Understanding effective treatments of myofascial trigger points

David G. Simons

This article considers specific treatment approaches and the role of etiological mechanisms in terms of clinical feature characteristics of MTrPs: increased muscle tension, pain and tenderness, painful stretch range of motion, initiating causes of MTrPs. Final sections note additional treatments that are currently used, and summarize the etiological and clinical distinctions between MTrPs and fibromyalgia.

Introduction

Truly effective treatment of neuromusculoskeletal pain often hinges on accurate identification of the sources of the pain. Myofascial trigger points (MTrPs) are unbelievably common yet commonly overlooked or poorly treated because the initial training of so few medical practitioners includes adequate instruction in the identification and treatment of MTrPs.

Although this article concerns primarily treatment of MTrPs, it is important to appreciate how remarkably common MTrPs are and how often they are a major cause of a patient's musculoskeletal pain complaint. Table 1 lists a number of commonly used diagnostic terms

Table 1 Widespread Nature of MTrPs

Common diagnoses	Common trigger point Causes
Tension-type headache	Sternocleidomastoid, upper trapezius, posterior cervical, and temporalis
Frozen shoulder	Subscapularis, supraspinatus, pectoralis major and minor, deltoid muscles
Epicondylitis	Finger and hand extensors, supinator, and triceps brachii
Carpal tunnel syndrome	Scaleni, finger extensors
Atypical angina pectoris	Left pectoralis, major intercostals
Lower back pain	Quadratus lumborum, iliopsoas, thoracolumbar paraspinals, rectus abdominis, piriformis, gluteus maximus and medius

David Simons
3176 Monticello Street, Covington, GA 30014, USA.

Correspondence to: D. Simons
Tel: +1 770 786 3540; Fax: +1 770 786 0000;
E-mail: loisanddavesimons@earthlink.net

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Box 1 Diagnostic features of active MTrPs [based on Simons et al. 1999—The Trigger Point Manual, Vol. 1, Ed. 2 including table 2.4B p. 25]

History
- Regional aching pain complaint–onset related to acute, chronic, or repetitive muscle overload.
- Pain intensity usually related to movement or positioning but may become continuous when severe

Diagnostic findings
- Painful limit to stretch range of motion
- Palpable taut band with exquisitely tender nodule
- Pressure on tender spot elicits pain familiar to patient and often a pain response (jump sign)

Confirmatory findings
- Local twitch response induced by snapping palpation or needle penetration of MTrP and evidenced by movement detected by vision, palpation, or ultrasound imaging
- Pain or altered sensation in expected location (published referred pain patterns for that muscle)
- Demonstration of endplate noise from an electromyographic needle gently inserted into the MTrP

that only identify the region of pain, but not its cause. These diagnostic terms are not diagnoses. Identification of MTrPs that are causing a patient's pain is a diagnosis.

As outlined in Box 1, the likelihood of a trigger point (TrP) causing the patient's pain should be established by taking a careful history of the onset and course of the pain. The presence of the TrP should be established by diagnostic findings of the physical examination whenever possible. The strength of the diagnosis can be reinforced by confirmatory findings, some of which are also valuable for research purposes.

Box 2 Myofascial trigger points can be inaccessible to palpation because of:

- Layer(s) of fat
- Intervening muscles(s)
- Intervening aponeuroses
- Tense, thick subcutaneous tissue
- Inadequate palpation skill

Box 2 lists reasons why MTrPs may be inaccessible to palpation for diagnostic purposes, making it necessary to rely on clinical judgment.

Box 3 identifies features of MTrPs that clarify how to distinguish them from other conditions.

Fortunately, the kinds of pathophysiological mechanisms responsible for MTrPs are becoming clear and can explain why some therapeutic approaches are so effective, and why others are of limited benefit for this condition.

Box 3 Distinguishing features of MTrPs

- MTrPs produce specific regional pain complaints and *NOT* widespread, total body pain and tenderness
- For practical purposes, only MTrPs are points of spot tenderness in a palpable taut band
- Not all tender points are MTrPs
- All MTrPs are tender points (points of spot tenderness)
- Referred tenderness as well as referred pain is characteristic of MTrPs
- All MTrPs have a taut band
- Not all taut bands are palpable (requires sufficient palpation skill and accessibility)
- All *active* MTrPs cause a clinical pain (sensory disturbance) complaint
- Only an *active* MTrP when compressed reproduces the clinical sensory symptoms. A *latent* MTrP produces no clinical sensory (pain or numbness) complaint.

