Crossfires: Trigger Point Injections and Botulinum Injections

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Chair: Lawrence W. Frank, MD

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Objectives

Objectives - Participants will acquire skills to (1) recognize the indications for the use of botulinum toxin in the treatment of painful conditions, (2) explain the pharmacology of botulinum toxin in the amelioration of pain, (3) discuss the purported mechanisms of myofascial pain, (4) demonstrate the clinical utility of trigger point injections, and (5) distinguish the difference between trigger points and tender points.

Target Audience:
• Neurologists, physical medicine and rehabilitation and other physicians interested in neuromuscular and electrodiagnostic medicine
• Health care professionals involved in the management of patients with neuromuscular diseases
• Researchers who are actively involved in the neuromuscular and/or electrodiagnostic research

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Botulinum Toxin for Pain: Yes, It Works

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Botulinum toxin (BTX) is currently approved by the Food Drug Administration (FDA) in the United States for the treatment of several conditions that are either painful or associated with pain. These include cervical dystonia, upper limb spasticity, chronic migraines, hemifacial spasm, and blepharospasm. Pain relief is the primary treatment goal for chronic migraines, while the other indications have pain as one but not the only outcome measure that suggests treatment efficacy. In addition to pain, other outcome measures such as spasticity, range of motion, and disability were improved.

There are multiple other painful conditions that also have been treated with BTX (Table). This is not to suggest that all of these problems should be treated with BTX, nor that BTX is effective in all of these, but there are at least anecdotal reports, and sometimes well-designed trials, which suggest improvement in these painful conditions. This discussion will not attempt in any way to separate the differences between the various available BTX treatments. The collective evidence is not, in the author’s opinion, able to clearly delineate efficacy in pain management between BTX formulations.

PAIN PHYSIOLOGY

Neurotransmitters

There are several potential areas in which BTX may alter and improve pain. Its primary mechanism of action is thought to be interference with the presynaptic release of acetylcholine, but it also interferes with other neurotransmitters involved with pain, including calcitonin gene-related peptide (CGRP), substance P, and glutamate. Theoretically, anywhere that BTX is present it will alter the neurotransmitters and produce pain relief. In rat studies BTX-A decreases the local release of glutamate and decreases nociceptive behaviors after injection into the hind paw.

<table>
<thead>
<tr>
<th>Painful conditions that have been treated with Botulinum toxin</th>
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<tr>
<td>Complex regional pain syndrome</td>
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<tr>
<td>Chronic interstitial cystitis</td>
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<td>Painful bladder syndrome</td>
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<td>Radiation fibrosis syndrome</td>
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<td>Hallux valgus</td>
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<td>Post whiplash neck pain</td>
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<td>Phantom and stump pain</td>
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<tr>
<td>Chronic facial pain associated with masticatory hyperactivity</td>
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<td>Temporomandibular pain</td>
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<td>Spastic shoulder pain</td>
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<tr>
<td>Spinal cord pathology</td>
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<tr>
<td>Cervical pain associated with cervical dystonia</td>
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<td>Headache</td>
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<td>Myofascial pain</td>
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<td>Plantar fasciitis</td>
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<td>Piriformis syndrome</td>
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<td>Chronic low back pain</td>
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<td>Joint pain (knee, shoulder, wrist)</td>
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<td>Lateral epicondylitis and other tendonitis</td>
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<td>Chronic pain and pelvic floor spasm</td>
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<td>Postherpetic neuralgia</td>
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Muscle Contraction

Prolonged muscle contraction is hypothesized to cause pain from both ischemia and release of bradykinins which contributes to sensitization and excitation of nociceptors. Botulinum toxin may reduce pain by decreasing muscle fiber contraction at the neuromuscular junction or by altering and decreasing the cholinergic muscle spindle activity.
Central Nervous System Effects of Botulinum Toxin

There is no convincing evidence that BTX injected peripherally can directly affect central nervous system (CNS) structures. However, functional organization of the CNS may be affected indirectly through peripheral mechanisms. The alpha and gamma motor endings can alter muscle spindle afferent inflow, which may alter both spinal and cortical physiology. Animal studies have shown the toxin to undergo retrograde transport to neurons in the CNS. It is unclear whether these toxins have physiological activity.

NON-FOOD AND DRUG ADMINISTRATION APPROVED PAINFUL CONDITIONS THAT BOTULINUM TOXIN IS BEING USED TO TREAT

Neuropathic Pain

There are at least two randomized, double-blind placebo-controlled trials looking at BTX and postherpetic neuralgia with injections into the painful area.

Diabetic Neuropathy

In a randomized, double-blind placebo-controlled trial in patients with diabetic neuropathy, 18 subjects received BTX-A in 12 intradermal injection sites. Crossover between groups occurred at 12 weeks. There was a decrease in the visual analog scale and in 4 weeks improvement in sleep quality. Another randomized, double-blind placebo-controlled study showed some efficacy with a one-time intradermal BTX-A administration.

Chronic Facial Pain Associated With Masticatory Hyperactivity

In 90 patients with chronic pain, a randomized, blinded placebo-controlled study showed a decrease in visual analog pain in patients with masticatory hyperactivity.

Promising Effects for Other Problems

Other problems that have reports that seem promising include radiation fibrosis syndrome, painful bladder syndrome, complex regional pain syndrome, chronic arthritis, anterior knee pain, spinal cord pathology, plantar fasciitis, stump pain (but not phantom pain), and piriformis syndrome.

Early reports on new treatments are frequently overly optimistic and tend to only report positive findings. Unfortunately, the number of negative trials that never got published is unknown. Thus, these early efficacious trials need to be interpreted by clinicians with skepticism; BTX trials almost surely share in this publication bias.

Myofascial Pain

Ever since BTX was released, there have been advocates for its use for myofascial pain. This is an attractive and logical treatment given the mechanism of action of BTX in the muscle and the theories of “muscle spasm” contributing to myofascial pain and trigger points. Numerous anecdotal reports and methodically flawed trials suggest efficacy of BTX in the treatment of myofascial pain. However, prospective, randomized controlled trials looking at myofascial pain and BTX have mixed results in demonstrating efficacy. Specifically, there are several trials that show BTX is no more efficacious than placebo.

Evolution of Myofascial Pain

Muscle pain has been described since the 16th century and has been labelled a variety of terms, including fibrositis, interstitial myofibrosis, myalgia, myofascial pain, idiopathic myalgia, myofascitis, perineuritis, myodysneuria, nonarticular rheumatism, and fibromyalgia. Acute muscle pain (e.g., delayed onset muscle soreness that occurs after eccentric muscle exercise) and other acute, reversible muscle strains and muscle tears are not usually considered some of the more chronic persistent muscle pains.

