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Invited Review Paper
Facial Pain

Myofascial pain syndromes in the maxillofacial area: a common but underdiagnosed cause of head and neck pain

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Abstract. Myofascial pain syndromes (MPS) are a large group of muscular disorders, characterized by the presence of hypersensitive spots called trigger points (TP). The maxillofacial region is a high-frequency area for developing TPs. The aim of this paper was to review and summarize the most important methods of management. A literature review was carried out from Medline and database sources. A range of study types were selected for analysis. TP formation and activity result in a reverberating circuit of sustained neural activity. Central mechanisms, primarily associated with psychosocial factors, lead to chronicity. Other synergistic factors are metabolic disorders, nutritional imbalances and regional anatomic disorders. A detailed history and physical examination are important for proper diagnosis. The aim of MPS management is pain relief and restoration of full muscle function. Treatment may require enhancing central inhibition, using pharmacological and/or behavioural techniques, and reducing peripheral inputs, using physical therapy. There are various effective methods of inactivation of TPs. Recognition and reduction of synergistic factors may be important. MPS have a very high prevalence in the general population, despite low awareness among physicians, affecting patients' quality of life. There is a need for interdisciplinary teams of health professionals to achieve proper diagnosis, management and sustainable outcomes.

Keywords: myofascial syndromes; head and neck pain; muscular disorders; trigger points; maxillofacial; treatment.

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Head and neck pain may be associated with inflammation, neoplasm, certain types of arteritis, ophthalmic diseases, orthopedic problems, neurological or psychiatric conditions, and other disorders. Differential diagnosis can be very difficult, and patients may be left under long-term pain-relief treatment without proper diagnosis.

Myofascial pain syndromes (MPS) form a large group of muscular disorders that are not well known by clinical practitioners and are usually underestimated or misdiagnosed, leaving a significant number of patients without proper treatment. The aim of the present study was to review the existing literature on MPS and summarize their most important features and methods of management.

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Materials and methods

An extensive search of the literature was performed in Medline and other available database sources, using the keywords ''myofascial'', ''pain syndromes'', ''maxillofacial'', ''trigger points'', ''taut bands'' ''motor end-plates'', ''nociceptors'' and ''treatment''. Information from electronic links and five related books was included in the analysis of data.

Results

Twenty-four randomized controlled trials, eight controlled clinical trials, 19 clinical trials, 11 laboratory studies, three retrospective studies, 31 reviews and four case reports met the defined criteria and were included in the study selection.

Discussion

MPS are characterized by the presence of hypersensitive spots called trigger points (TPs). Such a syndrome is a regional disorder concerning the muscle, its fascia or both, and is accompanied by pain in an affected area and/or a zone of reference, autonomous phenomena and malfunction of the affected muscle[30,95]. The presence of MPS in the maxillofacial area mainly concerns the masseter, temporalis, lateral and medial pterygoid muscles.

A TP elicits pain after palpation and/or causes pain radiation towards a zone of reference, and a local twitch response[2]. It can be active or latent. Active TPs cause specific pain during muscle movement, impeding full extension of the muscle and decreasing the range of motion. They also display pain during relaxation, resulting in continuous pain in the zone of reference which can be accompanied by autonomous phenomena. The final outcome may be muscle degeneration. Latent TPs are pressure sensitive and become painful only during palpation. They can be a predisposing factor for muscular malfunction. This state of latency can remain for years, resulting in acute painful bouts when activation occurs.

Pain in the reference zone originates from a TP and is perceived in a distant location. It can be accompanied by regional vasoconstriction, perspiration, sialorrhea and pilomotor phenomena. The characteristics of MPS may long outlast the initiating events, setting up a self-induced pain cycle that is perpetuated through continuing muscle tension and pain-reinforcing behavior, improper treatment and failure to reduce other synergistic factors.

Bibliographic references to MPS have made since the 16th century under various terms, such as muscular rheumatism, myalgic nodules, myalgia, myositis and fibrosis. MPS seem to be very common in the USA[25], as approximately 15% of the general population are estimated to have myofascial pain[98], and 21%–93% of people with localized pain may have MPS[25]. The prevalence has been seriously underestimated in current clinical practice, as a considerable number of physicians may not even know of the existence of MPS.

Although the presence of MPS is independent of age, the prevailing opinion is that the risk of developing active TPs is concomitant with age with a peak between 40 and 50 years, and there is a higher incidence among women. In older people, when activity is diminished, latent TPs appear prevalent. Nearly every American citizen develops a TP at some point in his life, and 25%–54% of people without symptoms have latent TPs. The maxillofacial region is considered a high-frequency area for developing active TPs, along with the neck area, shoulders, glutei and spinal muscles. A lower incidence is observed in the upper trapezius, scalene, sternocleidomastoid, levator of scapula and illiocostal muscle of the loins.

Women face almost three times the risk of developing chronic masticatory myofascial pain than men[97]. Research on female twins has supported the importance of environmental influence, as no difference in frequency of symptoms related to myofascial pain dysfunction syndromes was found between monozygous (identical) and dizygous (non-identical) female twins[43].

MPS are without doubt among the most common painful syndromes. When they become chronic, they are one of the primary causes of functional disability, leave of absence and compensation claims in the labor force.

Biopsies have shown that TPs are discrete spots, ranging from 2 to 5 mm in diameter, found within hard palpable bands of skeletal muscle and the fascial structure of tendons and ligaments[30]. Within each TP there is a hyperirritable spot, the 'taut band', which is composed of hyper-contracted extrafusal muscle fibres[98]. As well as the distinction between active and latent TPs, the terms satellite and secondary are used to describe TPs within the pain reference zone of another TP and those developed in either synergists or antagonists of the primarily affected muscle, respectively.

The structural changes that occur in a TP are visible only using electron microscopy. Biopsies of patients with palpable and very painful TPs showed both muscle fibre changes, in the form of local muscle dystrophy and complete disruption of cross striation, and alterations in the interstitial spaces, such as increases in mast cells, lymphocytes and leucocytes, with connective tissue proliferation[66]. A 'moth-eaten' appearance of type I and type II fibres was discovered in a later study conducted by Yunus et al.[106]. Ultra-microscopic findings in 12 patients demonstrated significant changes, such as myofibrillar lysis with deposition of glycogen and abnormal mitochondria as well as subsarcolemmal accumulation of glycogen and mitochondria, and in 11 of them papillary projections of the sarcolemmal membrane. More recently, biopsies of myofascial tissue in the vicinity of TPs have revealed 'contraction knots', described as large, rounded, darkly staining muscle fibres, and a statistically significant increase in the average diameter of muscle fibres[65]. In another study, biopsies demonstrated segmental shortening of groups of sarcomeres in individual muscle fibres, and possibly waves of contracted sarcomeres to account for palpable taut bands[86]. Other morphological disorders include formation of hyaline bodies in muscle fibres and deposition of non-specific inflammatory factors.

The formation of TPs is believed to occur as a consequence of biomechanical factors[63] that act either acutely, following a sudden muscle overload (acute trauma), or contribute to a more gradual development, following prolonged contractions or repetitive activity (repetitive microtrauma). TP development secondary to rheumatoid arthritis, lupus erythematosus, scleroderma, nerve injuries[36] and visceral diseases has been observed. Regardless of the cause, these injuries are thought to release cytokines, especially bradykinin, histamine, serotonin and prostaglandins, which stimulate nociceptors near the end-plate region. A great concentration of sensitive loci can be found in this area[45] which may contain one or more of the sensitized nociceptive nerve endings.

