Myofascial Pain Syndrome

Myofascial pain syndrome (MPS) is a disorder characterized by the presence of myofascial trigger points (MTrPs), distinct sensitive spots in a palpable taut band of skeletal muscle fibers1,2 that produce local and referred pain.

From: Botulinum Toxin, 2009

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Treatment of Oromandibular Dystonia, Bruxism, and Temporomandibular Disorders with Botulinum Toxin

Nwanmegha Young, Andrew Blitzer, in Botulinum Toxin, 2009

Myofascial Pain

Myofascial pain syndrome (MPS) is one of the common causes of TMD.[50-52] It manifests as discomfort or pain in the muscles that control jaw function and neck and shoulder muscles. It does not have a uniformly accepted definition or a well-understood pathology.[53-56] It is estimated that 14% of the US population suffers from chronic musculoskeletal pain and that 21% to 93% of these patients have MPS.[57-61] The clinical hallmark of MPS is the "trigger point," a region of focal tenderness in a taut band of muscle fibers that, on compression, produces referred pain in characteristic areas for specific muscles. Conventional treatments emphasize muscle relaxation using physical and pharmacologic therapies.[62,63] Previous studies demonstrated that a reduction in bite strength, concomitant with BoNT, resulted in pain relief.[64,65] In fact, the pain relief outlasts the weakening of the muscles

treated. BoNT therapy can alleviate pain of myofascial origin and, indirectly, pain of arthrogenic origin from diminished joint loading.

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PHYSICAL MEDICINE APPROACHES TO PAIN MANAGEMENT

Steven Stanos, ... Allison Baum, in Current Therapy in Pain, 2009

Myofascial Pain Syndrome

Myofascial pain syndrome is distinguished from other chronic pain syndromes by localized muscle tenderness, referred pain patterns, trigger points, local twitch response, pain in a taut band of muscle, withdrawal to pain when pressure is applied to a myofascial trigger point, and restriction of motion. It can occur in any muscle, asymmetrically, and may be due to one or all of these factors: acute tissue trauma, muscle deconditioning, sensitized nerve foci, postural abnormalities, and repetitive microtrauma. The main locations for myofascial trigger points include postural muscles in the neck, shoulders, pelvic girdle and the upper trapezius, scalene, levator scapulae, quadratus lumborum, and the lumbosacral muscles. Myofascial pain syndrome can be aggravated by multiple causes: acute tissue trauma, repetitive microtrauma, muscle deconditioning, postural abnormalities (in the workplace, home, or during recreational activities), poor sleeping habits, and metabolic issues (including vitamin deficiencies and hypothyroidism).

During an evaluation focusing on myofascial pain syndrome, the physiatrist as well as the therapist will focus on posture, body mechanics, dynamic joint function, and location and assessment of myofascial trigger points. When assessing trigger points, the clinician palpates for a rigid, fibrous nodule that is associated with the symptoms mentioned previously. At times, instead of a nodule, the clinician will palpate a defined hypersensitive collection of muscle associated with these symptoms.[76] The pain associated with the trigger point will usually travel in a proximal to distal pattern. Localized muscle groups may refer pain in distinct patterns. Common areas of presentation include the cervical and lumbar paraspinals, trapezius, gluteus medius and maximus, and piriformis muscle groups (Table 73–7).

Table 73–7. Common Referral Patterns for Myofascial Pain Syndrome

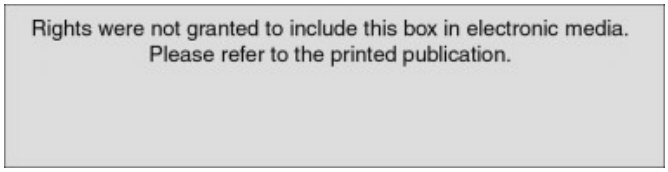
Muscle Group	Referral Pattern
Cervical paraspinals	Occiput, vertex, temple, masseter, frontal region
Trapezius	Shoulder, arm, hand

Lumbar paraspinals	Buttock, leg, calf, foot
Thoracic paraspinals	Trunk, shoulder, arm, hand
Gluteus medius and maximus	Buttock, leg
Piriformis	Buttock, leg, calf, foot

Adapted from Simons DG, Travell JG, Simons LS (eds): Travell and Simons Myofascial Pain and Dysfunction, The Trigger Point Manual, Vol.1, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 1999.

Active physical therapy and passive modalities may be combined as part of a comprehensive treatment plan. Physical therapy focuses on improving posture and body mechanics with functional tasks that may be contributing to pain and dysfunction. Therapy is focused on improving function of postural slow-twitch and phasic fast-twitch peripherally located muscles. Postural (type I) muscles are slow-twitch fibers with relatively low stores of glycogen and high myoglobulin and mitochondria, and they characteristically fatigue slowly. Under long-term stress, these muscles shorten, tighten, and demonstrate reasonable endurance. Phasic (type II) or fast-twitch muscles contain relatively high stores of glycogen and low myoglobulin and, under long-term stress, are prone to weaken.