When there are multiple names for the same problem in medicine it suggests ignorance of the pathophysiology and disagreement among physicians. Myofascial pain is clearly in this category. This discussion will not clarify this issue but will attempt to give some perspective. The term “fibrositis” coined by Gowers in 1904, which gradually turned into “fibromyalgia” by 1981, is a diffuse pain syndrome not limited to muscles. Most, but not all, clinicians think fibromyalgia is not synonymous with myofascial pain. There is considerable overlap in symptoms of these two conditions. The diagnosis of fibromyalgia usually involves the palpation of “tender points.” These tender points are usually, but not always, over muscle and are not the same as palpation of trigger points, although there is some overlap. For purposes of this debate, the discussion will be limited to more localized problems in the muscle and not the very diffuse fibromyalgia.

Muscle Pain, Myofascial Pain, and Trigger Points

Differentiating muscle pain, myofascial pain, and trigger points is virtually impossible. Some authors are vigorous and detailed in their description of the terminology while other authors are extremely, if not purposely, vague about what they are describing. Here is a brief (and incomplete) summary:

- **Muscle pain** is usually a simple, descriptive term to denote pain coming from muscles, usually with tenderness over the muscles.

- **Myofascial pain** is usually considered more specific and associated with complex and unproven theories about pathophysiology usually, but not always, involving myofascial trigger points.

- **Myofascial trigger points** were popularized and advocated by Travell and Simons in 1983 in the book “Myofascial Pain and Dysfunction: The Trigger Point Manual.” Trigger points are usually diagnosed by palpating perpendicular to the muscle fiber direction. Taut bands can be felt in an irritable spot in the muscle. When the trigger point is palpated there may be a local twitch response, local pain, referred pain, limitation of range of motion, some weakness of that muscle, and increased needle electromyography (EMG) activity.

- **Latent trigger point** is a term that confuses the situation. Latent trigger points are manifested by the palpatory findings of the trigger point in people with no muscle pain.
Most studies show poor inter-rater reliability and poor agreement on the identification of trigger points. The increased needle EMG activity in trigger points is unproven; and, in this author’s opinion, nonexistent in most patients with trigger points. The diagnosis of trigger points is based on history and physical examination and, thus, is a subjective diagnosis.

**Location of Myofascial Pain**

The location of myofascial pain is reported nearly anywhere there are muscles. Most trigger point charts localize trigger points in the neck and shoulder area, which includes the trapezius, levator scapulae, cervical paraspinals, rhomboids, scalenes, and sternocleidomastoid muscles. These muscles are nearly identical to many of the muscles considered hyperactive in patients with cervical dystonia.

**Increased Muscle Tone in Cervical Dystonia**

There is clearly increased needle EMG activity and muscle tone in the neck muscles required to make the diagnosis of cervical dystonia. This is a clinical diagnosis and there is no defining line between “normal” and “abnormal.” There is no objective way to define the line between normal and abnormal tone. Needle EMG activity and tone frequently changes in the same person in seconds. There is significant overlap in myofascial trigger points in myofascial pain syndrome and cervical dystonia.

**Subjective Nature of Diagnoses**

This author has not found any objective way to clearly diagnose either cervical dystonia or myofascial pain syndrome. They share common symptoms of increased muscle tone, increased neck pain, abnormal positioning of the neck, and subjective diagnostic criteria that require “expert physicians” to determine the diagnosis. There are virtually no studies clarifying the criteria and reliability of the diagnosis of cervical dystonia. Just by chance, there must be a lot of overlap. The severe cervical dystonia patient is fairly obvious and easy to diagnose. The diagnosis for the mild, subtle cervical dystonia patient is much more difficult and perhaps impossible to have confidence in making the diagnosis. Almost all the patients that this author sees who have cervical dystonia also have trigger points. The determination whether cervical dystonia is over or underdiagnosed is extremely subjective and lacks scientific evidence.

**Botulinum Toxin Improves Pain in Cervical Dystonia**

Multiple randomized controlled trials demonstrate that pain and quality of life is improved through the use of BTX in cervical dystonia. No study attempts to clarify how many of these patients had myofascial pain or if the pain relief was related to improvement in the myofascial components of their pain. This is likely an impossible study to perform, due in part to the subjective nature the diagnoses and outcomes of both of these problems. There is no reliable way to distinguish the two.

**Costs and Side Effects**

At this time BTX is still considered an expensive medication by many. An injection usually lasts about 3 months and, depending on the dosage and other issues, is frequently $600-$1,000. The injections are almost always painful. Its strengths are that its side effects are limited, it is injected locally, and it rarely has systemic effects.

**Comparison to Oral Medications**

Medications given for painful conditions, such as opiates and sedatives, have significant deleterious CNS and systemic side effects that are also costly to the patients and society. Botulinum toxin is likely less harmful than opioids for chronic pain problems, even if the oral medicines are cheaper. Nobody measures the other costs to the patient or society from addictive medications.

**Is This an Expensive Placebo?**

It is certainly possible that BTX is nothing more than an expensive placebo. Many pain clinicians will say, “so what.” If the BTX or placebo is efficacious, can increase function, decrease narcotic pain killer use, and the side effects are minimal, it may be worth it. A randomized controlled trial showed that patients given a “expensive” placebo had a better pain tolerance than patients who received a “cheap” placebo. Other studies that show that placebos that have medical rituals, use needles, and require physician skill are more effective. A BTX injection clearly meets these standards. Physicians report that patients receive relief of pain with BTX. It is a virtual certainty that there is bias from both the physician and patient. Overcoming these types of biases has not been accomplished for many treatments and not likely to occur with BTX injections. Many other questions need to be answered, in particular, “Is BTX is more cost effective than Lidocaine injections or other noninjection treatments for myofascial pain syndrome?”

**Use of BTX for painful conditions that are approved by the FDA should be less controversial and relatively proven given the current medical system. Using BTX for non-FDA approved pain problems is at this stage controversial. The question of whether this expensive treatment is cost effective is virtually impossible to adequately study, and it almost always comes down to somebody’s perspective on what “cost effective” actually means.**

**If BTX improves a patient’s function, decreases or eliminates opioids, improves work attendance, and increases other “well behaviors,” it may be considered a truly efficacious and cost effective treatment. If, on the other hand, this is an expensive treatment that is given to people who say, “I think I am better,” but continue to have dysfunctional pain behaviors, BTX should not be used for these patients with painful conditions, because it has not truly improved their quality of life and has utilized a considerable amount of resources. This treatment philosophy can be used even for patients who have conditions that are “FDA approved” for the use of BTX.**

**CONCLUSIONS**

1. There is considerable overlap between the diagnosis of myofascial pain syndrome and mild cervical dystonia. Many patients with cervical dystonia have myofascial pain. It is unclear how many of the patients diagnosed with myofascial pain also have mild, undiagnosed cervical dystonia. There is a risk of overdiagnosis of cervical dystonia to treat myofascial pain.
2. For FDA approved BTX use in diagnoses such as cervical dystonia that have pain relief as an outcome and treatment goal, BTX is helpful and not controversial.