The concept of etiology used here is based on the endplate (integrated) hypothesis that is fully described in two books (Simons et al. 1999, pp. 69–78; Mense et al. 2001, pp 240–259). This is a valuable working hypothesis for understanding current clinical practice and future research. Many details remain to be refined or filled in.

Increased muscle tension

Clinicians have recognized for more than a century that effective treatment of painful, tense, tender muscles includes stretching the involved muscle fibers, either locally in the region of tenderness (massage) or by lengthening the muscle as a whole. Frequently MTrPs were the cause of the symptoms and were what was being treated.

The reason that muscles with MTrPs feel abnormally tense on palpation and have a reduced stretch range of motion is the increased tension of the palpable taut band that is associated with the MTrP. This palpable increase in muscle tension is commonly mistaken for muscle spasm. Muscle spasm is clearly identified by motor unit activity that is identified electromyographically (Simons & Mense 1998) while taut bands show no motor unit activity at rest.

The problem

The increased tension of the palpable taut band is the result of regional shortening of the sarcomeres of numerous involved muscle fibers in the taut band. One demonstrated cause of that shortening of an affected muscle fiber is the presence of a contraction knot (Simons & Stolov 1976) that would be in the region of a motor endplate (Simons 1999, Mense et al. 2001). The sarcomeres in the contraction knot appear maximally

contracted (hypercontracted), while remaining sarcomeres of an involved muscle fiber are noticeably stretched to compensate for the missing length of the shortened sarcomeres. Due to the nature of titin – the spring-like molecule that holds the myosin molecules in place – the maximally contracted sarcomeres would tend to become stuck in this shortened position. The effect of these shortened sarcomeres is one likely source of increased resting tension in an involved muscle fiber. This effect is transmitted through the fiber as increased resting tension of the stretched sarcomeres (Wang 1985, 1996). The central MTrP is located close to the middle of the muscle fiber where the motor endplates are found. The primary goal of manual therapy treatment is to restore uniform sarcomere lengths in the affected muscle fibers that have developed unequal sarcomere lengths as illustrated in Figure 1.

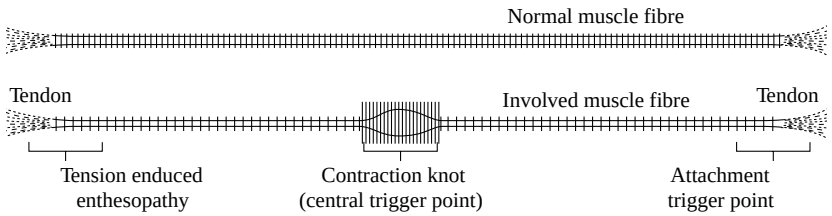

Fig. 1 Unequal sarcomere length: Comparison of the uniform, equal length of all sarcomeres of a normal muscle fiber compared to the severe shortening of a group of sarcomeres near the center of the fiber and the compensatory lengthening of the remaining sarcomeres. Note locations of central and attachment trigger points.

Solutions

Figure 2 illustrates why a *gentle* muscle contraction tends to equalize sarcomere length in fibers afflicted with the dysfunction that is characteristic of MTrPs. The relatively few (about 400 in a dog biopsy [Simons & Stolov 1976]) maximally contracted sarcomeres of the contraction knot are already as short as they can get. Therefore, based on the sarcomere length-tension curve, those sarcomeres are unable to produce any additional tension. On the other hand, the much larger number, about 20 000, in a 3-cm (1 1/2-inch) long muscle fiber (assuming 1.5 μm mid-range sarcomere length – 10 000 μm/cm; 6666 sarcomeres/cm × 3 cm = 20 000 sarcomeres/3 cm muscle fiber) is a ratio of 1:50 between the number of shortened contraction-knot sarcomeres and the number of lengthened sarcomeres. The shortened sarcomeres are greatly outnumbered. The remaining, somewhat stretched, sarcomeres in that fiber are still in the optimal range of their length-tension curve. Therefore, the fully shortened, weak sarcomeres are readily overpowered by the remaining strong sarcomeres during a gentle voluntary contraction (see Fig. 2).

Time can be a critical factor. Because of the stickiness of the titin molecules, releasing them can be expected to take time, and clinical experience confirms that slowly sustained stretches are much more effective at releasing MTrP tightness than rapid brief stretches. As the shortened sarcomeres lengthen, the stretched sarcomeres now return toward normal length.