The correlation between motor end-plates and TPs was first elucidated by Gunn and Milbrandt[41]. The pathophysiological and biochemical mechanisms that occur in the dysfunctional end-plate area are thought to include presynaptic disorders in the α-motor neurons leading to excessive acetylcholine (ACh) release[65,87], as well as synaptic and post-synaptic dysfunction due to prolonged ACh presence following acetylcholinesterase (AChE) deficiency and

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nicotine ACh receptor (nAChR) hyperactivity or hypersusceptibility, respectively. These dysfunctions can be genetic or acquired[56,58,72,76]. Excessive ACh release in the synaptic cleft following Ca^{++} channel dysfunction, sarcoplasmic reticulum destruction, prolonged ACh presence due to AChE deficiency, or even constant or extremely sensitized activity of the nAChR post synaptically leads to a vicious circle: excessive muscle contraction compresses the local sensory nerves and reduces the axoplasmic transport of molecules that normally inhibit ACh release[34,44].

Compression of local blood vessels can also occur when muscle contraction is prolonged, resulting in a reduction in the local supply of oxygen. The combination of poor oxygen supply and increased metabolic demand results in the rapid depletion of local ATP reserves, generating an ATP energy crisis[87]. ATP shortage results in decreased inhibition of ACh release in the α-neuron and impairs the normal function of the Ca^{++} pump in the muscle cell, decreasing the re-uptake of Ca^{++} and perpetuating the cycle. This hypothetical model is supported by biochemical findings, such as decreased levels of ATP, ADP and phosphoryl creatine, and increased levels of AMP and creatine in muscle biopsy samples[6], along with evidence of abnormal tissue oxygenation[59]. Other biochemical findings include localized increases of tissue water, chloride and acid mucopolysaccharides, decreased levels of serum lactic dehydrogenase fraction 1 (LDH1), and increased levels of LDH3, LDH4, LDH5 and muscle aldolase[47], indicative of inflammatory changes similar to polymyositis during TP development.

Focal demyelination of sensory nerves may be a result of the local production of sensitizing substances, creating abnormal impulse-generating sites capable of generating ectopic nociceptive impulses[10]. Dysregulation of the γ-motoneuron circuitry, leading to muscle disinhibition, is also proposed as a mechanism for the development of TP activity. The outcome of this dysregulation may be muscle hyperactivity after contraction, excessive electrical activity during movement, and/or inappropriate co-activity with other muscles during movement[16].

TP formation and consequent activity result in sensitization of the dorsal horns in certain spinal cord segments, and the establishment of a reverberating circuit of sustained neural activity[98]. Sensitization of the central nervous system is accelerated in the presence of abnormal impulse-generating sites[63], while the sensitized dorsal horns also receive nociceptive signals, concerning somatic or visceral dysfunctions, which converge in the same spinal cord segment[63]. Sensitization of second-order neurons in the brainstem, due to the activation of N-methyl-D-aspartate receptors, may be involved.

Other theories explaining the persistence of pain in MPS include spinal reflexes that alter the biochemical environment of the nociceptors in the skeletal muscles[64] and differences in muscle physiology[71]. A neural basis involving central pain, and/or peripheral hyperalgesia, has been considered[75]. Muscle dysfunction in MPS may be regarded as myospastic activity[52] and MPS may represent focal dystonia[80]. The fact that disturbance in non-rapid eye movement sleep has been related to a temporary appearance of musculoskeletal symptoms suggests that myofascial pain may be a non-restorative sleep syndrome[68]. MPS can be perpetuated by protective splinting of the affected muscle and by avoiding painful stretching. If normal muscle length is not restored and the painful process continues, the muscle band initially responds with hypertrophy, which later evolves to dystrophic changes and localized fibrosis.

Certain aspects of the above theories on the pathology of MPS have received criticism. Since the painful muscles in MPS are electrically silent, the suggestion of myofibrillar or myofascicular contraction is considered highly improbable[18]. The recovery from muscle injuries in normal subjects is complete, unless muscle tears or deep haematomas exist[67]. This debate and related research continue.

Biomechanical strain seems to be implicated in the onset of MPS, but central mechanisms primarily associated with psychosocial factors lead to the chronicity of the syndromes[29]. A large survey in Israel demonstrated that 1 in 6 patients with chronic widespread pain were found to have a mental disorder[9], and another study has suggested that MPS patients have a remarkable hypochondriac tendency and irrational way of thinking[53]. Evidence from electromyogram (EMG) responses indicates that patients with MPS display a stereotypic response to stress via increased activity in the facial muscles[51]. Although it is difficult to establish solid evidence of a direct relationship between stress and MPS, higher than normal levels of urine catecholamines and 17-hydroxycorticosteroids, which are usually indicative of stressful situations, were found in a group of patients with MPS[21]. Stress-related muscle habits, such as bruxism, result in myofascial pain[35] through augmented contraction of the masticatory muscles, and abnormal swallow patterns may predispose to the development of MPS due to hyperactivity of the digastric muscles[38]. Anxiety and depression have also been associated with masticatory myofascial pain[97].

The presence of metabolic disorders, such as McArdle's disease[94], may facilitate the development of MPS, while other disturbances such as hypothyroidism, hyperuricaemia, increased creatine levels, oestrogen deficiency, mild iron deficiency, anaemia, and low potassium and calcium reserves seem to be involved to some extent[95]. Nutritional disorders, such as low levels of C, E and B complex vitamins in the blood along with decreased plasma folic acid (common in chronic alcoholism), are also considered to be synergistic factors for the development of TPs[95]. Temporomandibular joint disorders (TMD), internal derangements, cervical osteoarthritis and disc disease can contribute to the development of TPs, mainly as a result of reflex muscle splinting to protect the joint from aggravating movement[95].

Some of these factors may be a result and not the cause, and the old 'chicken–egg' question is valid in many cases of MPS. For example, is stress a contributing factor to these syndromes, or are these syndromes causing stress to the patients?

At present, there is no reliable, objective test to confirm the presence of myofascial TPs[57] and therefore no gold-standard diagnostic criterion exists[84]. The diagnosis of MPS in the maxillofacial area is based upon the history and physical examination of the patient, which can be further assisted by laboratory findings indicative of predisposing or synergistic factors, such as hypothyroidism, vitamin deficiencies, hyperuricaemia, McArdle's disease, etc.

Imaging methods include ultrasonography, which has shown significant differences in the thickness at rest, and the increase ratio by contraction, between patients and control groups, possibly related to muscle oedema[3]. Temperature measurements of the skin overlying the upper trapezius muscle, using liquid crystal thermography, in patients with TMD have showed increased temperature on the symptomatic side[78]. The diagnostic value of this method has not been well documented[92].