3. For non-FDA approved BTX use in conditions such as myofascial pain, things are not so clear. If the BTX injection is associated with increased function and work and less opioids and other medications, it should be seriously considered as a treatment alternative.

4. If, after the BTX injection, the patient says the pain is better, but continues to remain disabled, and consumes opioids and/or sedatives; BTX should not be used. This poor functional outcome is strong evidence that BTX is not an efficacious treatment, even if there is some subjective pain relief.

REFERENCES


Botulinum Toxin Should Not Be Used for Trigger Point Injections

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INTRODUCTION

Botulinum toxin (BTX) types A and B are currently available for clinical use. Botulinum toxin enters the motor nerve terminal by endocytosis and lyses proteins which are integral to the exocytosis of synaptic vesicles containing acetylcholine. The uptake of BTX is mediated by the synaptic vesicle glycoprotein 2C (SV2C) receptor. Botulinum toxin A (BTX-A) cleaves the synaptosomal-associated protein 25 (SNAP-25) protein, and botulinum toxin B (BTX-B) cleaves VAMP (synaptobrevin). Both result in an inability of the synaptic vessels to fuse with the nerve terminal cell membrane, resulting in functional denervation of the neuromuscular junction.

FOOD AND DRUG ADMINISTRATION-INDICATED USES FOR BOTULINUM TOXIN A AND B

BTX-A (Botox®) is currently Food and Drug Administration (FDA) indicated for cosmetic treatments (decreasing “severe” glabellar lines [frown lines]), detrusor overactivity in neurologic conditions, chronic migraine headache, upper limb spasticity in adults, cervical dystonia in adults to improve head position and pain, severe axillary hyperhidrosis, blepharospasm, and strabismus. BTX-A (Dysport®) is FDA indicated as treatment for cervical dystonia and temporary improvement in the appearance of glabellar lines. BTX-B (Myobloc®) is FDA indicated as treatment for cervical dystonia.

Cervical dystonia manifests as abnormal contractions and movement of the cervical muscles, typically with simultaneous contraction of opposing muscles. There are three clinical rating scales commonly used: the Fahn-Marsden Rating Scale, the Unified Dystonia Rating Scale, and the Global Dystonia Scale. Genetic testing is available for some forms of primary pure dystonia. Abnormal neck or head position is required for the diagnosis of cervical dystonia. Pain in cervical dystonia is considered to be the result of both the abnormal position of the head or neck and the abnormal contractions of muscles associated with the abnormal position.

MYOFASCIAL PAIN AND TRIGGER POINTS

Trigger points are painful, tender points in the muscles associated with myofascial pain syndrome. These points are hyper-irritable, tender when compressed, and often associated with a “band” or “twitch” when compressed. Treatment of myofascial pain syndrome by modifying trigger point pain by injection, massage, heat, or laser therapy have all been reported to decrease the pain in myofascial pain syndrome. Systematic reviews of various injection techniques as a treatment for myofascial pain syndrome at trigger points have not been able to show whether needling a trigger point has any benefit outside of the mechanical action of the needle or placebo response.

CENTRAL NERVOUS SYSTEM EFFECTS OF BOTULINUM TOXIN

In the past 8+ years, a number of effects of BTX on the peripheral and central nervous system have been described. It decreases intrasynaptic (gamma) motor neuron activity, and evidence for retrograde transport to the central nervous system has been demonstrated. Matak and colleagues found evidence of retrograde transport of BTX-A in the hippocampus, visual system, and facial motor neurons after intramuscular, subcutaneous, or intraneural injection. They
also report evidence of enzymatically active BTX-A in the trigeminal sensory nucleus, and they demonstrated cleaved SNAP-25 in choline acetyltransferase positive neurons around motor neurons (possibly dendrites of the motor neurons).

Filipović and colleagues demonstrated decreased mechanical allodynia in a rat pain module, and they demonstrated decreased dural extravasation when BTX-A was injected prior to the injury. They also were able to block these effects by stopping axonal transport in trigeminal neurons with injection of colchicine into the trigeminal nerve ganglion.

In another direct demonstration of retrograde transport of enzymatically active BTX-A, Restani and colleagues all demonstrated transport of BTX-A from the optic tectum to the amacrine cells in the retina, with clear evidence of enzymatic activity (cleaving SNAP-25). They also demonstrated transport across at least two synapses (from amacrine cells to rod bipolar cells) and enlarged synaptic boutons in cells with SNAP-25 cleavage products.

In indirect study, Bach-Rojecky and colleagues demonstrated altered pain behavior in the contralateral extremity in rats (formalin injection pain model) and demonstrated similar effects of both peripheral (paw) and intrathecal injection of BTX-A.

As indirect support of BTX modifying pain centrally, a randomized, placebo-controlled human trial by Schulte-Mattler and colleagues in 2007 found that BTX injected subcutaneously was unable to block capsaicin-induced pain, and it did not alter thermal pain or electrically-induced pain thresholds at 4 weeks and 8 weeks.

**CLINICAL TRIALS FOR MYOFASCIAL PAIN**

Because BTX causes muscle weakness, it has been used from the onset to treat a variety of conditions with actual muscle overactivity (e.g., dystonia, upper motor neuron spasticity) or presumed muscle overactivity (e.g., trigger points, “spasm,” etc). Clinic evidence of beneficial effects in dystonia and upper motor neuron spasticity have been demonstrated in clinic trials leading to FDA approval for these conditions. The dystonia trials also report significant reduction in pain as an outcome in the study. Unfortunately, the outcome of other pain related studies is not as clear.

Randomized placebo controlled trials of BTX in the treatment of temporomandibular joint syndrome myofascial pain, trigger point injections, whiplash associated neck pain and myofascial pain in the neck have all found no significant difference between BTX, placebo, or lidocaine. Scott and colleagues performed a systematic review of published studies on the use of trigger point injections for chronic nonmalignant pain in 2009. Analysis of 15 studies that met criteria for review found that trigger point injections subjectively decreased symptoms when used as sole treatment. Trigger point injection was no more effective than other non-invasive treatments. BTX was not more effective than saline or lidocaine. Both Lidocaine and physiologic saline were more effective than dry needling. A recent Cochrane Review found there was inconclusive evidence to support the use of BTX in the treatment of myofascial pain syndrome.