Passive modalities may be provided by the therapist as a means of decreasing pain and facilitating therapy. Modalities targeted at deactivating symptomatic trigger points include massage, ultrasound, acupressure, spray and stretch therapy, and TENS (Box 73–11).[76] (See the section on "Passive Physical Modalities," earlier.)



Box 73–11. TRIGGER POINT DEACTIVATIONRights were not granted to include this box in electronic media. Please refer to the printed book.

From Alvarez PJ, Rockwell PG: Trigger point diagnosis and management. Am Fam Physician 2002;65:653-660.

Spray-and-stretch therapy is a technique performed by the therapist in which a passive stretch to the affected muscle is applied while concurrently spraying a local soft tissue coolant. This coolant spray contains dichlorodifuoromethane-trichloromonofluoromethane or ethyl chloride with the ability to decrease the temperature of the skin and provide an analgesic effect by blocking the spinal stretch reflex and central sensation of pain. With acupressure, clinicians apply gentle manual pressure over areas based on meridian and acupuncture pressure points.[77]

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Clinical Application of Botulinum Neurotoxin in the Treatment of Myofascial Pain Syndromes

Martin K. Childers, in Botulinum Toxin, 2009

INTRODUCTION

Myofascial pain syndrome (MPS) is a disorder characterized by the presence of myofascial trigger points (MTrPs), distinct sensitive spots in a palpable taut band of skeletal muscle fibers[1,2] that produce local and referred pain. Synonyms for MPS include myogelosis,[3,4] fibrositis,[5-7] and fibromyalgia.[7-12] MPS is characterized by both a motor abnormality (a taut or hard band within the muscle) and also by a sensory abnormality (tenderness and referred pain).[13] In addition to pain, the disorder is accompanied by referred autonomic phenomena as well as anxiety and depression.[14] The pathophysiology of MPS is not clearly understood due, in part, to the scarcity of reliable valid studies.[15] Moreover, concomitant disorders, and frequent behavioral and psychosocial contributing factors[16] in patients with MPS contribute to the complexity of human studies. Symptoms of MPS are generally associated with physical activities thought to contribute to "muscle overload," either acutely by sudden overload, or gradually with prolonged repetitive activity.[17] MPS can be classified as regional or generalized. Some authors broaden the definition of myofascial pain to include a regional pain syndrome of any soft-tissue origin. Thus, MPS may be considered either a primary disorder causing local or regional pain syndromes, or a secondary disorder that occurs as a consequence of some other condition.[1,13,16,18-20]

> Read full chapter

Acupuncture for Pain Control

Dietrich Graf Von Schweinitz, in Robinson's Current Therapy in Equine Medicine (Seventh Edition), 2015

Myofascial Pain Syndrome

Myofascial pain syndrome (MPS) is commonly seen at human pain referral centers, and voluminous medical textbooks and literature exist on the subject, most famously *Myofascial Pain and Dysfunction: The Trigger Point Manual*, volumes 1 and 2. In studies of chronic nontraumatic unilateral shoulder pain in humans, the pathophysiology of which is poorly understood, there is little supportive evidence for current treatment protocols, and the presence of MTPs is rarely mentioned. However, in an observational study of 72 cases evaluated by appropriate palpation, active MTPs were found in all subjects, mainly in the infraspinatus and trapezius muscles. Likewise, the author has found a high incidence of MPS in horses, with MTPs in the brachiocephalicus and other muscles associated with the shoulder. These animals have reduced stride length and stiffness, and when investigated by needle electromyography, show evidence of the presence of MTPs (Figure 17-4), as has been documented in humans and rabbits with myofascial pain. If one undertakes the required palpation efforts, MPS is a common finding and has varying associated performance consequences depending on the discipline in which the horse is used. The palpation skills required for detecting MTPs and the understanding of their significance are not generally understood, so affected horses are often neither diagnosed nor appropriately treated—direct dry needling being a recommended treatment. Muscles affected with MPS shorten and resist stretch, reducing the range of motion and inducing stiffness. When these horses also demonstrate frank lameness, a nerve block may abolish the latter but not the stiffness and shortened movement. Medicating an affected joint and NSAIDs may also resolve the frank lameness, but the stiffness persists unless the MTPs are appropriately treated.

