

Part 1

Pathophysiology

Chapter 1

Myofascial Trigger Points: Translating Molecular Theory into Manual Therapy

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Introduction

Simons, Travell, and Simons¹ defined the myofascial trigger point (MTrP) as “. . . a hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. The spot is tender when pressed, and can give rise to characteristic referred pain, motor dysfunction, and autonomic phenomena. . . .” Thus, each MTrP contains a sensory component, a motor component, and an autonomic component. These components comprise a new “integrated hypothesis” regarding the etiology of MTrPs.¹ This hypothesis involves local myofascial tissues, the central nervous system (CNS), and systemic biomechanical factors. The “integrated hypothesis” has changed our approach to treating MTrPs. The purpose of this chapter is to review new concepts concerning MTrPs and to describe our evolving approach to their treatment.

The Motor Endplate: Epicenter of the Myofascial Trigger Point

Simons² implicated the motor endplate as the central etiology of MTrPs. The motor endplate is synonymous with the neuromuscular junction (the first term describes structure, the latter term describes function); it is the site where an α -motor neuron synapses with its target muscle fibers. The α -motor neuron terminates in multiple swellings termed presynaptic boutons. Each bouton contains many acetylcholine (ACh) vesicles, clustered around structures called dense bars (see **Figure 1-1**). Voltage-sensitive calcium channels (VsCCs, specifically P/Q-type VsCCs) also cluster near dense bars. When voltage running down an

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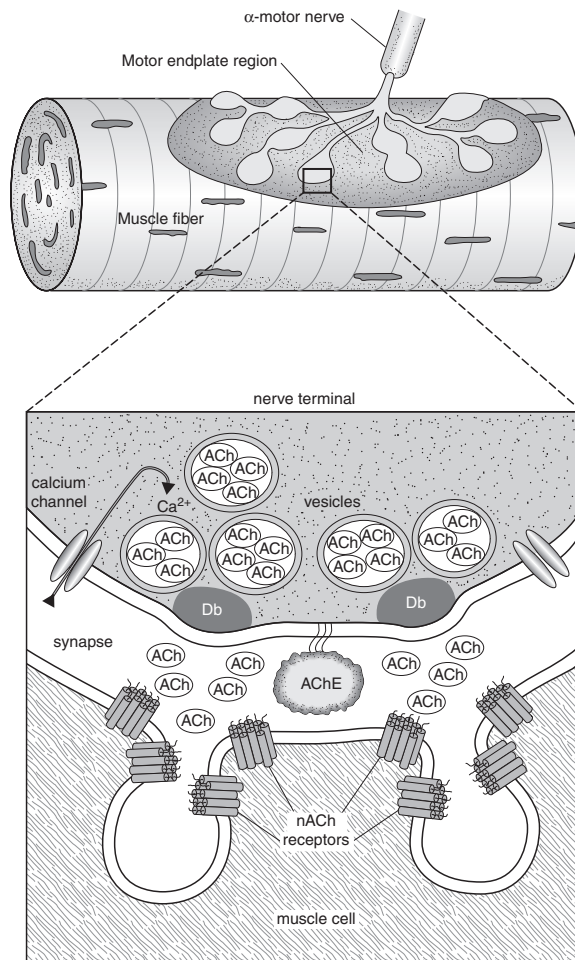


Figure 1-1 The motor endplate—proposed site of trigger point dysfunction. Top illustration: The junction between the α -motor neuron and the muscle fiber. Bottom illustration: Presynaptic boutons are separated from the postsynaptic muscle cell by the synaptic cleft. Within each bouton are many vesicles containing ACh, clustered around dense bars (Db). Also clustered around the Db are calcium channels. The Db is the site of ACh release into the synaptic cleft. Across the synaptic cleft from the Db, the postsynaptic muscle cell membrane forms junctional folds that are lined with nicotinic ACh receptors (nACh). ACh released into the synaptic cleft activates nACh receptors, then is inactivated by the acetylcholinesterase enzyme (AChE).