Barwood and colleagues reported significant reductions in postoperative pain in children with cerebral palsy undergoing tendon lengthening procedures; however, Nixdorf did not find any improvement in myogenic oral facial pain in a randomized, placebo-controlled trial. Foster and colleagues found significant improvement in chronic back pain with BTX-A at 3 weeks and 8 weeks, however De Andrés and colleagues found no significant changes in back pain in a randomized trial of BTX in patients with back pain, myofascial pain, and trigger points.

**BOTULINUM TOXIN EFFECTS ON OTHER NEUROPATHIC PAIN**

Botulinum toxin has been shown to be effective for neuropathic pain. Residual neuropathic pain or phantom pain is present after amputation in up to 65% of patients. In a trial comparing lidocaine or BTX-A, Wu and colleagues have shown significant long duration pain relief (up to 6 months) after a single injection with BTX-A in and around a neuroma with doses up to 250 U. Neuropathic pain behaviors in experimental animals with diabetic neuropathy have improved significantly with intrathecal injection of BTX-A, with a 24 hour latency to onset. In human subjects with painful diabetic neuropathy, Yuan and colleagues showed significant reductions in pain after intra-cutaneous injection of Botox® on the dorsum of the foot using a placebo controlled crossover trial. Pain scores were improved by BTX-A at each time point compared to placebo. In a small randomized, three arm placebo-controlled trial, Xiao and colleagues showed significant improvement in pain scores in postherpetic neuralgia, including significant reduction of opioid use beginning at 7 days and lasting for 3 months.

Ranoux and colleagues reported in 2008 that BTX was significantly better than placebo when used for neuropathic pain. In this study, there had to be clear evidence of a focal nerve injury, with neuropathic pain confined to a geographic area consistent with the nerve injury. Botulinum toxin A was injected in a grid pattern in the painful area, with significantly improved pain and allodynia at 4 and 12 weeks after injection.

However, BTX-A was not effective in a small randomized trial for the treatment of allodynia in complex regional pain syndrome (CRPS). This trial was stopped early because eight of the first nine patients reported the BTX injections were intolerable. Of note, patient nine had improvement but had a diagnosis of postherpetic neuralgia rather than CRPS.

Interestingly, Singh and colleagues report a randomized controlled trial of intra-articular BTX-A after total knee arthroplasty with improved pain in the BTX group. The same group also reported significantly improved pain with intra-articular BTX-A for refractory shoulder pain, with 61% of patients reporting clinically meaningful pain relief at 1 month.

**CONCLUSIONS**

Botulinum toxin does not have sufficient evidence that it is effective for myofascial pain in the absence of another diagnosis. Pain associated with objective evidence of muscle overactivity (e.g., dystonia, upper motor neuron spasticity, needle electromyography [EMG] evidence) has been shown to respond to the use of BTX as a treat-
ment of the underlying disorder. Clinical trials of BTX specifically for trigger point therapy have shown that it is no more effective than saline injections or lidocaine injections.

Botulinum toxin does appear to be effective for other pain conditions, with clinical trials suggesting effectiveness for neuropathic pain, and possibly for joint pain, and is currently indicated for the treatment of chronic daily migraine.

REFERENCES

Trigger Point Injections: Is There Evidenced-Based Treatment?
Trigger Point Injections: Is There Evidenced-Based Treatment?

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INTRODUCTION

Myofascial pain syndrome has been well described since Dr. Janet Travell made it famous with the treatment of John F. Kennedy. Drs. Travell and Simons wrote the first trigger point manual in 1983. Trigger points are defined as exquisite tenderness in a nodule in a palpable taut muscle. They are able to produce pain spontaneously or on compression. They may exhibit a local twitch response or jump sign response to palpation or dry needling. Active trigger points are trigger points that cause pain and are present with muscle shortening that only occurs with external pressure. Dr. Simons was the first one to describe the local twitch. There have been multiple attempts to reproduce the examination of trigger points with little success. In 1992, Dr. Simons tried to reproduce this but he was unable to do so. Lew and colleagues in 1997 found inter- and intra-reliability using two highly trained examiners was poor. Another study by Gerwin in 1997 found it impossible to reproduce the same results. Therefore, trigger point identification becomes the art and experience of the provider.

PATHOLOGY

Currently, there is no pathology for trigger points as they are difficult to find. Histological studies have been inconclusive. Trigger point identification has not been possible with thermography, ultrasound, or magnetic resonance imaging (MRI). There are multiple theories as to the cause of myofascial pain. First was the energy crisis theory from Bengtsson in 1996, Hong 1996, and Simons in 1998. This theory postulated that there was an increased demand on muscle from macrotrauma or recurrent microtrauma which leads to increased calcium release from the sarcolemma and prolonged shortening of the sarcomeres. This prolonged shortening would then hinder the circulation causing a reduced oxygen supply causing the muscle cells to become unable to produce enough adenosine triphosphate (ATP) to initiate the process to relax the muscles. Ischemic byproducts of metabolism were thought to cause the pain by directly sensitizing the nerves. To date, there are no studies to confirm this theory.

Another theory is the motor end plate hypothesis. Some needle electromyography (EMG) studies have found that each trigger point contains minute loci that produce characteristic electrical activity. End-plate noise seen on needle EMG is thought to represent an increased rate of release of acetylcholine. As those who perform needle EMGs know, needle placement in close proximity to the end plate causes significant pain. A small amount of activity at the motor end plate is not enough to cause a muscle contraction, but it may be enough energy to propagate a small distance along the muscle cell membrane. This may activate a few contractile elements and be responsible for shortening of the muscle.