Although routine EMG studies have failed to show significant abnormalities associated with TPs[18], such as evidence of denervation or focal muscle spasm, some specialized EMG studies have

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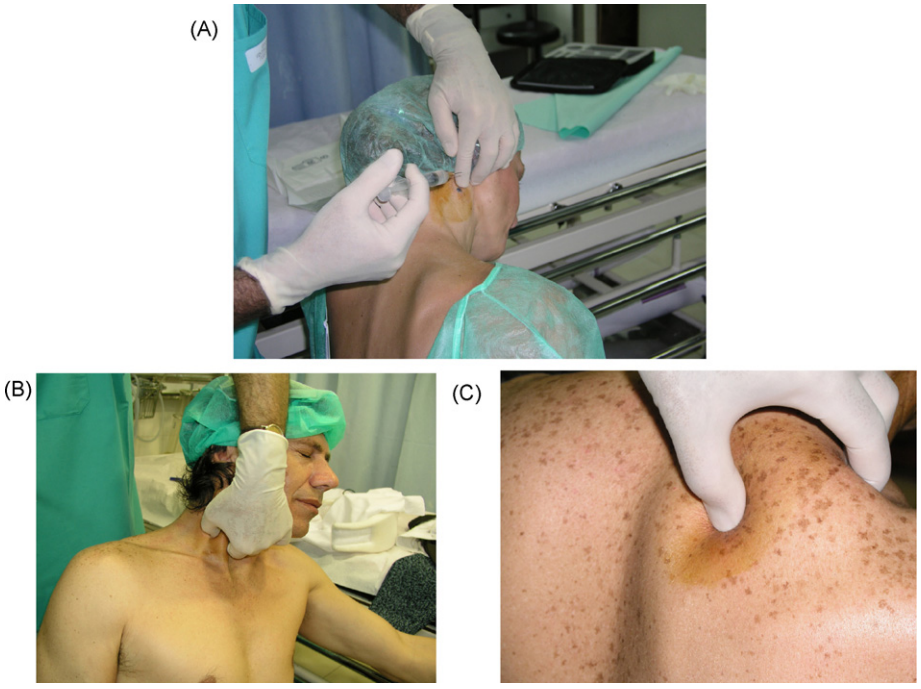

Fig. 1. Physical examination.

revealed certain differences. End-plate noise is significantly more prevalent in myofascial TPs than at other sites within the end-plate zone, suggesting that it may be a characteristic of the TP region but not restricted to it[85]. An interesting EMG difference appears between active and latent TPs during TP needling. The former show bilateral or mirror-image EMG activity associated with unilateral needle stimulation[4].

Since laboratory testing and imaging techniques cannot reliably diagnose MPS, a detailed history and physical examination are essential. The latter requires specific skills, such as innate palpation ability, authoritative training and extensive clinical experience[86]. Problems of reliability concerning findings of taut bands, muscle twitch and active TPs occur, even among experts[104]. Pain on palpation of a TP can be prompt or delayed for a few seconds. Usually this pain is deep and blunt, causing muscular dysfunction and reducing the range of muscle movement, but its intensity depends on the irritability of the TP rather than the size of the affected muscle. The pattern of pain referral is both reproducible in the same patient and consistent between patients, enabling the clinician to use the zone of reference to pinpoint the TP in question. The patient's behavioural reaction to firm palpation of a TP is characterized as a positive 'jump sign' and is a distinguishing feature of MPS. Attention should be paid during physical examination to abnormalities, such as malocclusion and protective or restricted movements, during jaw opening. Taut bands can be felt either by exerting pressure with the fingertips along the muscle (Fig. 1A, left hand) or by 'cupping' the muscle venter between thumb and index finger and rolling forwards and backwards (Fig. 1B and C).

TPs in the superficial layers of the masseter muscle cause referral pain in the mandible, molars and related gingivae, whereas the pain from deep-layer TPs reflects at the temporomandibular joint (TMJ) and into the ear. Inspection of the masseter muscle is performed with an open mouth in order not to cause pain as a result of muscle tension. Palpation with regard to the superficial layers is performed either by pressing the muscle against the mandible (for anterior TPs, Fig. 2A), or by 'cupping' the muscle between thumb and index finger (Fig. 2B). Deeper TPs may be found along

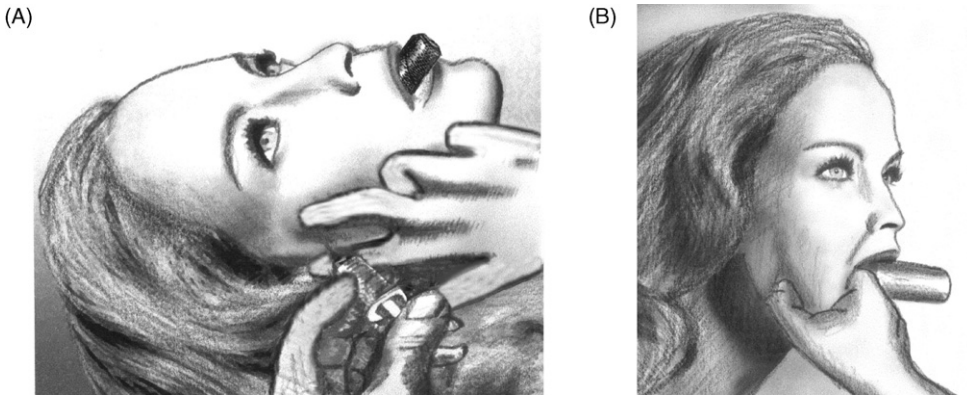

Fig. 2. Physical examination for TPs in the masseter muscle.

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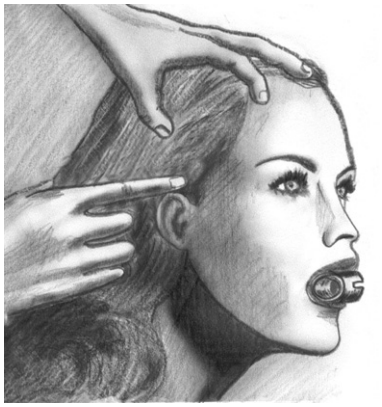

Fig. 3. Physical examination for TPs in the temporalis muscle.

the ramus of the mandible or the floor of the maxillary sinus.

TPs in the temporalis cause pain in the supraorbital area and incisors of the maxilla (anterior section), premolar area (middle section) or upper molars and occipital area (posterior section). Inspection of the temporalis muscle is facilitated when the jaws are wide open so that the muscle is slightly tensed. The anterior and middle section can be checked on the upper limit of the zygomatic arch, and the posterior section over the ear (Fig. 3). The inspection is completed from inside the mouth, with palpation of the temporalis insertion on the coronoid process.

TPs of the medial pterygoid muscle cause pain in the tongue, hard palate, TMJ and cervical muscles, swallowing difficulties and decreased jaw opening, whereas TPs of the lateral pterygoid cause deep pain in the TMJ and maxilla and mastication disorders. The former can be palpated either outside (Fig. 4A) or through the open mouth (Fig. 4B). The latter require a jaw opening of 5–8 mm with the muscle palpated between the cyst of Stafne and the zygomatic process (Fig. 4C and D).

The differential diagnosis of MPS in the maxillofacial area includes TMD, arthritis, fibromyalgia, temporal arteritis, fibrositis and polymyositis.

MPS may settle without medical intervention within 5–10 days, but if they become chronic can be quite disabling. Early diagnosis and targeted treatment may prevent chronicity occurring[27]. The aim of MPS management is pain relief and restoration of full-muscle function, which is associated with complete muscle length, posture and full joint range of motion, to avoid chronic complications such as muscular dystrophy and permanent disability. Treatment may require enhancing central inhibition, with the use of pharmacological and/or behavioural techniques, and reducing peripheral inputs, using physical therapy (exercises and TP-specific treatment)[40]. Recognition and reduction of other synergistic factors[33] or correction of possibly unconscious postural habits[19] may be important.