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α -motor neuron reaches VsCCs in the bouton, the VsCC channels open, leading to an influx of calcium ions (Ca^{2+}) into the bouton from the extracellular space. The influx of Ca^{2+} causes the ACh vesicles to release their transmitter into the synaptic cleft (Figure 1-1).

Across the synaptic cleft, the postsynaptic muscle cell membrane forms junctional folds that are lined with nicotinic ACh receptors (nAChs). The nACh is a ligand-gated cation channel, and ACh is its ligand. Binding of ACh to nACh opens its channel, allowing sodium ions (Na^+) and potassium ions (K^+) to move in and out of the muscle cell membrane. Movement of Na^+ and K^+ depolarizes the postsynaptic cell, forming a miniature endplate potential (MEPP). A sufficient number of MEPPs activate VsCCs (specifically L-type VsCCs), which subsequently trigger another Ca^{2+} channel, the ryanodine receptor. The ryanodine receptor is imbedded in the membrane of an intracellular structure called the sarcoplasmic reticulum, which houses intracellular stores of Ca^{2+} . Activation of the ryanodine receptor releases Ca^{2+} from the sarcoplasmic reticulum into the cytoplasm of the muscle cell. This triggers the interaction between actin and myosin, and the sarcomere contracts.

Electromyography (EMG) studies of MTrPs have reported spontaneous electrical activity (SEA) in MTrPs, while adjacent muscle tissues are electrically silent.³ Hubbard and Berkoff³ originally attributed the source of SEA action potentials to sympathetically activated intrafusal muscle spindles. These researchers were unaware of previous work by Liley of New Zealand, who had demonstrated that SEA was a consequence of ACh release at motor endplates.⁴ Simons⁵ “connected the dots” by correlating SEA with “endplate noise” that had been described by electromyographers, and he linked SEA to excessive ACh release, which he proposed as the primary cause of MTrP development. This “motor endplate” hypothesis was tested in Hong’s laboratory,⁶ where MTrPs were injected with botulinum toxin type A, which blocks ACh release at the motor endplate. This treatment significantly decreased SEA activity. Mense et al⁷ confirmed the hypothesis using a rat MTrP model. They injected diisopropylfluorophosphate (DFP), a drug that increases synaptic ACh, into the proximal half of the gastrocnemius muscle, and the motor nerve was electrically stimulated for 30–60 minutes to induce muscle contractions. The distal half of the muscle, which performed the same contractions, served as a control. Proximal and distal sections of the muscle were then examined for morphological changes. The DFP-injected proximal half exhibited significantly more contracted and torn muscle fibers compared to the distal half of the muscle.

Myofascial tension may play a role in excess ACh release. Chen and Grinnell⁸ showed that a 1% increase in muscle stretch at the motor endplate evoked a 10% increase in ACh release. These researchers postulated that tension upon integrins (cell-surface proteins that bind connective tissues) in the presynaptic membrane was transduced mechanically into ACh vesicle release.

Expanding the Endplate Hypothesis

Simons’ description of a presynaptic dysfunction (excessive ACh release), however, is only one way to interpret the endplate hypothesis. We can expand the hypothesis to include

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presynaptic, intrasynaptic, and postsynaptic dysfunctions.⁹ Intrasynaptic ACh must be deactivated; otherwise, it will continue to activate nAChRs in the muscle cell membrane. ACh is normally deactivated by the enzyme acetylcholinesterase (AChE), which is held in the synaptic cleft by a structural protein (collagen Q, ColQ) that anchors it to the plasma membrane (Figure 1-1). AChE deficiency permits excess ACh to accumulate in the synaptic cleft, tonically activating nAChRs. Several genetic mutations cause AChE deficiency, including mutations in ColQ. The gene for AChE expresses several splice variants,¹⁰ which are alternative ways in which a gene's protein-coding sections (exons) are joined together to create a messenger RNA molecule and its translated protein. AChE splice variants are less effective at deactivating ACh, and the expression of these splice variants can be induced by psychological and physical stress.¹⁰ Drugs and other chemicals may cause AChE deficiency. DFP, the drug used in the aforementioned experiment by Mense et al,⁷ is an AChE antagonist. Organophosphate pesticides are AChE antagonists, and poisoning by these pesticides causes changes in motor endplates and MTrP-like pathology.¹¹⁻¹³ Muscle damage caused by AChE antagonists has been reduced by pretreatment with postsynaptic L-type VsCC blockers such as quinidine¹² and diltiazem.¹³