The third theory is that myofascial pain is a direct result of a protective mechanism of the muscles to protect the joints and the nerves. An example would be people who have listing of their spine in response to a radiculopathy. An X-ray would show a functional scoliosis and the patient would have taut bands protecting the spine from touching the site where the disc material is pushing out onto the nerve root. If someone had a left side radiculopathy, they would splint to the right. Over time, the muscle would shorten. Once the radiculopathy finally subsides, the patient would still feel pain due the shortened muscles. In patients with psychological issues, this listing becomes a protective mechanism and the patients refuse
to move because of the amplified pain. This in turn leads to more shortening of the muscles and more deconditioning. In patients with poor ergonomics, muscle shortening can also occur. Regular use of a non-ergonomically positioned computer can cause cervical facet joints to become inflamed. The cervical and shoulder girdle muscles have to try to protect the head and also allow the arms and hands to perform activities. This causes fatigue of the muscles and pain due to the reduced oxygen supply and lack of ATP.\textsuperscript{11,13}

The needle EMG findings in myofascial pain have been studied in great detail, but the study by Hubbard and Bergkoff in 1993 found no abnormal activity in trigger points. That same year, other studies showed some activity which was thought to be end-plate activity.\textsuperscript{21,34}

The muscle pain seen in myofascial pain has been described as early as 1938 by Kellgren.\textsuperscript{27} The pain is likely transmitted by group III and group IV nerve fibers. Neurotransmitters responsible for the pain are bradykinin, serotonin, and prostaglandins. Substance P has also been implicated, as well as prostaglandin E\textsubscript{2}.\textsuperscript{22}

There are many theories about trigger point pain and pain with compression from trigger points. The most popular theory in the precipitation of trigger points is that there are sensitized nociceptors that have increased responses to normal mechanical stimuli. The convergence projection theory accounts for the referred pain by positing that, since each dorsal horn neuron is connected to more than one body part, when noxious stimulus is received from another area it is misinterpreted as coming from a usually recognized site of pain. Another theory is that not all convergent connections are active all the time. Previously dormant connections can be unmasked and respond to painful stimuli. There is an agreement that this referred muscle pain must have a central basis.\textsuperscript{22} Researchers have applied stimuli to known receptive fields of specific dorsal horn neurons in rats and have found new receptive fields open for these neurons. Hong showed that this can cross to different spinal cord levels with increased substance P and calcitonin gene related peptide (CGRP).\textsuperscript{22}

Another way to look at this is to think of the myofascial covering as a gauze-like network that shapes the entire body and makes a network of 3D structures under the skin covering all the interior. Fill the gauze with structures including blood vessels and lymph structures. Myofascial pain can be in response to stressors like trauma or infection. The myofascia can trap blood vessels and lymph vessels or nerves causing diagnostic confusion. Fascia even surrounds the heart and holds other organs in place. When there is a trigger point in a muscle, it can cause pain at the end range of motion when a muscle is stretched. Because it hurts to move that muscle, the muscle shrinks and becomes less healthy; circulation is lost and microcirculation becomes impaired around the trigger point. Oxygen cannot be delivered easily and it is difficult to remove waste. The lymph system becomes stagnant as well. Other muscles do the work of the trigger point-weakened muscles and they develop satellite trigger points that develop areas of pain referred from the primary trigger point.\textsuperscript{1} Again, these referred pain sites create false impressions that they are due to other pathology unless they are correctly recognized. Secondary and satellite trigger points spread creating rapid patterns covering all four quadrants of the body. They look like fibromyalgia but they are not. The fascia is not only connected as sheets around a single muscle (the so-called epimysium) but is also present as intramuscular septa. Like tendons, the fascial part of the body is connective tissue and composed of cellular elements, fibers, and intracellular substances in various combinations. Specific for the fascia are the flat arrangement of bundles of collagen fibers. The fibers may pass from one bundle to another. The tendons can be stretched and will return to their original length after they are unloaded. It is not known if the construction of fascia is similar to that of the tendons and, if it is, they too would unload and return to normal length after being stretched. It is well known that when a limb is immobilized, it results in contracture and passive stretching is needed to reestablish the normal length of the joint and muscle fascia.\textsuperscript{37} There are mechano-receptors in the broad fascial sheath which serve a proprioceptive function. Therefore, the pathology appear to be consistent with the energy crisis theory as well as that of the protective action of the joint resulting in shortened myofascia and contracture. There does not seem to be any disagreement that this is true. The end-plate theory remains controversial.

**TREATMENT FOR MYOFACIAL PAIN**

To treat myofascial pain, a diagnostic workup, including imaging studies such as X-ray, ultrasound, and MRI along with neurodiagnostic testing, is necessary to rule out other sources of pain. If there is a source of significant pathology such as a cervical radiculopathy or a subacromial bursitis, these problems need to be treated. Once these are diagnosed and treated, then restoring function is important and the myofascial pain needs to be resolved. A recent review by Annaswamy on evidence-based treatment for myofascial pain treatment showed that very few medications have evidence that they work. Pharmacologically, the only positive study was for clonazepam. All others did not meet the criteria (Table). Myofascial release has not been proven to help.\textsuperscript{34} Stretching, use of a transcutaneous electrical nerve stimulation (TENS) unit, therapeutic ultrasound, and lasers have all shown some limited relief.

**Table.** Medications that do not meet evidence based criteria for relief of myofascial pain

<table>
<thead>
<tr>
<th>Muscle relaxer</th>
<th>Sedatives and hypnotics</th>
<th>Neuropathic analgesics and antidepressants</th>
<th>Anticonvulsants</th>
<th>Topical analgesics</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tizanidine</td>
<td>Alprazolam</td>
<td>Amitriptyline</td>
<td>Gabapentin</td>
<td>Lidocaine patch</td>
<td>Ibuprofen</td>
</tr>
<tr>
<td>Diclofenac</td>
<td>Nortriptyline</td>
<td>Pregabalin</td>
<td>Methyl salicylate patches</td>
<td>Diclofenac</td>
<td></td>
</tr>
<tr>
<td>Benozoquinine</td>
<td>Blockmode</td>
<td>Methyl patches</td>
<td>Celecoxib</td>
<td>Ketamine</td>
<td></td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>Procainamide patches</td>
<td>Thiacolchomide oxide gum</td>
<td>Milnacipran</td>
<td>Oxicam</td>
<td></td>
</tr>
<tr>
<td>Duloxetine</td>
<td>Ultraceprox</td>
<td></td>
<td>Oxicam</td>
<td>Ketamine</td>
<td></td>
</tr>
<tr>
<td>Citalopram</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

Practically, there needs to be some type of pain relief prior to stretching and strengthening to restore the muscle to its proper length so the patient can tolerate therapy. Local anesthetic injections have been used since the 1980s.\textsuperscript{13} The one consistent finding is that the pain relief outlasts the half life of the injected solution. Therefore, the mechanism involves more than a pure pharmacological effect from the anesthetic. Overall, local anesthetic injections when used with physical therapy modalities have proved to increase range of motion and increase the pressure threshold.\textsuperscript{30,32} This may be explained by the central modulation of pain, but, in reality, once the main problem is resolved, if the muscles are restored to normal flexibility and strength, the pain will resolve. If given too much anesthetic, there have been reports of myotoxicity as seen in ischemia and necrosis of muscle fibers. These are worse in the presence of vasoconstrictors.
Injections of sterile water have been tried and have not been as effective as treatment with the anesthetic. Dry needling involves advancement of an acupuncture-type needle in the muscle trying to reproduce the patient’s symptoms, visualization of the local twitch response, and then hopefully relief of the muscle tension and pain. Dr. Annaswamy’s review and the American College of Occupational and Environmental Medicine (ACOEM) Occupational Medicine Practice Guidelines: Evaluation and Management of Common Health Problems and Functional Recovery in Workers state that trigger point injections and acupuncture have a grade B recommendation (good scientific evidence as defined by this database) for myofacial pain. One Cochrane review also supported this as well as practice guidelines from the major insurance carriers. In practice, the author uses dexamethasone with trigger points injections and does not perform more than three injections in a 3-month period. This has produced better results than dry needling or local anesthetic alone. In the author’s opinion, there are other pathologies causing the muscle to protect the area which then causes the muscle shortening. The corticosteroid may decrease the inflammation in the primary pathology so the patient can do the stretching and strengthening exercises and not experience an increase in pain and thereby get better with exercise. Corticosteroids also have a direct anesthetic effect. Of course, there are no studies that support this supposition.