Methods of inactivation of TPs include non-invasive mechanical disruption (using massage, acupressure or ultrasound), skin and muscle temperature changes (using vapour coolants, moist heat application, magnetic stimulation, electrical stimulation or laser therapy), and direct mechanical or chemical treatment (TP injections, acupuncture or anaesthetic patches).

Massage therapy is considered more effective than ultrasound application in reducing the number and intensity of TPs[31], but when combined with neck-stretching techniques ultrasound treatment has been found to be as effective as TP injection[20]. When a high level of cooperation between the patient and therapist was established, a high-power, pain-threshold, static ultrasound technique, before stretching the muscle, was found to be more effective than conventional ultrasound in acute MPS[60].

With the stretch-and-spray technique, a mild application of vapour coolant spray is followed by passive stretching, providing immediate reduction in pain (Fig. 5). TP inactivation may be more attributable to the muscle stretching than freezing. This technique can be applied in cases of multiple TPs where injection may be difficult to perform. Moist heat application after stretching may further aid muscle relaxation. Passive stretching of the masticatory muscles can be accomplished by placing a trimmed and sterile cork between the incisors. During active stretching, rapid movements of the muscles should be avoided in order to reduce risk of injury.

Transcutaneous electrical nerve stimulation is one of the most frequently employed treatments in MPS, resulting in significant improvement in TP charac-

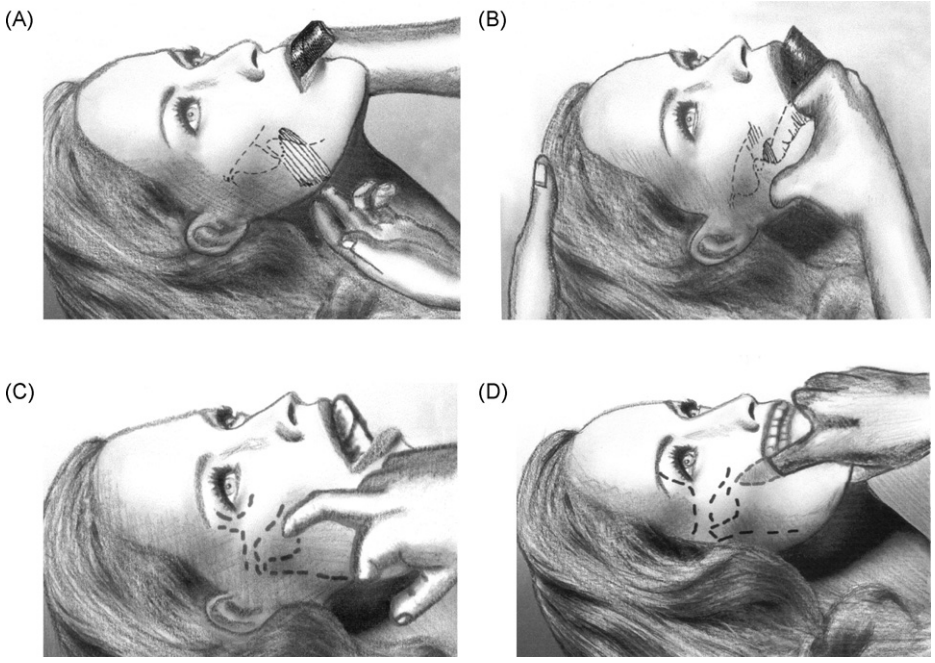

Fig. 4. Physical examination for TPs in the pterygoid muscles.

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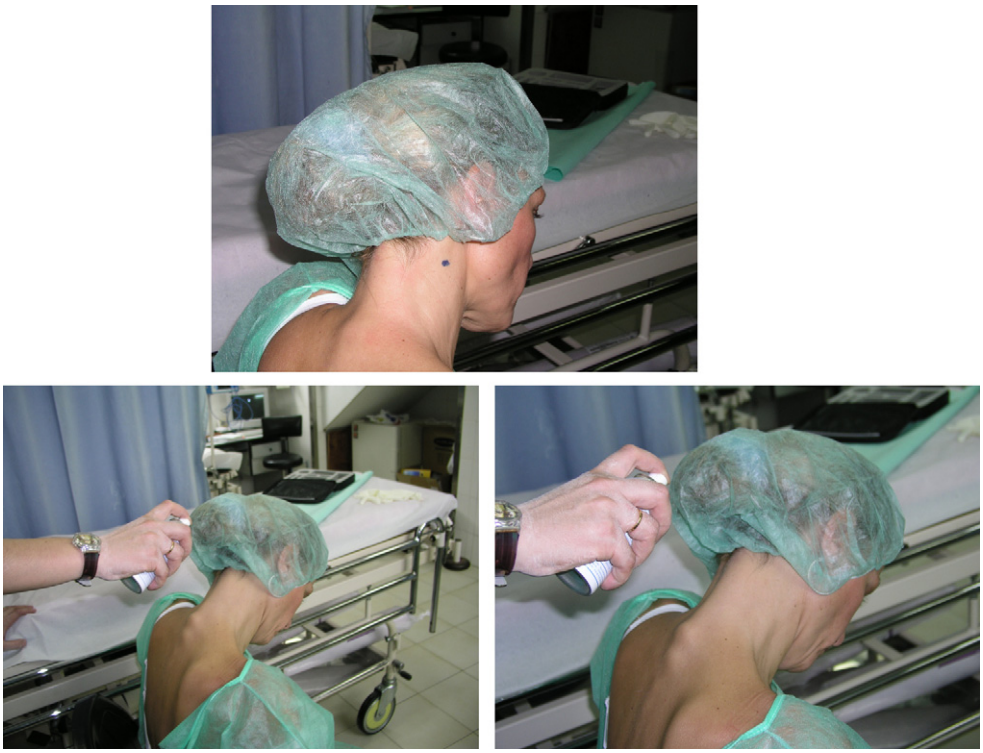

Fig. 5. Stretch-and-spray technique.

teristics and range of motion of the affected muscles. Modified therapeutic regimens, such as frequency-modulated neural stimulation, seem more effective with regard to medium-term outcomes or algometry measurements[23]. Peripheral repetitive magnetic stimulation was also found to be more effective in terms of algometry measurements and medium-to-long-term outcomes[88].

Low-level laser therapy could be effective in pain relief and improvement of muscle function and quality of life in patients with MPS[42]. The decrease in pain at rest and during activity has been either temporary[48] or not statistically significant compared to placebo[1,17], but no double-blind randomized studies have to date included the maxillofacial area.

TP injections have been found to reduce pain and increase both range of motion and exercise tolerance. When a TP is needled, a sudden contraction of the muscle called 'local twitch response' is observed, as well as a temporary intensification of pain over the muscle or in the zone of reference. Relief of pain should occur within a few minutes, after which full-range manual stretching of the muscle can be attempted. The presence of persistent pain, 2–8 h after needling, is frequent and attributed to microscopic haemorrhage which may occur during penetration. This phenomenon can be avoided using a local anaesthetic such as procaine 0.5% or lidocaine 0.5% or 1% (Fig. 6). Although local anaesthetics should induce a nerve conduction block that facilitates muscular activity, in one study they were not found to be superior to normal saline[96]. More recently, lidocaine 0.5% injection has been considered as the treatment of choice in MPS[50]. Some clinicians suggest adding corticosteroids to the local anaesthetic to increase effectiveness over inflamed fascial structures, such as joints and ligaments.