Postsynaptically, a “gain-of-function” defect of the nAChR may confer muscle hyperexcitability, a hallmark of MTrPs. Gain of function refers to an increased response by the nAChR, via several possible mechanisms: nAChR overexpression, constitutively active nAChRs,¹⁴ nAChRs that gain responsiveness to choline (an ordinary serum metabolite),¹⁴ or nAChRs whose channels remain open longer than normal.¹⁵ The nAChR is an assembly of 5 subunits; at least 16 genes encode these subunits, so that the nAChR is particularly susceptible to mutational defects. Motor endplate nAChRs express a unique subunit assembly, whereas nAChRs in the central nervous system and in autonomic nerves express a different subunit configuration.¹⁶

The relative consequences of presynaptic, synaptic, and postsynaptic dysfunctions are under debate. Wang et al¹⁷ used a variety of pharmacological tools to conclude that presynaptic mechanisms modulate the motor endplate rather than synaptic (AChE) or postsynaptic (nAChR density) mechanisms. Conversely, Nakanishi et al¹⁸ determined that postsynaptic manipulation (using α -bungarotoxin, an nAChR antagonist) modulated motor endplates to a greater degree than presynaptic manipulation (using botulinum toxin, an inhibitor of ACh release).

Motor Component

MTrPs have a motor component, whereas tender points found in patients with fibromyalgia do not. MTrPs have been biopsied and found to contain “contraction knots” described as “large, rounded, darkly staining muscle fibers and a statistically significant increase in the average diameter of muscle fibers. . . .”¹⁹ Thus, the structure of contraction knots differed from that of normal muscle fibers. Functionally, excessive motor activity initiates several perverse mechanisms that cause MTrPs to persist. Muscle contraction compresses local sensory nerves, which reduces the axoplasmic transport of molecules

that normally inhibit ACh release.^{20,21} Muscle contraction also compresses local blood vessels, reducing the local supply of oxygen. Reduced oxygen, combined with the metabolic demands generated by contracted muscles, results in a rapid depletion of local adenosine triphosphate (ATP).

The resultant “ATP energy crisis”¹ triggers a cascade of pre- and postsynaptic compensations. Presynaptic ATP directly inhibits ACh release,²² so depletion of ATP increases ACh release. Postsynaptic ATP powers the Ca^{2+} pump that returns Ca^{2+} to the sarcoplasmic reticulum. Hence, loss of ATP impairs the reuptake of Ca^{2+} , which increases contractile activity, creating a vicious cycle.¹⁹ Excess Ca^{2+} may snowball into “ Ca^{2+} -induced Ca^{2+} release,” where Ca^{2+} induces further Ca^{2+} release from intracellular stores via ryanodine receptors, triggering actin and myosin, leading to muscle spasm.

Some controversy surrounds adenosine, a breakdown product of ATP. Adenosine normally decreases motor endplate activity by activating presynaptic adenosine A1 receptors, which reduce P/Q VsCC currents, thus reducing ACh release.²³ However, high levels of synaptic adenosine, from excess ATP breakdown (as is hypothesized to occur in the ATP energy crisis model), may activate postsynaptic adenosine A2 receptors, which recruit L-type VsCCs currents, thus triggering muscle contraction.²⁴