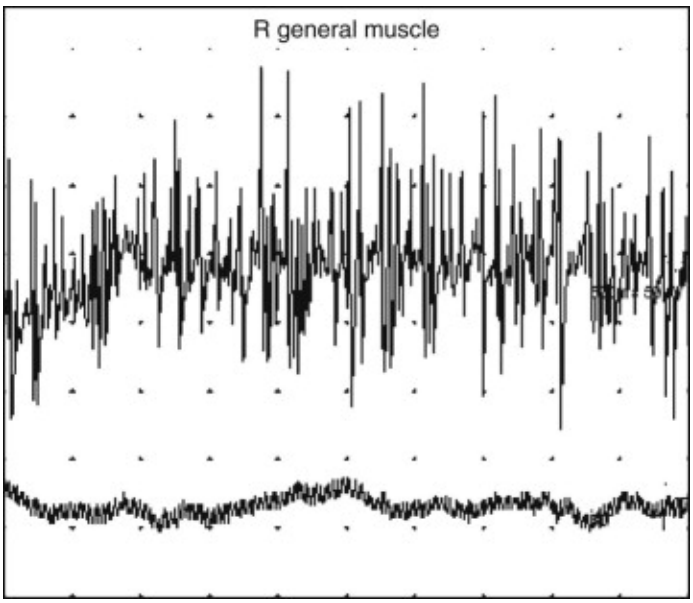

Figure 17-4. Typical electromyographic (EMG) recordings of abnormal end-plate noise or spontaneous electrical activity and spike activity from a myofascial trigger point *(upper trace)* and a nontrigger point *(lower trace)* in the brachiocephalic muscle in a horse with signs of MPS.

(From Macgregor J, Graf von Schweinitz D. Needle electromyographic activity of myofascial trigger points and control sites in equine cleidobrachialis muscle: an observational study. *Acupunct Med* 2006;24[2]:61-70, with permission from *Acupuncture in Medicine*.)

The pathophysiology of MPS and MTPs is not understood, but it is not associated with inflammation, hence the use of the term *syndrome* rather than *disease*. Often, MTPs exist in the absence of overt lameness, and even the most thorough conventional investigation fails to discover the primary factor contributing to the stiff h orse that is performing poorly. Findings on thermography, scintigraphy, and other imaging modalities are unremarkable or even misleading. The current hypothesis is that MPS represents abnormal muscle physiology involving local dysfunctional motor end plates and integrative spinal cord mechanisms that lead to central sensitization and pain windup.

Fibromyalgia is an extreme form of diffuse pain in humans that shares some pathophysiology with MPS. Although some horses have signs similar to those seen in human fibromyalgia, their constant pain and associated uncooperative behavior make these animals unlikely to be in competition. Fibrositis is a misnomer for f-ibromyalgia because there is no inflammatory process. Rather, there is severe central pain sensitization with widespread hyperesthesia and allodynia. Fibromyalgia has been suggested to occur in horses with diffuse back pain. In humans, fibromyalgia is preponderant among women with high levels of anxiety, depression, and sleep disturbance. Acupuncture in humans with fibromyalgia is often not tolerated and may further increase pain, whereas acupuncture generally is well tolerated and improves signs in those with MPS. Despite a significant prevalence in human pain syndromes, MPS and MTPs have not yet been featured in mainstream equine veterinary publications. Information can, however, be found in publications by veterinarians involved in complementary medicine (see Suggested Readings).

> Read full chapter

BOTULINUM TOXINS FOR tHE TREATMENT OF PAIN

Catalina Apostol, ... Mark Wallace, in Current Therapy in Pain, 2009

Myofascial Pain Syndrome

Myofascial pain syndrome (MPS) presents as acute or chronic skeletal muscle pain that originates in specific trigger points and affects surrounding soft tissue and

fascia. Although the etiology of MPS is still unclear, muscle spasm resulting from increased release of Ach at dysfunctional end motor plates may play a fundamental role. Contracted fibers cannot relax, leading to pain, stiffness, and fatigue. Symptoms are reported by up to 21% of patients presenting to orthopedic clinics and 85% to 93% of patients seen in pain-management centers.[127]

The high incidence of MPS and the growing body of evidence linking this disorder to excessive muscle spasm have made BTX a promising treatment (Table 67–1). BTX allows taut muscle bands to regress to a latent, asymptomatic phase by inhibiting Ach release at motor nerve endings. This has led to FDA approval of BTX-A in 1989 for conditions involving abnormal muscle contraction such as essential blepharospasm and hemifacial spasm. Its use in MPSs, however, remains off label, and currently, no definitive studies have established the efficacy of BTX for the treatment of MPS or fibromyalgia.[128]

Table 67–1. Medline Search for "Myofascial Pain" and "BTX" Spanning 1966–2006

Studies That Show No Significant Pain Improvement after BTX Treatment

Reference	Study	*N*	Muscles Affected	Outcome Measures	Doses BTX-A	Results
Querama et al[135]	Double-blind, placebo-controlled, parallel	30	Infraspinatus	EMGPressure algometry-Flexibility tests	50 U vs. saline control	BTX-A decreases motor endplate activity and influences EMG pattern but does not change pain level
Tuula et al[136]	Double-blind, randomized, controlled	31	Neck, shoulder	Pressure point threshold (dolorimeter) questionnaires	15–35 U	No difference between small BTX doses and saline injections
Grabowski et al[137]	Randomized, double-blind	17	Neck, shoulder, hip, back	Likert format VAS questionnaires (cost estimations)	25 U vs. 0.5% bupivacaine	*P* =0.3BTX VAS 2.705 ± 3.31Bupivacaine 0.5% 2 ± 2.03
Ferrante et al[138]	Randomized, double-blind, placebo-controlled	132	Cervical/shoulder	VASPressure algometry-Need for rescue	10, 25, 50 U BTX-A	Improved pain with placebo and BTX but no increased benefit from BTX vs. saline