Chemical treatment of a TP includes botulinum toxin (BTX) and topisetron injection. BTXs have clinical application when sustained focal muscle relaxation is required, and can be advised for pain relief in cases of predominant dystonia and spasticity[5]. In principle, their action inhibits muscle contraction by blocking the release of ACh from peripheral nerves. There is a possibility that BTXs block γ-motor end-

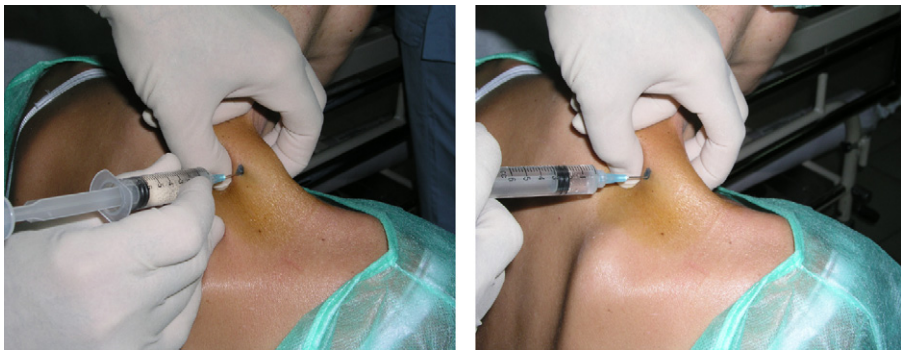

Fig. 6. Injection of local anaesthetic.

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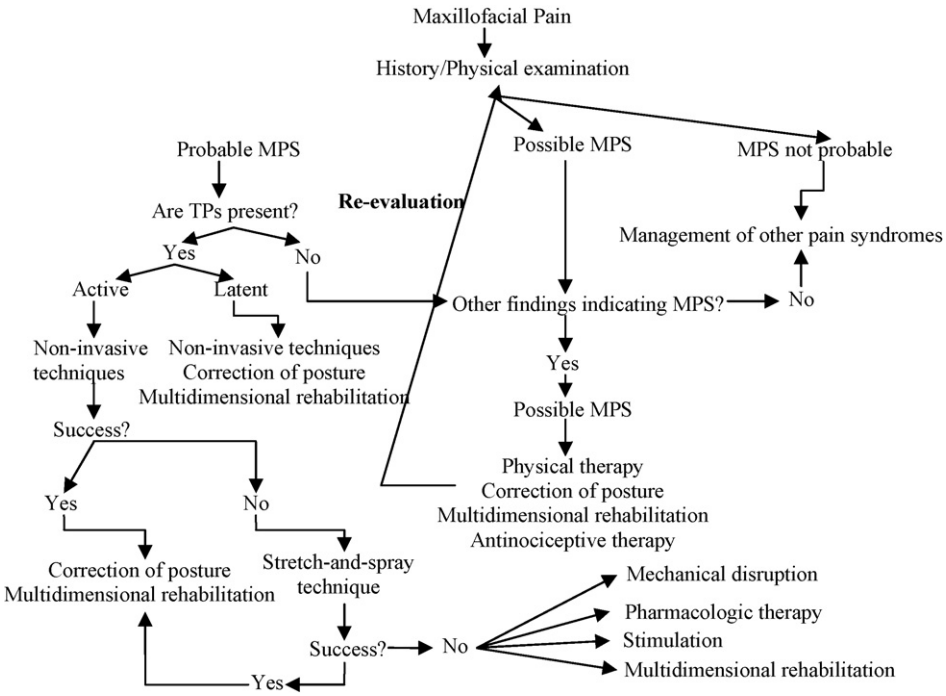

Fig. 7. A flow chart for MPS management.

ings in addition to α-motor endings. This may reduce spindle inflow to α-motoneurons resulting in a decrease of the reflex muscular tone[24,81,101]. The non-toxic components of the BTX complex may have unrecognised clinical significance in terms of retarding tissue diffusion or imparting stability to the complex[8].

Serotype A (BTX-A), which produces a dose-dependent relaxation[28], is the only BTX currently used for TP injections[55]. BTX-A injections can significantly reduce spasticity- and dystonia-related local pain[49,103] or focal muscular pain, and in many cases the benefit outlasts the expected action of the toxin[99]. Results from a double-blind, placebo-controlled study showed at least a 30% reduction in pain in four out of six patients with chronic MPS following injection with BTX-A but not normal saline, as estimated by visual analog scales, verbal descriptors for pain intensity and unpleasantness, palpable muscle firmness, and pressure pain thresholds[11].

BTX-A injection can be considered an effective treatment for patients with MPS[14] and may be selectively used in patients resistant to conventional methods of treatment[50], but the cost effectiveness should be further assessed. Two randomised trials showed BTX-A injections to be as efficient as topical anaesthetics (bupivacaine 0.5%, lidocaine 0.5%) in terms of duration or magnitude of pain relief, function and quality of life, but less cost effective[39,50]. Other randomized trials do not support a specific antinociceptive and analgesic effect of BTX-A compared to placebo (normal saline)[79], although this discrepancy may be partially attributable to adverse events that occur in the treatment group due to the nature of the toxin (i.e. weakness)[100]. Efforts to reduce the prevalence of such events by the employment of low-dose protocols were not found to be successful in terms of treatment efficiency[73]. Dose-ranging studies to determine the optimal treatment as well as potential immunoresistance from repeated injections, and the value of EMG guidance regarding the outcome of treatment, remain important clinical issues to be addressed[93]. The prevalence of end-plate noise in the myofascial TP region may work as an objective indicator of the therapeutic effectiveness of BTX-A injection[54].

Local injections of topisetron, a 5-hydroxytryptamine3 receptor antagonist, may contribute to rapid and prolonged relief, with an analgesic effect far superior to the action of local anaesthetics[70]. The efficacy of this drug has also been attributed to its antiphlogistic effect, which may be related to the inhibited release of substance P and other neuropeptides from nociceptors, and the blocked release of phlogistic substances from macrophages, monocytes, etc[22,69,70,91].

Chemical TP blockage with topical lidocaine 5% patches was also found to be effective in reducing average pain intensity and improving quality of life ($P < 0.05$), and can be used in the management of myofascial pain[12,13].

Acupuncture exerts a positive influence on the signs and symptoms of MPS[90], and provides statistically significant short-term pain reduction in chronic orofacial pain ($P < 0.0001$)[37]. During rehabilitation, an appropriate exercise regime should also be followed to restore flexibility and balance to the muscles, in order to avoid high recurrence rates[105].

A variety of factors (social, psychological, economic, regarding substance abuse, etc.) may influence the treatment outcome of TPs, confirming the multidimensional aspect of MPS[46]. Multidimensional rehabilitation programs that include cognitive behaviour therapy show a statistically significant decrease in pain intensity, duration and frequency compared to baseline measurements in patients with MPS[7], and a significant increase in quality of life over time[102].

Physical manoeuvres may also be an effective therapy[82]. Due to the difficulty in isolating a sole aetiologic factor for MPS, their employment seems quite reasonable, especially at the beginning of treatment[74]. Exercise protocols for the masticatory muscles show a statistically significant reduction in pain compared to controls ($P = 0.019$), and produce objective physiologic results on EMG during maximal voluntary clench ($P = 0.007$)[32].