MTrPs exert profound, yet unpredictable, influences upon motor function. MTrPs may excite or inhibit normal motor activity in their muscle of origin or in functionally related muscles. Latent MTrPs can be equally influential upon motor function. Motor inhibition is often identified clinically as muscle weakness, but treatment often focuses on strengthening exercises that only augment abnormal muscle substitution until the inhibiting MTrPs are inactivated. This inhibition can also cause poor coordination and muscle imbalances. These MTrP effects have gone largely unrecognized because of a lack of published research studies. Headley has explored these effects using surface electromyography, describing inhibition of the trapezius by MTrPs in the same muscle,²⁵ inhibition of anterior deltoid by MTrPs in the infraspinatus,²⁵ inhibition of gluteal muscles by MTrPs in the quadratus lumborum,²⁵ and excitation (referred spasm) of the paraspinals by MTrPs in the tensor fascia lata.^{26,27}

Sensory Component

MTrPs are painful. Pain begins in peripheral tissues as nociception, transmitted by $\text{A}\delta$ - and C-fiber afferent sensory neurons (nociceptors). Mechanical pressure, thermal stimuli, and many chemicals activate nociceptors; potassium ions, protons, and free O_2 radicals are by-products of muscle metabolism and the hypothesized ATP energy crisis. Histamine is released from mast cells that migrate into injured tissues. Serotonin is released from platelets after they are exposed to platelet-activating factor (released from the mast cells). Bradykinin is cleaved from serum proteins. All of these chemical “activators” bind to receptors in the nociceptor and initiate an action potential. “Sensitizers” are also released from damaged tissue; examples include prostaglandins, leukotrienes, and substance P. Sensitizers decrease the activation threshold of a neuron, so that the nociceptor fires with less activation. This leads to peripheral sensitization and hyperalgesia.

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Sensitizing substances may also generate a focal demyelination of sensory nerves. Demyelination creates abnormal impulse-generating sites (AIGS) capable of generating ectopic nociceptive impulses.²⁸ Shah et al²⁹ used a microdialysis needle to sample tissue fluids from the upper trapezius muscle in nine subjects; elevated concentrations of protons, bradykinin, serotonin, substance P, norepinephrine, calcitonin gene-related peptide, tumor necrosis factor- α , and interleukin-1b were detected in active MTrPs, compared to latent MTrPs and control subjects without MTrPs. The difference was statistically significant ($P < 0.01$) despite the small sample size.

A persistent barrage of nociceptive signals from MTrPs may eventually cause “central sensitization,” a form of neural plasticity involving functional and/or structural change within the dorsal horn of the spinal cord. The sensitized dorsal horn becomes a “neurologic lens,” consolidating other nociceptive signals that converge upon the same segment of the spinal cord, including other somatic dysfunctions and visceral dysfunctions.¹ As a result, postsynaptic spinal neurons exhibit decreased activation thresholds, increased response magnitudes, and increased recruitment of receptive field areas. They fire with increased frequency or fire spontaneously, transmitting nociceptive signals to supraspinal sites, such as the thalamus and cerebral cortex. Central sensitization may also modulate spinal interneurons and descending inhibitory pathways. Central sensitization is symptomatically expressed as allodynia (pain to normally nonpainful stimuli) and hyperalgesia (abnormally increased sensation of pain). Simons, Travell, and Simons¹ described the CNS as an “integrator” of MTrPs, akin to Korr’s description³⁰ of the CNS as an “organizer” of somatic dysfunction.

Autonomic Component

Autonomic phenomena associated with MTrPs include localized sweating, vasoconstriction or vasodilation, and pilomotor activity (“goose bumps”).¹ MTrPs located in the head and neck may cause lacrimation, coryza (nasal discharge), and salivation.¹ The autonomic nervous system (ANS) is primarily involved in reflex arcs, exerting control of cardiac muscle and smooth muscle in blood vessels, glands, and visceral organs. Hubbard and Berkoff³ reviewed the literature that demonstrated ANS involvement in skeletal muscles and MTrP formation. Sympathetic neurons innervating vessels in skeletal muscles may exit the perivascular space and terminate among intrafusal fibers within muscle spindles. Sympathetic neurons release norepinephrine, a neurotransmitter involved in the “fight-or-flight” response. Norepinephrine activates α_1 -adrenergic receptors in the intrafusal muscle cell membrane. Activation of α_1 -adrenergic receptors depresses the feedback control of muscle length, detrimentally affecting motor performance and possibly contributing to the ATP energy crisis.³¹ Norepinephrine has been shown to augment the amplitude and duration of MEPPs in frog leg motor endplates.³² Phentolamine, an antagonist of α_1 -adrenergic receptors, decreases SEA in MTrPs.³³ Similar effects have been seen with local intramuscular injections of phenoxybenzamine, another α_1 -adrenergic antagonist.³⁴

The ANS may indirectly exacerbate MTrP formation via viscerosomatic reflexes. Visceral autonomic afferents from disturbed viscera carry signals to the dorsal horn.