				medications		

Studies That Show a Decrease in Myofascial Pain after BTX Treatment

Reference	Study	*N*	Muscles Affected	Outcome Measures	Doses BTX	Results
Lew et al[139]	Randomized, double-blind, placebo-controlled, single-center, prospective	29	Neck and upper back	VAS for painNDI-SF-36	BTX-A (50 U) per site (not exceeding 200 U per treatment and 100 U per side) vs. saline	Minimally positive study improved SF-36 bodily pain scale at 2 and 4 mo and mental health scale at 1 mo
De Andres et al[140]	Open-label, interventional, prospective	77	Various muscles	VASEMG-Oswestry Questionnaire	10–20 U BTX-A vs. 1 ml 0.5% lidocaine vs. control: dry needling	Baseline VAS of 8.1 improved to 6.47 at 15 days after BTX;VAS at 30 days: 5.84VAS at 90 days: 5.97
Lang[141]	Retrospective, open-label, single-center, chart review, comparing BTX-A vs. BTX-B	91	Levator muscle, splenius capitis, semispinalis capitis, piriformis	VASPatient global assessment	VAS BTX-A (100–600 U)VAS BTX-B (9000 U)	BTX-A VAS down by 2.7 (better pain relief than BTX-B and longer pain relief)-BTX-B VAS down by 1.8

Argoff[142]	Observational study of CRPS patients who also have myofascial pain	11	Sternocleidomastoid, trapezius, splenius capitis, levator scapular, etc.	Questionnaire	25–50 U	Improved burning and dysesthesiaNormalization of skin color
Foster et al-[143]	Randomized, double-blind	28	Paravertebral	VASOswestry Low Back Questionnaire	40 U	Pain relief at 3 and 8 wk
Porta[144]	Single-center, randomized	40	Iliopsoas-Piriformis-Scalenus anterior	VAS	BTX-A 80-150 U + bupivacaine 0.5% vs. methylprednisolone 80 mg + 0.5% bupivacaine	Results at 30 days: nonsignificant at 60 days: VAS decreased by 5.5 for BTX vs. 2.5 for steroid
Wheeler et al[145]	Randomized, double-blind, prospective	33	Cervicothoracic paraspinal muscles		BTX-A 50 U vs. 100 U vs. saline	All groups with pain relief but no difference among BTX and saline (**UNLESS** a second 100-U injection is done, then improvement seen with BTX)
Cheshire et al[146]	Randomized, double-blind, placebo-controlled, cross-over	6	Cervical paraspinal muscles	VASVerbal descriptors (Gracely)-Pressure algometry	50 U BTX-A	>30% improvement with BTX-A but not with saline

BTX, botulinum toxin; CRPS, complex regional pain syndrome; EMG, electromyographic; *N*, number of patients; NDI, Neck Disability Index; SF-36, Short Form 36-item questionnaire (Medical Outcomes Study); VAS, visual analog scale.

Evaluation

Unlike the diagnosis of fibromyalgia, an international diagnostic criteria for MPS does not exist. However, identification of trigger points is an important first step. Trigger points are found by gentle palpation in the direction of the muscle fibers. They have a characteristic nodularity, and palpation is extremely painful. Clinical reliability in identifying trigger point varies considerably unless examiners are consistently trained. In a study by Sciotti and coworkers,[129] four blinded examiners who were trained together were able to agree up to 80% of the time on the location of trigger points in the upper trapezius muscle.

Management

Adjunct Therapies

Traditional therapies for MPS include, but are not limited to, pharamacotherapy and injection therapies. Nonsteroidal anti-inflammatory drugs, steroids antidepressants, □-adrenergic agonists, vasodilators, skeletal muscle relaxants, and opioids in conjunction with massage, physical therapy, and transcutaneous electrical nerve stimulation have been employed with some success. Injection therapy with local anesthetics, with or without steroids, or simply dry needling has been shown to be beneficial. BTX has only recently been introduced as a possible treatment modality for MPS.

INJECTION PROTOCOLS

Therapy with BTX is individualized according to specific patient indications. There is variation between injection techniques, dosing, and number of injections. Ultimately, the technique should be adapted to the patient's specific situation. Injections into deep compartments of the back generally require the use of special imaging techniques and/or electromyographic guidance, whereas superficial injections can be undertaken without monitoring (Figs. 67–2 to 67–4).