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When physical therapy is combined with counselling it may result in a significant improvement in pain and jaw function[15], as chronic pain is often a product of both physical and psychosocial factors that complicate convalescence[98].

Pharmacologic treatment of MPS may involve alpha-2 agonists that have myorelaxant properties, such as tizanidine hydrochloride[89]. A significant decrease in pain intensity and disability compared to baseline measurements ($P < 0.001$) and an improvement in pressure thresholds and sleep during treatment ($P < 0.001$) were reported in a clinical trial that assessed the efficacy of tizanidine. This drug has been rated as good to excellent in relieving pain by 89% of patients and 79% of their doctors[61]. In another study, 54% of MPS patients treated with tizanidine showed absence of clinical symptoms and 23% showed improvement[62]. Other drugs that have antinociceptive effects in MPS-associated pain and may be used in chronic refractory situations include clonazepam[26] and tricyclic antidepressants[77]. Interventions concerning dietary tryptophan uptake have also been attempted[83].

In conclusion, MPS have a high prevalence in the general population despite the low level of awareness among doctors, and affect patients' quality of life. The management of MPS is a long-term process that requires close cooperation between patients and physicians (Fig. 7). Difficulties in diagnosis and management that may influence long-term results lie not only in treating TPs but in changing factors related to the patient's attitudes, way of living, and social and physical environment. There is a great need for interdisciplinary teams of various health professionals in order to achieve proper diagnosis, management and sustainable outcomes.

References

1. Altan L, Bingol U, Aykac M, Yurtkuran M. Investigation of the effect of GaAs laser therapy on cervical myofascial pain syndrome. Rheumatol Int 2005: **25**: 23–27.
2. Alvarez DJ, Rockwell PG. Trigger points. diagnosis and management. Am Fam Physician 2002: **65**: 653–660.
3. Ariji Y, Sakuma S, Izumi M, Sasaki J, Kurita K, Ogi N, Nojiri M, Nakagawa M, Takenaka M, Katsuse S, Ariji E. Ultrasonographic features of the masseter muscle in female patients with temporomandibular disorder associated with myofascial pain. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004: **98**: 337–341.
4. Audette JF, Wang F, Smith H. Bilateral activation of motor unit potentials with unilateral needle stimulation of active myofascial trigger points. Am J Phys Med Rehabil 2004: **83**: 368–374 quiz 375–7, 389.
5. Benecke R, Dressler D, Kunesch E, Probst T. The introduction of botulinum toxin in the therapy of muscular pain [in German]. Schmerz 2003: **17**: 450–458.
6. Bengtsson A, Henriksson KG, Larsson J. Reduced high-energy phosphate levels in the painful muscles of patients with primary fibromyalgia. Arthritis Rheum 1986: **29**: 817–821.
7. Bogart RK, McDaniel RJ, Dunn WJ, Hunter C, Peterson AL, Wright EF. Efficacy of group cognitive behavior therapy for the treatment of masticatory myofascial pain. Mil Med 2007: **172**: 169–174.
8. Borodic GE, Acquadro M, Johnson EA. Botulinum toxin therapy for pain and inflammatory disorders: Mechanisms and therapeutic effects. Expert Opin Investig Drugs 2001: **10**: 1531–1544.
9. Buskila D. Fibromyalgia, chronic fatigue syndrome, and myofascial pain syndrome. Curr Opin Rheumatol 2001: **13**: 117–127.
10. Butler DS. The Sensitive Nervous System. Adelaide, Australia: Noigroup Publications 2000.
11. Cheshire WP, Abashian SW, Mann JD. Botulinum toxin in the treatment of myofascial pain syndrome. Pain 1994: **59**: 65–69.
12. Dalpiaz AS, Dodds TA. Myofascial pain response to topical lidocaine patch therapy: case report. J Pain Palliative Care Pharmacother 2001: **16**: 99–104.
13. Dalpiaz AS, Lordon SP, Lipman AG. Topical lidocaine patch therapy for myofascial pain. J Pain Palliative Care Pharmacother 2004: **18**: 15–34.
14. De Andres J, Cerda-Olmedo G, Valia JC, Monsalve V, Lopez-Alarcon. Minguez A. Use of botulinum toxin in the treatment of chronic myofascial pain. Clin J Pain 2003: **19**: 269–275.
15. De Laat A, Stappaerts K, Papy S. Counseling and physical therapy as treatment for myofascial pain of the masticatory system. J Orofac Pain 2003: **17**: 42–49.
16. Donaldson CC, Nelson DV, Schulz R. Disinhibition in the gamma motoneuron circuitry: a neglected mechanism for understanding myofascial pain syndromes? Appl Psychophysiol Biofeedback 1998: **23**: 43–57.
17. Dundar U, Evcik D, Samli F, Pusak H, Kavuncu V. The effect of gallium arsenide aluminum laser therapy in the management of cervical myofascial pain syndrome: a double blind, placebo-controlled study. Clin Rheumatol 2007: **26**: 930–934.
18. Durette MR, Rodriquez AA, Agre JC, Silverman JL. Needle electromyographic evaluation of patients with myofascial or fibromyalgic pain. Am J Phys Med Rehabil 1991: **70**: 154–156.
19. Edwards J. The importance of postural habits in perpetuating myofascial trigger point pain. Acupunct Med 2005: **23**: 77–82.
20. Esenyel M, Caglar N, Aldemir T. Treatment of myofascial pain. Am J Phys Med Rehabil 2000: **79**: 48–52.
21. Evaskus DS, Laskin DM. A biochemical measure of stress in patients with myofascial pain-dysfunction syndrome. J Dent Res 1972: **51**: 1464–1466.
22. Farber L, Haus U, Spath M, Drechsler S. Physiology and pathophysiology of the 5-HT3 receptor. Scand J Rheumatol Suppl (119):2004: 2–8.
23. Farina S, Casarotto M, Benelle M, Tinazzi M, Fiaschi A, Goldoni M, Smania N. A randomized controlled study on the effect of two different treatments (FREMS AND TENS) in myofascial pain syndrome. Eura Medicophys 2004: **40**: 293–301.
24. Filippi GM, Errico P, Santarelli R, Bagolini B, Manni E. Botulinum A toxin effects on rat jaw muscle spindles. Acta Otolaryngol 1993: **113**: 400–404.
25. Finley JE. Myofascial Pain. Physical Medicine and Rehabilitation. 2006. [electronic link: www.eMedicine.com, accessed Dec 3, 2006].
26. Fishbain DA, Cutler RB, Rosomoff HL, Rosomoff RS. Clonazepam open clinical treatment trial for myofascial syndrome associated chronic pain. Pain Med 2000: **1**: 332–339.
27. Forst R, Ingenhorst A. Myofascial pain syndrome [in German]. Internist (Berl) 2005: **46**: 1207–1217.
28. Freund BJ, Schwartz M. Treatment of whiplash associated neck pain with botulinum toxin-A: a pilot study. J Rheumatol 2000: **27**: 481–484.
29. Fricton JR. Masticatory myofascial pain: an explanatory model integrating clinical, epidemiological and basic science research. Bull Group Int Rech Sci Stomatol Odontol 1999: **41**: 14–25.
30. Fricton JR. Myofascial pain syndrome. Neurol Clin 1989: **7**: 413–427.
31. Gam AN, Warming S, Larsen LH, Jensen B, Hoydalsmo O, Allon I, Andersen B, Gotzsche NE, Petersen M, Mathiesen B. Treatment of myofascial trigger-points with ultrasound combined with massage and exercise–a randomised controlled trial. Pain 1998: **77**: 73–79.
32. Gavish A, Winocur E, Astandzelov-Nachmias T, Gazit E. Effect of controlled masticatory exercise on pain and muscle performance in myofascial pain patients: A pilot study. Cranio 2006: **24**: 184–190.
33. Gerwin RD. A review of myofascial pain and fibromyalgia–factors that pro-