Chronic input eventually facilitates neurotransmission at that spinal level.³⁵ This form of central sensitization accelerates in the presence of nociceptor AIGS and ephaptic crosstalk with neighboring autonomic nerves.⁹ Ephaptic crosstalk (cross-excitation) is the nonsynaptic interaction between two nerves that are parallel and relatively close together so that their action potentials influence each other.

Translating Theory to Therapy

The motor endplate and ATP energy crisis hypotheses have changed our approach to treating MTrPs. For example, the 1999 edition of *Travell and Simons' Myofascial Pain and Dysfunction: The Trigger Point Manual*,¹ abandoned the application of heavy ischemic compression upon MTrPs. Deep digital pressure that produces additional ischemia is not beneficial. Instead, Simons, Travell, and Simons¹ recommended applying *gentle* digital pressure to MTrPs to avoid exacerbating tissue hypoxia. They named their technique “trigger point pressure release.” A single finger pad palpates the MTrP while the affected muscle is passively lengthened to a point of tissue resistance. Next, the MTrP is pressed with slowly increasing pressure until the palpating finger encounters a barrier (local tissue resistance). The engaged barrier is held until a release of tension is palpated. The finger “follows” the released tissue by taking up tissue slack, engaging a new barrier, and repeating the sequence. This press and stretch is believed to restore abnormally contracted sarcomeres to their normal resting length. We hypothesize that press and stretch mechanically uncouples myosin from actin, a process that normally requires ATP, so that the technique reduces ATP demand and breaks the energy crisis cycle. Press and stretch may also help release the “stuck” spring function of the titin connection to the Z bands within sarcomeres.

Simons, Travell, and Simons' new manual¹ also emphasized the relationship between MTrPs and nearby articular dysfunctions. They correlated suboccipital MTrPs with occipito-atlantal dysfunction, semispinalis capitis MTrPs with occipito-atlantal and atlanto-axial dysfunctions, and splenius MTrPs with upper thoracic articular dysfunctions.¹ This close association between MTrPs and articular dysfunctions is the result of a positive feedback loop. Lewit has emphasized this close association in several publications.^{36,37} An MTrP in a muscle that crosses an articulation reduces this articulation's full range of motion, and the MTrP taut band exerts continuous compression upon the articulation. Soft tissues surrounding the articulation cannot withstand chronic compression or tension, and they respond with increased sensitivity. When sufficiently sensitized, these structures send continuous nociceptive messages to the CNS, which responds by further activation of MTrPs, which in turn increases the muscle tension. This positive feedback loop aggravates the articular distress. Articular dysfunctions can be treated directly by muscle energy technique (similar to contract-relax or postisometric relaxation techniques), joint mobilization, and high-velocity, low-amplitude thrust techniques. Articular dysfunctions can be treated indirectly with techniques that address dysfunctional muscles or fascia that cross the articulations, such as strain-counterstrain and myofascial release. Indications and precautions for these techniques are the same as with any articular dysfunctions.

Methods for treating MTrPs and articular dysfunctions work best when combined with patient education.