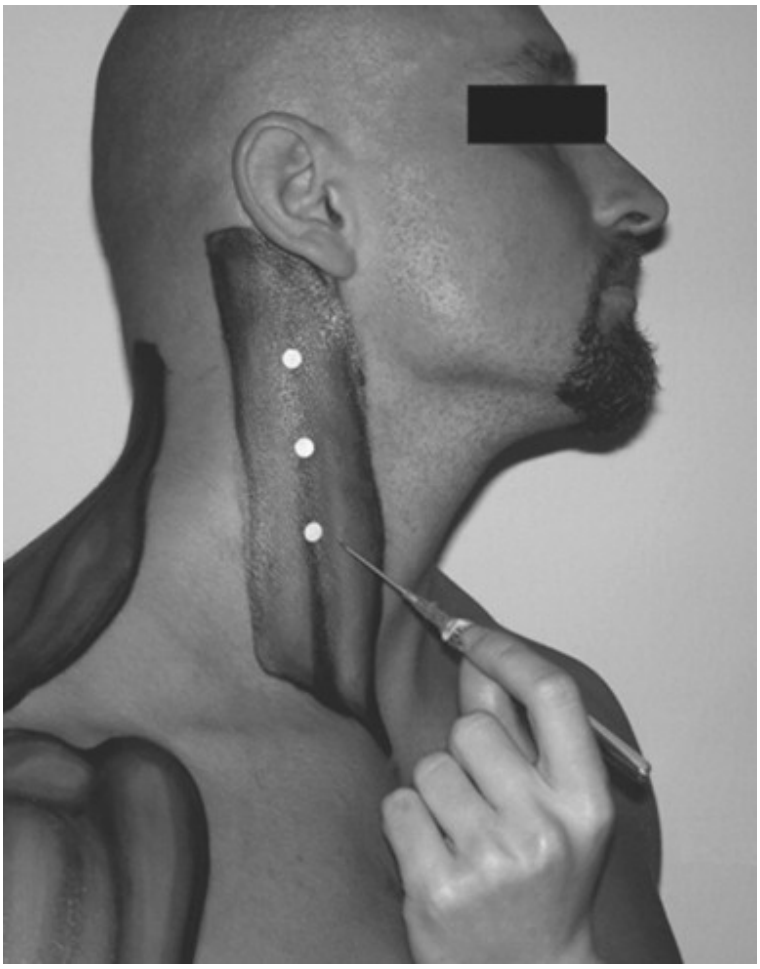

Figure 67–2. **Sternocleidomastoid muscle injection.** Muscle function: contralateral neck torsion, anterior flexion. Recommended botulinum toxin (BTX) dose: 50 mouse units (MU).

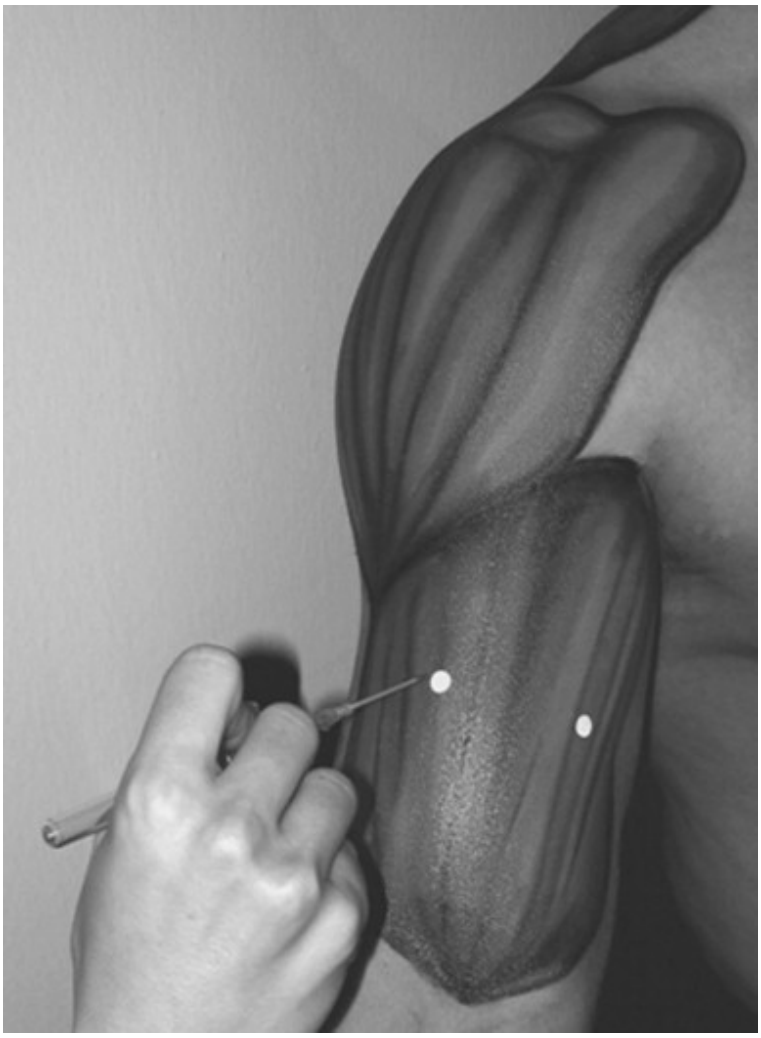

Figure 67–3. **Biceps muscle injection.** Muscle function: primary function is to move the forearm toward the shoulder (elbow flexion). The secondary function is supination of the forearm (turning the hand from a palms-down to a palms-up position). Recommended BTX dose: 100 MU in 2 ml saline injected at two sites.