**Trigger Points Versus Tender Points**

Trigger points are seen in myofascial pain. Tender points are seen in fibromyalgia. Fibromyalgia and myofascial pain can exist together but are different. Fibromyalgia is a disease associated with central sensitization. It is diffuse, not localized, pain. Hyperalgesia or pain application in allodynia can also cause pain from non-painful stimuli such as noise and light. To control fibromyalgia pain, one must control the central pain generators. In fibromyalgia, systemic stimulus causes pain; in myofascial pain syndrome, the trigger points generate pain. They are treated differently. Patients with fibromyalgia tend to have an increased range of motion or hypermobility, causing the muscles to work harder and become fatigued; whereas in myofascial pain syndrome the muscle pain is a result of muscles shortening in response to a specific structural problem. This is a secondary problem due to deconditioning that needs to be rectified with stretching and then strengthening. In fibromyalgia, strengthening is needed and rarely stretching. Aerobic conditioning is also more important in fibromyalgia because of the central mechanism pathology (Figures 1, 2, 3, and 4).
COST EFFECTIVENESS OF TREATMENT MODALITIES

In the era of best practiced medicine, there are two models: the fee-for-service model and the capitation model. At this time, in 2012, the fee-for-service model is most common. With the recent health care changes, some believe that capitation is going to become the normal treatment model required for Medicare, Medi-Cal, and regular insurance in all things except for Workers’ Compensation. Therefore, when encountering a patient who has myofascial pain, a provider needs to determine the most cost-effective way to treat them. In this author’s estimation, the solution is to diagnose and treat any underlying cause, such as the radiculopathy or bursitis. Once this is addressed, the stretching and strengthening of the muscle should be undertaken. If pain prevents the patient from doing this effectively, then such things as spray and stretch can be implemented. If it is performed by a physical therapist the cost of treating the patient can be significant. For recurrent pain, performing a local trigger point injection with anesthesia, with or without corticosteroids, is a very inexpensive way to treat the patient. The patient then can continue the stretch and strengthening program on their own. Ifpray and stretch or trigger point injection does not lead to improvement, then the patient would need to return to a manual therapist or undergo further evaluation. After exhausting these routes, botulinum toxin (BTX) can be used.

In the new capitated system, physicians would pay for the BTX out of their own pockets. In this model, the physician is responsible for the cost of imaging studies (e.g., MRI), needle EMG, physical therapy, and medication. In this model, the treatment of trigger point injections with local anesthetic and soluble corticosteroids becomes a very cost-effective treatment modality. In the fee-for-service model, these types of injections are still reasonably cheap, and even with the use of ultrasound for better guidance of the injections it is still relatively inexpensive if one owns an ultrasound machine. It does not cost much to use an ultrasound to guide the trigger point injection placement in the correct muscle. There are probes, costing about $5,000, that can be very helpful for guiding injections (even though they may not be of much help for diagnostic evaluations). Finally, trigger point injections are safe. If performed wisely, there is very little risk of problems, excluding pneumothorax and infections.

CONCLUSION

Myofascial pain is experienced by patients due to changes in a muscle secondary to a primary problem. After the primary issue is resolved, the muscles need to be restored to their normal length. To this end, stretching followed by strengthening is necessary, as is enhancing the patient’s ability to stretch and strengthen. Trigger point injections are a cost-effective way to accomplish this. Trigger point injections are recognized by the ACOEM Guidelines as well as most major insurance companies as evidence based and are reimbursable.

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Trigger Point Injections for Myofascial Pain: They Don't Work!
Trigger Point Injections for Myofascial Pain: They Don’t Work!

Lawrence W. Frank, MD
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Myofascial pain is a commonly reported source of musculoskeletal pain. Among physicians there is disagreement on whether myofascial pain truly exists and, if so, whether treatment of myofascial pain is helpful or necessary. Among nonoperative specialists there is significant literature regarding the diagnosis and clinical treatment of this condition. Among surgical practitioners there is little to no reference to myofascial pain, even in major textbooks.

DOES MYOFASCIAL PAIN EXIST?

Muscle pain is ubiquitous. It is well-known that muscle is innervated by autonomic nociceptive fibers. Everyone can agree that structural injuries of the musculoskeletal system are frequently associated with muscle spasm or direct muscle injury. More difficult to establish is the process by which, outside of an identifiable primary muscular disease or obvious trauma, muscle can spontaneously become a primary pain generator.

Studies of myofascial pain indicate a high correlation with other cofactors including psychosocial stress, catastrophizing thoughts, de-conditioning, insomnia, poor posture, and participation in sedentary jobs, particularly desk work. Taking the above cofactors in mind, it is evident that many of the noninjection treatments for myofascial pain, including physical rehabilitation and pharmacologic management seem to directly address these cofactors, perhaps more so than the painful muscle itself. More so, the existence of these cofactors in relation to myofascial pain begs the question: “Are they a cause or an effect?”

THE DEFINITION OF PAIN

One of the biggest challenges encountered in treating painful conditions, particularly when the etiology is poorly defined, is the subjective nature of pain. In 1994, the International Society for the Study of Pain (IASP) developed a clinical definition for the usage of the word pain: “An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.” Not only is the sensation of pain a sensory experience, but it is just as much an emotional experience. In fact, pain and the emotional state are proportional. Both of the following statements are true:

- The worse the pain, the worse the emotional state.
- The worse the emotional state, the worse the pain.

The second statement is perhaps the most difficult for patients and busy physicians to accept, and perhaps this statement best clarifies why psychiatrically-active medicines are interestingly such a common pharmacologic treatment of myofascial pain.