Patient Education

Postural training is paramount. Postural disorders often contribute to the perpetuation of MTrPs. For example, postural strain of the suboccipital muscles may cause MTrPs in these muscles,¹ thus leading to further deterioration in muscle structure and function, including radiating somatic pain and atrophic changes, such as muscle atrophy, fibrosis, and decreased tensile strength.³⁸ Suboccipital muscles contain a high density of proprioceptors,³⁹ so muscle atrophy leads to a loss in proprioceptive balance and a loss of proprioceptive “gate control” at the dorsal horn. This gives rise to chronic pain syndromes, including neck pain and headache.³⁸ In these patients, proprioceptive exercises can be very helpful, such as close-eyed balance training. Biomechanical factors that stress muscles, such as repetitive activities, must be avoided. Biomechanical stress of a *cold* muscle is a key factor in the formation of MTrPs.¹ Cooling the muscle apparently up-regulates nAChR activity at the motor endplate.⁴⁰

Patients with MTrPs should avoid excess coffee⁴¹; caffeine up-regulates the motor endplate by acting as a ryanodine receptor agonist.⁴² Tobacco should also be avoided, because nicotine up-regulates L-type VsCCs and nAChR expression, which may lead to muscle hyperexcitability.⁴³ Nicotine activates nAChRs in the CNS and autonomic nerves. Although nAChRs in motor endplates are not normally activated by nicotine, mutational defects may sensitize motor endplate nAChRs. One study indicated that ethanol also facilitates motor endplate activity, via a presynaptic mechanism.⁴⁴

Simons, Travell, and Simons¹ recommended a diet adequate in vitamins and minerals for the prevention of MTrPs. Amazingly, in the more than 20 years since that recommendation, no well-designed study has been published concerning the effects of vitamin supplementation upon MTrPs. However, a wealth of clinical experience suggests that low-normal and subnormal levels of vitamins and minerals act as strong perpetuating factors of MTrPs. Many case histories attest to patients who responded weakly to manual and/or injection treatment, but adequate supplementation (the return of blood vitamin levels to within mid-normal range) brought about an effective response to the same treatment, and with continued supplementation the patients had no relapse. Interestingly, in two cases a VA hospital physician advised discontinuation of “unnecessary” vitamin supplements, and within a few months the patients returned to the myofascial pain clinic with active MTrPs as before. Reinstatement of their supplement regimen and a replication of previous treatment restored their health (Simons, unpublished data). Similarly, anemia is a perpetuating factor of MTrPs that must be corrected to achieve lasting results from treatment.⁴⁵ Inadequate hemoglobin perpetuates the hypoxia present in MTrPs.⁴⁶ The importance of calcium and magnesium for normal muscle function is well documented, and trace elements are well known to be essential for many body functions, including muscle function. Supplementing the diet with phosphatidyl choline has been recommended for the

treatment of fibromyalgia,⁴⁷ but this may actually provoke MTrPs in some patients. Choline is a precursor to ACh, and an nAChR gain-of-function mutation may enable choline to directly activate the mutated receptors.¹⁴ High doses of phosphatidyl choline are found in supplements containing lecithin, with lower amounts in raw egg yoke, organ meats, soybeans, peanuts, wheat germ, and brewer's yeast.

An estimated 50% of patients with chronic musculoskeletal pain take herbal remedies, so it behooves all health practitioners to understand the mechanisms of herbal medicines.⁴⁸ Clinical experience has shown that myofascial pain can be improved with many herbal remedies and essential oils,⁴⁷ including lavender (*Lavandula angustifolia*), lemon balm (*Melissa officinalis*), rosemary (*Rosmarinus officinalis*), kava kava (*Piper methysticum*), skullcap (*Scutellaria lateriflora*), passionflower (*Passiflora incarnata*), rose (*Rosa* species), and valerian (*Valeriana officinalis*). Nearly all these herbs contain linalool, a monoterpene that inhibits ACh release (a presynaptic mechanism) and nAChRs (a postsynaptic mechanism).⁴⁹ Marijuana (*Cannabis* species) also produces linalool, although the herb's efficacy may be due to tetrahydrocannabinol (THC), which inhibits P/Q-, N-, and L-type VsCCs via cannabinoid receptors found in the motor endplate.⁵⁰ Sativex, a standardized extract dispensed as an oromucosal spray, has been approved for the treatment of muscle spasticity and pain in Canada.⁵¹ THC works by mimicking an endogenous neurotransmitter named anandamide.⁵⁰ Anandamide and THC bind to the same neuroreceptor, known as the cannabinoid receptor. Enhanced release of "endocannabinoids" may be one of the mechanisms of osteopathic manipulative treatment,⁵² parallel to the effects of manipulative treatment upon serum endorphin levels.⁵³