Figure 67–4. **Trapezius muscle injection.** Muscle function: scapular elevation (shrugging up), scapular adduction (drawing the shoulder blades together) and scapular depression (pulling the shoulder blades down). Recommended BTX dose: 10–15 MU/trigger point (50 MU total).

EMG

EMG or motor point stimulation is useful for monitoring the injection of limb muscles, such as the small forearm muscles. EMG is a useful guide in identifying the anatomy of the muscles to be injected. It is based on the observation that trigger points are found in the proximity of motor endplates. Motor endplates occur predictably in bands throughout the muscle where small motor nerves terminate. EMG is typically connected to a needle electrode that is used to search for the characteristic noise of the motor endplate. Upon finding the motor endplate, a low-voltage (10–40 μV) increase in the baseline will occur, and the EMG device produces a sound similar to that heard in a seashell held up to one's ear. There are irregularly firing spikes, and the patient experiences a deep pain over these points.

Motor endplates coincide with motor points, the areas at which motor nerves terminate in the muscles and where phenol and alcohol blocks typically take place. If a motor endplate cannot be localized with EMG, electrical stimulation can be used to

find motor points and injection can be undertaken because the two usually coincide. Motor points may be localized with peripheral nerve stimulators whose electrodes are connected over the muscle belly and when 5 to 10 mA and 0.5 sec duration are applied.[130]

Dosage

The severity and chronicity of disease, number of muscles involved, previous response, and coexisting conditions affect the dosing. Optimally, BTX is used in the least amount needed to achieve muscle relaxation and improve range of motion without causing weakness or other side effects. Administration is proportionate to body mass. Potency is expressed in mouse units (MU) that were determined according to a standard Swiss-Webster mouse of 20 g. Generally, 1 MU is the median lethal dose (LD_{50}) that has been determined across several animal species. No specific studies were done in humans. Human LD_{50} is approximately 3000 MU for a 70-kg adult. Large muscle groups receive anywhere between 60 and 400 MU per treatment, although the recommended ceiling dose is closer to 360 MU given 12 weeks apart.[131]

Preparations

BOTOX is dispensed in 100U vials, whereas Dysport contains 500 U. The potency of the two forms differs with a 1:4 conversion rate for BOTOX to Dysport. Most practitioners dilute BOTOX with 1 to 4 ml of preservative-free saline for a concentration of 2.5 to 10 U/0.1 ml. The preparation is best used within 4 hours of reconstitution. The pH should be maintained from 4.2 to 6.8, and the temperature kept at less than 20°C. BTX-B is available in vials of various volumes, each at a concentration of 5000 U/ml. It is recommended that this be stored at between 2°C and 8°C.

Complications and Side Effects

Owing to its specific mechanism of action, specific side effects are uncommon and systemic effects are even less common. Short-lived flulike symptoms of soreness, headache, fever, chills, lightheadedness, hypertension, diarrhea, and abdominal pain have been reported. Muscular weakness, however, remains the predominant side effect that patients should be made aware of prior to BTX injections. Clinicians should have a clear understanding of the functional consequence of injecting particular muscles when these muscles serve crucial functions such as swallowing (Table 67–2).

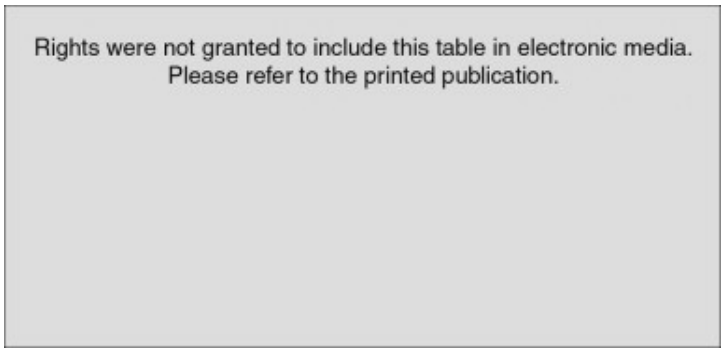

Table 67–2. Exercise Caution When Injecting These MusclesRights were not granted to include this table in electronic media. Please refer to the printed book.

From Childers MK, Wilson JW, Simison D. Equipment and injection techniques. In Childers MK, Wilson JW, Simison D (eds): Use of Botulinum Toxin Type A in Pain Management. New York: Demos Medical Publishing, 1999; pp 64–92.

Recently, some reports of adverse events including death have been linked to the use of BTX in children, leading to a warning by the FDA.[132] According to the FDA, adverse events suggesting botulism and felt to be potentially associated with distant spread generally occurred at relatively high dosages of 100 to 700 U in BTX-A recipients and 10,000 to 20,000 U in BTX-B recipients.[132] Botulism cases in children younger than 16 years, who were treated for limb muscle spasticity associated with cerebral palsy, were associated with adverse events including dysphagia, respiratory insufficiency requiring use of gastric feeding tubes and ventilatory support, hospitalization, and death. Doses in these cases ranged from 6.25 to 32 U/kg for BTX-A (some >□ 20 U/kg max) and from 388 to 625 U/kg for BTX-B. Public Citizen, a consumer advocacy group, stated that its investigation of the FDA's adverse event database indicates that 16 BTX recipients (the majority receiving BTX-B), including 4 children, died after being injected with the products; although the FDA has not determined whether these deaths were caused by the use of the products or were attributable to other causes.[132]

Absolute Contraindications

BTX-A is contraindicated in the presence of infection at the proposed site of injection or hypersensitivity to any ingredient in the BTX formulation. Hypersensitivity reactions are rare but may be accompanied by serious reactions such as anaphylaxis, soft tissue edema, and dyspnea. If a hypersensitivity reaction is suspected, BTX-A injection should be discontinued immediately and appropriate medical intervention instituted.