The IASP definition also states that pain may be associated with “potential tissue damage, or described in terms of such damage.” Patients who strongly believe they have an injury despite the absence of anatomic pathology may experience pain. Such injury perceptions are well known to be accentuated in patients with somatization disorder or perhaps more commonly in suggestible patients exposed to unsubstantiated theories of well-meaning friends and relatives. As anyone with medical training can attest, such pain cannot and should not be treated with trigger point injections.
WHAT IS A MYOFASCIAL TRIGGER POINT AND HOW IS IT DIAGNOSED?

There are two types of myofascial pain defined by whether they cause local or radiating pain upon palpation of sore muscle. Tender points are defined as points within musculature that recreate the patient’s usual nonradiating pain. Trigger points were classically described as palpable portions of muscle that cause radiating pain to predefined areas, although the definition has changed over time. The latest pathophysiologic explanation of a trigger point comes from Hong and Simons, who have proposed a theory of sensitive and active loci corresponding to free nerve endings and motor end plates, respectively. The overlap of these loci results in trigger points (Figure 1).

Figure 1. Theory of trigger points. A random distribution of active (X) and sensitive (—) loci exist in muscle. Where they overlap, trigger points exist.9

ATrP = insertion trigger point, CTrP = central trigger point

No laboratory or pathologic tests are available for trigger point detection. Increased concentrations of histochemicals and low pH have been found in muscle underlying acupuncture points, corresponding to common myofascial trigger point locations. These changes were more pronounced in subjects experiencing myofascial pain compared to normal subjects, but were also noted in asymptomatic muscle of myofascial pain patients. Although these findings suggest possible systemic sensitization, they are not specific to myofascial trigger point locations. Magnetic resonance elastography and diagnostic ultrasound have revealed that the muscle twitch response can be visualized, although the twitch criteria are no longer considered diagnostic for myofascial pain.

Since there are no reliable diagnostic tests for myofascial pain, the diagnosis remains clinical. The diagnosis is based on palpation and dependent upon the experience of the examiner. That being said, the inter-rater reliability for the detection of trigger points is poor. In addition, the clinical diagnostic criteria for trigger points have changed over time, making the diagnosis a moving target. Travell and Simons, the authoritative sources for the diagnosis and treatment of myofascial pain, are presently recommending trifold criteria: a tender spot in a taut muscle, patient pain recognition on tender spot palpation, and limited painful range of motion. Discarded criteria include production of a characteristic referred pain pattern, a local twitch response under the examiner’s finger, the “jump sign” on muscle palpation, and muscle weakness. Classically, myofascial trigger points have had more strict criteria for diagnosis than tender points, but, as time has passed, trigger points and tender points are looking more and more alike.

HOW SHOULD MYOFASCIAL TRIGGER POINTS BE TREATED?

As in most benign disorders, treatment typically progresses from the benign to the more aggressive. Exercise therapy is a mainstay, including the recommendation for regular aerobic exercise, postural restoration, and various forms of stretching of the affected muscle. The technique of “spray and stretch” has been recommended, whereby the muscle is stretched after the overlying skin is sprayed with a vapocoolant such as ethyl chloride. Other techniques include myofascial release, massage therapy, contract-relax, muscle energy, strain-counterstrain, and proprioceptive neuromuscular facilitation.

Medication management may include antidepressants including tricyclics, which have been found to be particularly helpful in tension headache. Gabapentin demonstrated a trend toward relief of myofascial pain in one study, but the relief was not statistically significant. Restoration of dysfunctional sleep may significantly aid in the relief of myofascial pain.

Myofascial trigger point injections are the most aggressive form of treatment. Particularly in the cervical spine and trunk, caution should be exercised as myofascial trigger points may overlie important structures including the lungs, great vessels of the neck, the brachial plexus, and the spinal cord. Injuries to these structures from trigger point injections are rare but reported.

MYOFASCIAL TRIGGER POINT INJECTION TECHNIQUE

After the myofascial trigger point is identified, the skin is steriley prepared. A needle is then introduced into the offending muscle (Figure 2). Production of a muscle twitch response is thought to verify proper needle placement. Various substances can be injected, including local anesthetics, steroids, and botulinum toxin. Despite the common administration of pharmacologic agents, research indicates that trigger point needling without injection is equally effective. Mechanical stimulus of the muscle may be more important than pharmacologic treatment of underlying pathophysiology. In fact, more painful injections have been found to have greater placebo effects than nonpainful ones.

TRIGGER POINT INJECTION OUTCOME STUDIES

Outcome studies are difficult to interpret because it is impossible to separate the presence of the placebo effect. Attempts at the development of a placebo for an injection technique have proven quite difficult. Two systemic, evidence-based reviews of needling therapies in the management of myofascial trigger point pain indicate that the effect of these therapies is likely due to the needle effect or placebo rather than the activity of any injected drug.
review indicated that no reported treatment, including trigger point injections, are more efficacious than control interventions. Yet another study examining five randomized controlled trials of trigger point injections indicated that they were as effective as a “general approach,” but not more effective than the placebo.

**WHY NOT TO PERFORM TRIGGER POINT INJECTIONS**

In order to have effective treatment, a disease must be identifiable. Unfortunately, there is no agreement on how to identify myofascial trigger points. Over the past 30-40 years, the diagnostic criteria have changed continually, making the diagnosis a moving target. In fact, the diagnostic criteria have become increasingly broad, trading sensitivity for specificity. It is not at all clear whether the psychological and postural cofactors are a cause or an effect of myofascial pain.

As for trigger point injections themselves, the literature clearly shows that the short term benefits do not exceed the effect of placebo. The injection of pharmacologicals has no value. As for long term benefits, general care with a reasonable medication regimen and an exercise program is equally effective. There are rare but potentially significant complications to these procedures, including pneumothorax, hematoma, and spinal injury.

Patient reports of short term benefits from trigger point injections may give physicians a false sense of confidence in their procedural skills, blinding physicians to the fact that pain is being relieved by the placebo effect. Such false confidence can cause physicians to overprescribe trigger point injections and lose perspective of the need to address underlying cofactors including psychosocial stress, depression, anxiety, poor physical condition, and sedentary lifestyles. Avoiding the use of trigger point injections maintains the clinical focus on the lifestyle modifications and discipline necessary for real disease modification, and it decreases patient dependence on the physician in the long run.

**WHAT SHOULD BE DONE FOR PATIENTS WITH MYOFASCIAL PAIN?**

Despite the evidence, why do physicians continue to perform trigger point injections? From the physician’s perspective, it is not only psychologically easier, but also financially more rewarding, to give a trigger injection during a busy clinic than to spend time educating, convincing, and encouraging patients to make lifestyle changes necessary for symptom relief. From the myofascial pain patient’s perspective, receiving an on-demand trigger point injection is often much easier than adhering to a strict regimen of lifestyle changes.