Getting to the Point

Needling may be necessary to inactivate MTrPs. The motor endplate hypothesis led to the injection of MTrPs with botulinum toxin type A, which blocks ACh release.⁵⁴ A variety of VsCC blockers have also been injected. Recall that P/Q-type and L-type VsCCs are the primary pre- and postsynaptic Ca²⁺ channels (respectively) in normal adult motor endplates. The P/Q-specific antagonist omega-agatoxin IVA (also known as omega-conotoxin GVIIC) has shown promise in rat studies,⁵⁵ while verapamil, a L-type VsCC blocker, reduced MTrP excitability in rabbits.⁵⁶ The drug had no effect on MEPP (a presynaptic measure), but it decreased postsynaptic currents.⁵⁷ Thus, verapamil may function as an nAChR antagonist, rather than by way of its known VsCC antagonism. Similarly, quinine, another L-type antagonist, also downregulates nAChRs and may restore AChE activity.¹² Diltiazem also merits investigation. This L-type Ca²⁺ channel blocker corrects myopathies caused by defects in AChE activity.¹³ However, nifedipine, yet another L-type antagonist, unexpectedly increased ACh activity at motor endplates due to a unique effect upon ryanodine-sensitive intracellular Ca²⁺ stores.⁵⁸ Hence, research with VsCC blockers has generated conflicting results, and recent clinical trials with botulinum toxin type A have produced mixed results.⁵⁹ Dry needling is usually as effective as injecting anything; if the procedure elicits a local twitch response, dry needling should be as effective as botulinum toxin type A and much less expensive.

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Some nAChR antagonists and channel blockers can directly penetrate skin, so they need not be injected. Lidocaine patches have recently been suggested.⁶⁰ In 1983, Simons, Travell, and Simons¹ recommended dimethisoquin ointment for massaging MTrPs in superficial muscles such as the orbicularis oculi, frontalis, and occipitalis. Dimethisoquin, a local anesthetic, inhibits voltage-gated Na⁺ channels (conferring its anesthetic effect), but it also acts as an nAChR antagonist.⁶¹ Its potency is much greater than lidocaine and procaine, and dimethisoquin is uniquely selective for the motor endplate nAChR subtype. Massage with capsaicin cream (available over the counter as a 0.075% cream) is useful for treating MTrPs located in surgical scars,⁶² which are particularly refractory to treatment.¹ This seems counterintuitive: Capsaicin is the primary active ingredient in hot peppers, and it activates the vanilloid receptor (TRPV1) in nociceptors. However, with repeated exposure to capsaicin, TRPV1 receptors become desensitized, which explains the seemingly paradoxical use of capsaicin as an analgesic.⁶³ Another massage treatment of MTrPs uses frequency-specific microcurrent (FSM), which delivers electromagnetic currents through graphite-conducting gloves. In relation to the MTrP ATP energy crisis model, studies have shown that FSM increases ATP production in muscle tissues as well as reducing cytokine levels.⁶⁴ To successfully treat MTrPs with FSM, a clinician must be skillful at finding MTrPs.

Conclusion

The MTrP, according to our working hypothesis, centers upon dysregulated motor endplates, sustained by a neural loop of sensory afferents and autonomic efferents. The resulting ATP energy crisis links with a spinal reflex disorder known as central sensitization. Treatment must simultaneously address the symptomatic trigger points and their underlying causes. Appropriate treatment includes dry needling (also known as acupuncture), vapocoolant spray-and-stretch, and thermal treatment (including ultrasound and infrared laser), some of which are discussed in other chapters of this book. New approaches described in this chapter, including manual techniques (“press-and-stretch” and articular methods), patient education, and ACh- or VsCC-attenuation techniques (e.g., medications, herbs, and nutrition), have evolved from our new etiological concepts.

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