Relative Contraindications

Relative contraindications are administration to individuals who cannot understand the risks and benefits of BTX-A and treatment in patients with neuromuscular disorders or myopathies characterized by generalized muscle weakness. Patients with amyotrophic lateral sclerosis, myasthenia gravis, or Lambert-Eaton syndrome should receive BTX treatment only with caution. The toxin may be potentiated by aminoglycoside antibiotics, spectinomycin, or any other drug that might interfere with the neuromuscular transmission (e.g., tubocurarine, tetracyclines, lincomycin) or drugs that interfere with the intraneuronal concentrations of calcium.[133,134]

> Read full chapter

Lumbar Myofascial Pain Syndrome

Steven D. Waldman MD, JD, in Atlas of Uncommon Pain Syndromes (Third Edition), 2014

Treatment

Lumbar myofascial pain syndrome is best treated with a multimodality approach. Physical therapy, including correction of functional abnormalities (e.g., poor posture, improper chair or computer height) and the use of heat modalities and deep sedative massage, combined with nonsteroidal anti-inflammatory drugs (NSAIDs) and skeletal muscle relaxants represents a reasonable starting point. If these treatments fail to provide rapid symptomatic relief, local trigger point injection of anesthetic and steroid into the myofascial trigger point area is a reasonable next step. Underlying diffuse muscle pain and sleep disturbance and depression are best treated with a tricyclic antidepressant compound, such as nortriptyline, which can be started at a single bedtime dose of 25 mg.

When performing trigger point injections, careful preparation of the patient before injection helps optimize results. Trigger point injections are directed at the primary trigger point, rather than the area of referred pain. It should be explained to the patient that the goal of trigger point injection is to block the trigger of the persistent pain and, it is hoped, provide long-lasting relief. It is important that the patient understand that for most patients with myofascial pain syndrome, more than one treatment modality is required to provide optimal pain relief. The use of the prone or lateral position when identifying and marking trigger points and when performing the actual trigger point injection helps decrease the incidence of vasovagal reactions. The skin overlying the trigger point to be injected should always be prepared with antiseptic solution before injection to avoid infection.

After the goals of trigger point injection are explained to the patient and proper preparation of the patient has been carried out, the trigger point to be injected is reidentified by palpation with a sterile gloved finger (Figure 81-2). A syringe containing 10 mL of 0.25% preservative-free bupivacaine and 40 mg of methylprednisolone to be injected is attached to a 25-gauge needle of a length adequate to reach the trigger point. For the deeper muscles of posture in the low back, a 3½-inch needle is required. A volume of 0.5 to 1 mL of solution is injected into each trigger point. The patient should be informed that a series of two to five treatment sessions may be required to abolish the trigger point completely.

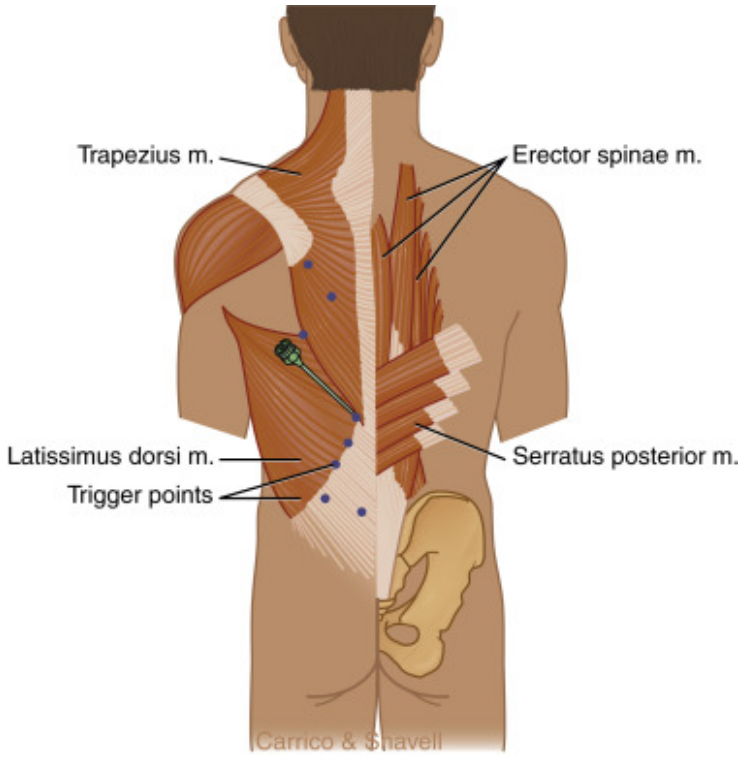

Figure 81-2. Injection technique to relieve lumbar myofascial pain.