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mote their persistence. Acupunct Med 2005: **23**: 121–134.
34. Gessa GL, Casu MA, Carta G, Mascia MS. Cannabinoids decrease acetylcholine release in the medial-prefrontal cortex and hippocampus. Eur J Pharmacol 1998: **355**: 119–124.
35. Glaros AG, Williams K, Lausten L. The role of parafunctions, emotions and stress in predicting facial pain. J Am Dent Assoc 2005: **136**: 451–458.
36. Glyn JH. Rheumatic pains: Some concepts and hypotheses. Proc R Soc Med 1971: **64**: 354–360.
37. Goddard G. Short term pain reduction with acupuncture treatment for chronic orofacial pain patients. Med Sci Monit 2005: **11**: CR71–CR74.
38. Goldstein LB, Last FC, Salerno VM. Prevalence of hyperactive digastric muscles during swallowing as measured by electromyography in patients with myofascial pain dysfunction syndrome. Funct Orthod 1997: **14**: 18–22 24.
39. Graboski CL, Gray DS, Burnham RS. Botulinum toxin A versus bupivacaine trigger point injections for the treatment of myofascial pain syndrome: a randomised double blind crossover study. Pain 2005: **118**: 170–175.
40. Graff-Radford SB. Myofascial pain: diagnosis and management. Curr Pain Headache Rep 2004: **8**: 463–467.
41. Gunn CC, Milbrandt WE. Tenderness at motor points: an aid in the diagnosis of pain in the shoulder referred from the cervical spine. J Am Osteopath Assoc 1977: **77**: 196–212.
42. Gur A, Sarac AJ, Cevik R, Altindag O, Sarac S. Efficacy of 904 nm gallium arsenide low level laser therapy in the management of chronic myofascial pain in the neck: a double-blind and randomize-controlled trial. Lasers Surg Med 2004: **35**: 229–235.
43. Heiberg A, Heloe B, Heiberg AN, Heloe LA, Magnus P, Berg K, Nance WE. Myofascial pain dysfunction (MPD) syndrome in twins. Community Dent Oral Epidemiol 1980: **8**: 434–436.
44. Hohmann AG, Herkenham M. Cannabinoid receptors undergo axonal flow in sensory nerves. Neuroscience 1999: **92**: 1171–1175.
45. Hong CZ. Pathophysiology of myofascial trigger point. J Formos Med Assoc 1996: **95**: 93–104.
46. Hopwood MB, Abram SE. Factors associated with failure of trigger point injections. Clin J Pain 1994: **10**: 227–234.
47. Ibraham GA, Awad EA, Kottke FJ. Interstitial myofibrositis: Serum and muscle enzymes and lactate dehydrogenase-isoenzymes. Arch Phys Med Rehabil 1974: **35**: 23–28.
48. Ilbuldu E, Cakmak A, Disci R, Aydin R. Comparison of laser, dry needling, and placebo laser treatments in myofascial pain syndrome. Photomed Laser Surg 2004: **22**: 306–311.
49. Jankovic J, Schwartz K. Botulinum toxin injections for cervical dystonia. Neurology 1990: **40**: 277–280.
50. Kamanli A, Kaya A, Ardicoglu O, Ozgocmen S, Zengin FO, Bayik Y. Comparison of lidocaine injection, botulinum toxin injection, and dry needling to trigger points in myofascial pain syndrome. Rheumatol Int 2005: **25**: 604–611.
51. Kapel L, Glaros AG, McGlynn FD. Psychophysiological responses to stress in patients with myofascial pain-dysfunction syndrome. J Behav Med 1989: **12**: 397–406.
52. Kitagawa Y, Enomoto S, Nakamura Y, Hashimoto K. Asymmetry in jaw-jerk reflex latency in craniomandibular dysfunction patients with unilateral masseter pain. J Oral Rehabil 2000: **27**: 902–910.
53. Kitami K. Interrelation between physical disease and chronic pain–importance of understanding myofascial pain syndrome [in Japanese/Abstract]. Nippon Rinsho 2001: **59**: 1768–1772.
54. Kuan TS, Chen JT, Chen SM, Chien CH, Hong CZ. Effect of botulinum toxin on endplate noise in myofascial trigger spots of rabbit skeletal muscle. Am J Phys Med Rehabil 2002: **81**: 512–520 quiz 521-3.
55. Lang AM. A preliminary comparison of the efficacy and tolerability of botulinum toxin serotypes A and B in the treatment of myofascial pain syndrome: a retrospective, open-label chart review. Clin Ther 2003: **25**: 2268–2278.
56. Lester HA, Karshin A. Gain of function mutants: ion channels and G protein-coupled receptors. Annu Rev Neurosci 2000: **23**: 89–125.
57. Lewis J, Tehan P. A blinded pilot study investigating the use of diagnostic ultrasound for detecting active myofascial trigger points. Pain 1999: **79**: 39–44.
58. Losavio A, Muchnik S. Spontaneous acetylcholine release in mammalian neuromuscular junctions. Am J Physiol 1997: **273**: C1835–C1841.
59. Lund N, Bengtsson A, Thorborg P. Muscle tissue oxygen pressure in primary fibromyalgia. Scand J Rheumatol 1986: **15**: 165–173.
60. Majlesi J, Unalan H. High-power pain threshold ultrasound technique in the treatment of active myofascial trigger points: a randomized, double-blind, case-control study. Arch Phys Med Rehabil 2004: **85**: 833–836.
61. Malanga GA, Gwynn MW, Smith R, Miller D. Tizanidine is effective in the treatment of myofascial pain syndrome. Pain Physician 2002: **5**: 422–432.
62. Manfredini D, Romagnoli M, Cantini E, Bosco M. Efficacy of tizanidine hydrochloride in the treatment of myofascial face pain. Minerva Med 2004: **95**: 165–171.
63. McPartland JM. Travell trigger points – molecular and osteopathic perspectives. J Am Osteopath Assoc 2004: **104**: 244–249.
64. Mense S. Considerations concerning the neurobiological basis of muscle pain. Can J Physiol Pharmacol 1991: **69**: 610–616.
65. Mense S, Simons DG. Muscle Pain: Understanding its Nature, Diagnosis, and Treatment. Philadelphia: Lippincott Williams & Wilkins 2001.
66. Miehlke K, Schulz G, Eger W. Clinical and experimental studies on the fibrositis syndrome. Z Rheumaforsch 19:310–330, 1960. [Quoted in: Fricton JR. Myofascial pain syndrome. Neurol Clin 7:413-27, 1989].
67. Mills KR, Newham DJ, Edwards RHT. Muscle pain. In: Wall PD, Melzack R, eds: Textbook of Pain 2nd edn. Edinburgh: Churchill Livingstone 1989: 420–432.
68. Moldofsky H, Scarisbrick P, England R, Smythe H. Myoskeletal symptoms and non-REM sleep disturbance in patients with ''fibrositis syndrome'' and healthy subjects. Psychosom Med 1975: **37**: 341–351.
69. Muller W, Fiebich BL, Stratz T. New treatment options using 5-HT3 receptor antagonists in rheumatic diseases. Curr Top Med Chem 2006: **6**: 2035–2042.
70. Muller W, Stratz T. Local treatment of tendinopathies and myofascial pain syndromes with the 5-HT3 receptor antagonist tropisetron. Scand J Rheumatol Suppl (119):2004: 44–48.
71. Nadler SF, Feinberg JH, Reisman S, Stitik TP, DePrince ML, Hengehold D, Weingand K. Effect of topical heat on electromyographic power density spectrum in subjects with myofascial pain and normal controls: a pilot study. Am J Phys Med Rehabil 2001: **80**: 809–815.
72. Ohno K, Engel AG. Congenital myasthenic syndromes: genetic defects of the neuromuscular junction. Curr Neurol Neurosci Rep 2002: **2**: 78–88.
73. Ojala T, Arokoski JP, Partanen J. The effect of small doses of botulinum toxin a on neck-shoulder myofascial pain syndrome: a double-blind, randomized, and controlled crossover trial. Clin J Pain 2006: **22**: 90–96.
74. Orlando B, Manfredini D, Bosco M. Efficacy of physical therapy in the treatment of masticatory myofascial pain: a literature review. Minerva Stomatol 2006: **55**: 355–366.
75. Pearce JM. Myofascial pain, fibromyalgia or fibrositis? Eur Neurol 2004: **52**: 67–72.
76. Pellegrino MJ, Waylonis GW, Sommer A. Familiar occurrence of primary