As general treatment is just as efficacious as trigger point management, aggressive noninvasive treatment of myofascial pain should include the following:

- A short course of active physical therapy to stretch affected muscle, build muscle mass, and teach self-management by providing a home exercise program.
- Aerobic exercise to increase muscle endurance and combat deconditioning.
- Healthy diet modifications for weight loss and steady energy throughout the day.
- Thirty minute position changes for sedentary workers, as needed.
- Treatment of insomnia.
- Aggressive treatment of clinical anxiety and depression.
- Patient compliance monitoring for all the above.

**SUMMARY**

Unfortunately, the medical literature supports neither the diagnosis of myofascial trigger points as a clinical or pathophysiologic entity, nor the efficacy of trigger point injections. Trigger point injections are a questionable treatment looking for a disease. Unlike advances in other diseases, which demonstrate increasing clarity over time, the literature on myofascial pain syndrome and trigger point injections continue to lead down a road to nowhere.

**REFERENCES**

TRIGGER POINT INJECTIONS FOR MYOFASCIAL PAIN: THEY DON'T WORK!


Crossfires CME Questions

1. Which of the following is MOST TRUE regarding painful conditions and botulinum toxin (BTX)?
   A. The Food and Drug Administration (FDA) has approved BTX for treatment of 14 painful conditions.
   B. There are significant evidence-based differences between the different BTX types and pain outcome measures for cervical dystonia.
   C. BTX has been shown to modify transmission of other neurotransmitters besides acetylcholine that are involved in pain transmission and modulation.
   D. BTX has been shown to decrease pain behavior by direct central nervous system neurotransmitter blockade.

2. When differentiating myofascial pain syndrome from cervical dystonia which of the following is MOST TRUE?
   A. There is no clear way to separate these two problems in some patients.
   B. The clear patterns of muscles affected in cervical dystonia are usually different from the muscles affected in myofascial pain and this clarifies the diagnosis.
   C. Two separate evidence-based studies have clarified techniques skilled physicians can use to separate these two diagnoses.
   D. Palpation of trigger points will usually rule out cervical dystonia and rule in myofascial pain.

3. Which of the following is MOST TRUE regarding terminology and myofascial pain?
   A. Fibromyalgia syndrome and myofascial pain syndrome are usually considered the same syndrome.
   B. Patients with latent trigger points present to physicians with pain complaints.
   C. Muscle pain is usually synonymous with myofascial pain syndrome.
   D. Patients with myofascial pain usually have trigger points.

4. Which of the following is MOST TRUE regarding the use of BTX as a treatment?
   A. BTX has been shown to be superior to placebo in the treatment of pain from cervical dystonia.
   B. BTX has FDA approval for the treatment of myofascial pain.
   C. Head-to-head evidence-based trials have shown that BTX is less cost effective than lidocaine injections for the treatment of myofascial pain.
   D. BTX injections combined with oral opioid medications will likely prove to be the most effective treatment of myofascial pain.

5. BTX-A works by which of the following?
   A. Blocking endocytosis of acetylcholine containing vesicles.
   B. Directly affecting muscle cell membrane.
   C. Cleaving to the SNAP-25 protein.
   D. Both A and C.
   E. None of the above.

6. BTX-B works by which of the following?
   A. Cleaving synaptobrevin/vesicle-associated membrane protein (VAMP).
   B. Interfering with the motor axon membrane resting potential.
   C. Causing functional denervation of the neuromuscular junction.
   D. Both A and C.
   E. None of the above.

7. Randomized, placebo-controlled trials show BTX A decreased pain in the following conditions EXCEPT:
   A. Chronic migraine headaches.
   B. Cervical dystonia.
   C. Diabetic neuropathy.
   D. Myofascial pain syndrome.
   E. Lupus.

8. Side effects from BTX injection may include which of the following?
   A. Diplopia.
   B. Dysphagia.
   C. Dysarthria.
   D. Generalized weakness.
   E. All of the above.
9. BTX-A (Botox®) is FDA indicated for all of the following diagnoses EXCEPT:
   A. Chronic migraine headaches.
   B. Axillary hyperhidrosis.
   C. Cervical dystonia.
   D. Peri-orbital wrinkle lines.
   E. Urinary bladder over activity.

10. Myofascial pain is believed to be caused by which of the following?
    A. Muscle shortening.
    B. Lack of oxygen in the muscles.
    C. Antibodies to muscles.
    D. A and B.
    E. B and C.

11. Which of the following medicines has clinical evidence to treat myofascial pain?
    A. Ibuprofen.
    B. Nortriptyline.
    C. Gabapentin.
    D. Clonazepam.
    E. Hydrocodone.

12. Which of the following modalities has evidence-based research to treat myofascial pain?
    A. Electrical stimulation.
    B. Manual therapy.
    C. Therapeutic ultrasound.
    D. All of the above.
    E. None of the above.

13. Fibromyalgia is another name for myofascial pain syndrome.
    A. True.
    B. False.

14. Trigger point injections for treatment of myofascial pain syndrome has clinical research to support its efficacy.
    A. True.
    B. False.

15. According to the International Society for the Study of Pain, the definition of pain encompasses all the following EXCEPT:
    A. An unpleasant sensory experience.
    B. An unpleasant emotional experience.
    C. Association with aching and dysesthesias.
    D. Associated with actual tissue damage.
    E. Associated with potential tissue damage.

16. According to the latest theories of myofascial pain:
   A. Trigger points may exist at the overlap of active and sensory loci within muscle.
   B. Muscle contains active loci associated with free nerve endings.
   C. Muscle contains sensory loci associated with end plates.
   D. Trigger points can be identified with characteristic pain referral patterns.
   E. Trigger points can be identified with a local twitch response upon palpation.

17. Myofascial pain is BEST diagnosed by which of the following?
    A. Magnetic resonance elastography.
    B. Diagnostic ultrasound.
    C. Muscle biopsy.
    D. Palpatory examination.
    E. Assay of histochemicals from painful muscle.

18. First line management of myofascial pain should include all the following EXCEPT:
    A. Sleep restoration.
    B. Spray and stretch techniques.
    C. Massage therapy.
    D. Aerobic conditioning restoration
    E. Trigger point injections.

19. According to the medical literature, which of the following is the BEST explanation for the effect of trigger point injections?
    A. Decreases local inflammation with injected steroids.
    B. Direct mechanical stimulation of muscle.
    C. Trigger point identification has high inter-rater reliability.
    D. Desensitizes sensory loci with local anesthetic.
    E. Neuromuscular blockade with BTX.