to the patient’s current pain presentation. The patient’s current area(s) of pain can be visualized through patient pain drawings. While patients communicate their pain patterns, the clinician can begin identifying those muscles and active MTrPs most likely involved in the pain problem. For example, in a patient with complaints of temporal headaches, the pain complaint may direct the clinician to the sternocleidomastoid, trapezius, temporalis and inferior oblique capitis muscles. The patient’s head posture in slight side bending and rotation may implicate the scalene muscles, although the referred pain pattern from MTrPs in the scalene muscles does not include the head region. If the patient in addition presents with a paradoxical breathing pattern, it will be necessary to examine and treat the accessory breathing muscles as well, including the pectoralis minor, scalenes, sternocleidomastoid and upper trapezius musculature that may be overloaded due to increased demands. Teaching the patient a normal diaphragmatic breathing pattern and fostering awareness of relaxation techniques for the upper chest and neck region will aid in the long term management of the headaches. Dentists should limit their direct treatment to muscles cranially from the clavicles to stay within the scope of dental practice.

Habitual postural patterns need to be assessed (162-164). Does the patient have a forward head posture? Are there certain work postures or habits that maintain the head in an antero-position? Forward head posture is associated with multiple static and dynamic changes in total body alignment. Patients with forward head posture present with posterior cervical rotation of the upper cervical and subcranial motion segments, a posteriorly displaced mandible, altered occlusion, as
well as shoulder girdle protraction, internally rotated humeri, increased thoracic kyphosis, a loss of lumbar lordosis, and an increase in posterior pelvic rotation. Dentists need to be familiar with the consequences of forward head posture. For example, if a patient with forward head posture receives a splint, the dentist may have to adjust the splint after the patient’s head posture has been corrected by a physical therapist. Close working relationships between dentists and physical therapists are very important and benefit patients.

Simons, Travell and Simons advocated the spray and stretch technique, which combines use of a vapocoolant spray with passive stretching of the muscle (33). Application of vapocoolant spray stimulates thermal and tactile A-beta skin receptors, thereby inhibiting C-fiber and A-delta fiber afferent nociceptive pathways and muscle spasms, MTrPs, and pain when stretching. Prior to applying the spray and stretch method, the patient is positioned comfortably. The muscle involved is sprayed with a few sweeps of a vapocoolant spray, after which the muscle is stretched passively. With the muscle in the stretched position, the spray is applied again over the skin overlying the entire muscle, starting at the trigger zone and proceeding in the direction of and including the referred pain zone. Following the stretch and spray, the area is heated with a moist heat pack for 5-10 minutes. The patient is encouraged to move the body part several times through the full range of motion. The spray and stretch technique can be used in physical therapy as a separate modality or following myofascial trigger point injections. Muscle stretching can be combined with muscle energy techniques or post isometric relaxation, during which the muscle is stretched after a brief sub-maximal isometric contraction. All techniques are used diagnostically and therapeutically (61, 158). Some patients can learn to use spray and stretch techniques at home. Self treatment programs can be very effective (165).

Inactivation of MTrPs by injection or dry needling appears to be the result of the mechanical action of the needle, since it can be successfully accomplished without the use of local anesthetics or other materials. In 1979, Lewit confirmed that the effects of needling were primarily due to mechanical stimulation of a MTrP with the needle. Dry needling of a MTrP using an acupuncture needle caused immediate analgesia in nearly 87% of needle sites. In over 31% of cases, the analgesia was permanent. Twenty percent had several months of pain relief, 22% several weeks, and 11% several days. Fourteen percent had no relief at all (166).

When using injection needles, the use of a local anesthetic is more comfortable for many patients and results in a longer lasting reduction in MTrP pain (167). The use of solid acupuncture needles versus injection needles has not been examined at this time. After identifying and manually stabilizing the tender area in the taut band with the fingers, the needle is quickly passed through the skin and then into the trigger zone. A LTR or a report of referred pain indicates that the trigger zone has been entered. A small amount, usually 0.1 or 0.2 ml, of local anesthetic may be injected into the trigger zone. There is no benefit to adding steroids. The needle is withdrawn to just below the skin, the angle of the needle changed, and the needle is again passed through the muscle to another trigger zone. A conical volume of muscle can thus be examined for active trigger points without withdrawing the needle through the skin. The trigger zone is explored in this manner until no further LTRs are obtained. Dry needling a MTrP is most effective, when LTRs are elicited (71, 138). A LTR has been shown to inhibit abnormal endplate noise. Current (unpublished) research strongly suggests that a LTR is essential in altering the neurochemical milieu of a MTrP (Shah, 2003, personal communication). Patients commonly describe an immediate reduction or elimination of the pain complaint after eliciting LTRs. Once the pain is reduced, patients can start active stretching and strengthening programs. Eliciting a LTR with dry needling is usually a rather painful procedure. Post-needling soreness may last for one to two days, but can easily be distinguished from the original pain complaint. At this point, the taut band is usually gone, and the spontaneous pain of the trigger point has subsided. Patients who have previously undergone treatment can tell when MTrPs remain, and when they have been sufficiently inactivated. A knowledgeable patient will urge the clinician to continue in an area until a key MTrP is inactivated, at which time there will be a noticeable decrease in pain. The process is repeated until the symptomatic MTrPs are treated throughout the functional muscle unit (61).

There is no limit to the number of MTrP that can be needled. Common sense and patient comfort dictate restraint. Nevertheless, when treating a regional MPS, a sufficient number of muscles in the region must be treated to resolve the problem and allow effective post-needling stretching. All the muscles in a functional muscle unit must be released and returned to full length, if possible, either by needling or by MTrP compression and stretching. Inadequate treatment that leaves critical MTrPs within a functional muscle unit.
usually results in the recurrence of trigger points throughout the muscles of the functional unit. Five to ten different MTrP sites can readily be treated per session, and some physicians and physical therapists skilled in MPS management will treat considerably more in one session. Repeat injections or dry needling into the same area are best done after an interval of one week to allow the muscle to recover. Muscles of the affected functional unit must always be stretched, to their full length if possible, after MTrP needling. Moist heat is applied to the muscle to improve the local circulation and to reduce post-injection soreness. Otherwise, MTrPs will recur because of residual muscle dysfunction. Local anesthetic patches can be applied to reduce the superficial or cutaneous soreness from needling.

Trigger point injections or dry needling are a highly effective way to reduce the local pain and contraction of the taut band. This does not, however, constitute the whole treatment of MPS and MTrPs. The causes that led to the condition must be corrected, when possible. Mechanical, medical, and psychological perpetuating factors must also be eliminated or alleviated in order to reduce the chance of recurrence. Inadequate attention to these aspects of treatment leads to failure to relieve pain and restore function (61, 168).

Summary

Myofascial pain syndrome as defined by Simons, Travell and Simons offers clinicians an excellent entry point into patients’ acute and chronic pain problems. Myofascial trigger points are a common source or contributing factor to many musculoskeletal disorders. In the craniomandibular region, myofascial trigger points are frequently responsible for local and referred pain patterns, causing headaches, TMJ pain and dysfunction, tooth aches, and facial pains. Clinicians should routinely incorporate myofascial trigger points in their examinations and differential diagnoses.

The results of many scientific studies are beginning to validate several aspects of the integrated trigger point hypothesis and refine others. A better understanding and working knowledge of MTrPs and MPS offers an effective approach to relieve pain, restore function, and contribute significantly to patients’ quality of life.

References


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