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fibromyalgia. Arch Phys Med Rehabil 1989: **70**: 61–63.
77. Pettengill CA, Reisner-Keller L. The use of tricyclic antidepressants for the control of chronic orofacial pain. Cranio 1997: **15**: 53–56.
78. Pogrel MA, McNeill C, Kim JM. The assessment of trapezius muscle symptoms of patients with temporomandibular disorders by the use of liquid crystal thermography. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996: **82**: 145–151.
79. Qerama E, Fuglsang-Frederiksen A, Kasch H, Bach FW, Jensen TS. A double-blind, controlled study of botulinum toxin A in chronic myofascial pain. Neurology 2006: **67**: 241–245.
80. Rivner MH. The neurophysiology of myofascial pain syndrome. Curr Pain Headache Rep 2001: **5**: 432–440.
81. Rosales RL, Arimura K, Takenaga S, Osame M. Extrafusal and intrafusal muscle effects in experimental botulinum toxin-A injection. Muscle Nerve 1996: **19**: 488–496.
82. Saxen MA. Myofascial pain syndrome: characteristics, diagnosis, and treatment. J Indiana Dent Assoc 1998: **77**: 9–12.
83. Seltzer S, Dewart D, Pollack RL, Jackson E. The effects of dietary tryptophan on chronic maxillofacial pain and experimental pain tolerance. J Psychiatr Res 1982-83: 17: 181–6.
84. Simons DG. Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. J Electromyogr Kinesiol 2004: **14**: 95–107.
85. Simons DG, Hong CZ, Simons LS. Endplate potentials are common to midfiber myofacial trigger points. Am J Phys Med Rehabil 2002: **81**: 212–222.
86. Simons DG, Mense S. Diagnosis and therapy of myofascial trigger points [in German]. Schmerz 2003: **17**: 419–424.
87. Simons DG, Travell JG, Simons LS. 2nd edn. Travell and Simons' Myofascial Pain and Dysfunction The Trigger Point Manual Upper Half of Body, 1. Baltimore: Williams & Wilkins 1999.
88. Smania N, Corato E, Fiaschi A, Pietropoli P, Aglioti SM, Tinazzi M. Repetitive magnetic stimulation: a novel therapeutic approach for myofascial pain syndrome. J Neurol 2005: **252**: 307–314.
89. Smith H, Elliot J. Alpha2 receptors and agonists in pain management. Curr Opin Anaesthesiol 2001: **14**: 513–518.
90. Smith P, Mosscrop D, Davies S, Sloan P, Al-Ani Z. The efficacy of acupuncture in the treatment of temporomandibular joint myofascial pain: a randomised controlled trial. J Dent 2007: **35**: 259–267.
91. Stratz T, Muller W. Local treatment of rheumatic diseases with the 5-HT3 receptor antagonist tropisetron [in German]. Schmerz 2003: **17**: 200–203.
92. Swerdlow B, Dieter JN. An evaluation of the sensitivity and specificity of medical thermography for the documentation of myofascial trigger points. Pain 1992: **48**: 205–213.
93. Thant ZS, Tan EK. Emerging therapeutic applications of botulinum toxin. Med Sci Monit 2003: **9**: RA40–RA48.
94. Thornhill MH. Masticatory muscle symptoms in a patient with McArdle's disease. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996: **81**: 544–546.
95. Travell JG, Simons DG. Myofascial Pain and Dysfunction The Trigger Point Manual The Upper Extremities, 1. Baltimore: Williams & Wilkins 1983.
96. Tschopp KP, Gysin C. Local injection therapy in 107 patients with myofascial pain syndrome of the head and neck. ORL J Otorhinolaryngol Relat Spec 1996: **58**: 306–310.
97. Velly AM, Gornitsky M, Philippe P. Contributing factors to chronic myofascial pain: a case-control study. Pain 2003: **104**: 491–499.
98. Wheeler AH. Myofascial pain disorders: theory to therapy. Drugs 2004: **64**: 45–62.
99. Wheeler AH, Goolkasian P. Open label assessment of botulinum toxin A for pain treatment in a private outpatient setting. J Musculoskeletal Pain 2001: **9**: 67–82.
100. Wheeler AH, Goolkasian P, Gretz SS. Botulinum toxin A for the treatment of chronic neck pain. Pain 2001: **94**: 255–260.
101. Wiegand H, Wellhoner HH. The action of botulinum A neurotoxin on the inhibition by antidromic stimulation of the lumbar monosynaptic reflex [Abstract]. Naunyn Schmiedebergs Arch Pharmacol 1977: **298**: 235–238.
102. Wigers SH, Finset A. Rehabilitation of chronic myofascial pain disorders [in Norwegian/Abstract]. Tidsskr Nor Laegeforen 2007: **127**: 604–608.
103. Wissel J, Muller J, Dressnandt J, Heinen F, Naumann M, Topka H, Poewe W. Management of spasticity associated pain with botulinum toxin A. J Pain Symptom Manage 2000: **20**: 44–49.
104. Wolfe F, Simons DG, Fricton J, Bennett RM, Goldenberg DL, Gerwin R, Hathaway D, McCain GA, Russell IJ, Sanders HO et al. The fibromyalgia and myofascial pain syndromes: a preliminary study of tender points and trigger points in persons with fibromyalgia, myofascial pain syndrome and no disease. J Rheumatol 1992: **19**: 944–951.
105. Yap EC. Myofascial pain–an overview. Ann Acad Med Singapore 2007: **36**: 43–48.
106. Yunus MB, Kalyan-Raman U, Kalyan-Raman K, Masi AT. Pathologic changes in muscle in primary fibromyalgia syndrome. Am J Med 1986: **81**: 38–42.

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