(From Waldman SD: *Atlas of pain management injection techniques,* ed 2, Philadelphia, 2007, Saunders, p 330.)

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Headache

Rigmor Jensen, Paola Torelli, in Handbook of Clinical Neurology, 2010

Botulinum toxin

Botulinum toxin is used in the treatment of dystonia and myofascial pain syndrome. The rationale for the use of botulinum toxin in pain conditions, and especially in TTH, is the combination of muscle relaxant and antinociceptive action in the periph-

eral nervous system and the CNS (Guyer, 1999). The efficacy of botulinum toxin in TTH was first evaluated in open-label studies (Relja, 1997) and indicated positive results. Unfortunately, randomized placebo-controlled trials produced conflicting, mostly negative, results (Göbel et al., 1999; Smuts et al., 1999; Rollnik et al., 2000) and the most recent large multicenter study in CTTH was also unable to demonstrate a positive effect (Silberstein et al., 2006).

In summary, data on efficacy of botulinum toxin in the treatment of TTH are based on a limited number of studies with several methodological reservations and, as large-scale randomized controlled trials are negative, botulinum toxin cannot be recommended in the preventive therapy of CTTH.

Future drug therapies should primarily focus on prevention of the frequent ETTH and CTTH, and both third-generation antidepressants, nitric oxide inhibitors, Na_+ and/or Ca_{2+} channel modulators, and new anticonvulsants are promising candidates. Developments in animal models of chronic pain and in more specific preventive compounds are also very promising and will undoubtedly require further investigation in randomized controlled trials.

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Managing Back Pain

Kevin K. Haussler, in Robinson's Current Therapy in Equine Medicine (Seventh Edition), 2015

Chronic Soft Tissue Back Pain

Chronic soft tissue back pain may be characterized as a general myofascial pain syndrome or as discrete palpable hypertonic bands within specific muscles (e.g., the middle gluteal muscle), which may be either active (i.e., painful to deep palpation) or inactive (i.e., palpable, but not painful to deep palpation). These hypertonic bands are termed *trigger points* and are characterized as hyperirritable foci in skeletal muscle that are associated with palpable taut bands of muscle fibers. In humans, trigger point therapy includes deep ischemic compression, mechanical vibration, pulsed ultrasound, laser therapy, electrostimulation, dry needling, “spray and stretch” with a vapocoolant spray, and localized stretching techniques. Direct injection of the trigger points may be effective when more conservative therapies fail. Injectates include saline, local anesthetics, corticosteroids, and botulinum toxin. There are no published reports on the effectiveness of the above treatment approaches in affected horses, but anecdotally many of these techniques have been applied with reported clinical effectiveness.

> Read full chapter

Myalgia

Charles S. Bockman, Joan Eckerson, in xPharm: The Comprehensive Pharmacology Reference, 2007

Associated Disorders

Patients with fibromyalgia typically present with one or more comorbid conditions. The most common of these are myofascial pain syndrome, migraine headache, irritable bowel syndrome, and a history of chronic fatigue and depression. Other comorbid conditions associated with fibromyalgia include insomnia, restless leg syndrome, and temporomandibular joint syndrome Millea and Halloway (2000).

Polymyalgia rheumatica may be accompanied by giant cell arteritis, although they appear to be two separate entities that occur together for unknown reasons. Histologic findings of vasculitis are often present when the two are present at the same time. Granulomatous myocarditis and hepatitis have also been reported to be associated with polymyalgia rheumatica, which may explain the mild elevation of liver enzymes Cohen and Ginsberg (1990).

Comorbid conditions associated with chronic fatigue syndrome include migraine headache, irritable bowel syndrome, depression, and fibromyalgia. Some patients also exhibit comorbid psychiatric conditions, such as panic disorder Natelson (2001). Treatment of these comorbid disorders may significantly improve the quality of life in patients with chronic fatigue syndrome Craig and Kakumanu (2002).

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Levator Ani Pain Syndrome

Steven D. Waldman MD, JD, in Atlas of Uncommon Pain Syndromes (Third Edition), 2014

Testing

No specific test exists for levator ani pain syndrome. Testing is aimed primarily at identifying occult pathological conditions or other diseases that may mimic myofascial pain syndrome (see discussion of differential diagnosis). Plain radiographs help delineate bony abnormality of the pelvis and hip, including arthritis, avascular

necrosis of the hip, fracture, congenital abnormalities, and tumor. All patients with recent onset of myofascial pain syndrome should undergo magnetic resonance imaging (MRI) of the lumbar spine and pelvis to rule out occult pathological processes. Screening laboratory tests, consisting of complete blood count, erythrocyte sedimentation rate, antinuclear antibody testing, and automated blood chemistry testing, should be performed to rule out occult inflammatory arthritis, infection, and tumor.

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