Unfortunately, as soon as the muscle relaxes, the sarcomeres immediately tend to return to their previous state unless something more is done.

Figure 3 presents schematically the essence of postisometric relaxation or contract–relax. Contraction alone is not adequate treatment. The gentle (approximately 10% of maximum) contraction is immediately followed by relaxation and movement to take up slack that develops in the muscle. Better terminology might be 'postisometric relaxation and release' or 'contract–release'. The term '*stretch*' could be used in place of '*relax*' or '*release*' but that term has been avoided because it is so commonly associated with forceful movement and this technique should be a painless, gentle one. Figure 3 illustrates the stepwise nature of the process.

Methods of release

The relaxation and release phase can be accomplished in a number of ways. Immediate elongation of the muscle encourages equalization of sarcomere lengths throughout the length of affected muscle fibers, and when done slowly helps to reset the new sarcomere lengths so they tend to stay that way. This principle for effectively releasing muscle tension

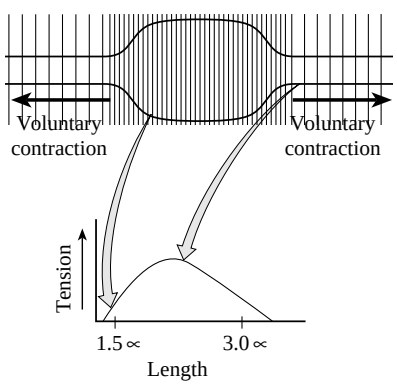

Fig. 2 Voluntary contraction. The length–tension curve below shows that the relatively few (roughly 1 in 50 but maximally contracted sarcomeres of contraction knots are in a range where they can no longer exert any additional contractile force because they are already maximally shortened. On the other hand, the much larger number of slightly elongated sarcomeres between the trigger point and the muscle fiber attachments are at, or close to, the optimal strength portion of the curve. Therefore, gentle voluntary contraction allows the lengthened sarcomeres to exert an effective elongation force on the shortened sarcomeres of the contraction knot.

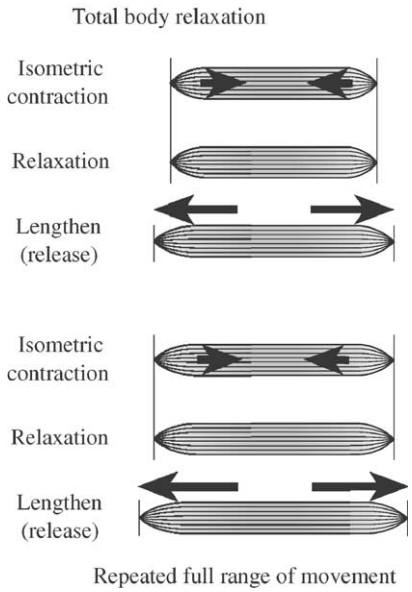

Fig. 3 Postisometric relaxation (contract–release). The essence of many effective myofascial trigger point release techniques is to repeat a series of incremental releases each of which are achieved by a gentle voluntary contraction of the muscle that is followed by active or passive elongation of the muscle to restore full pain-free range of motion and to inactivate its trigger points. Other names that are used for this principle of treatment include muscle energy techniques, and contract–relax.

(and for release of restricted joint movement that is often caused by muscle tightness) has been recommended by various schools of thought with many names, such as 'contract–relax', 'postisometric relaxation', and 'muscle energy technique'. In any case, full relaxation of the patient is an essential prerequisite to effective release.

Release can be achieved passively or actively. In passive release, the movement is done for the patient; in active release it is done primarily by the patient. Lewit (1991) emphasizes the advantage of using gravity for passive release. When properly positioned for appropriate muscles, gravity is gentle and helps the patient relax more completely. Much operator skill is required to sense the tissue resistance that indicates how much movement is optimal. Too little movement is ineffective and too much movement, too fast causes pain that inhibits release.

With active release, the patient extends the muscle to take up the slack by actively contracting its antagonist muscles. This has the advantages of adding the effect of reciprocal inhibition to facilitate release of the muscle being elongated. Many patients can learn quickly to optimize the effort, but must understand what to do, why to do it, and have the innate capacity to learn to do it effectively. It can be a powerful tool for a patient's home program. This technique is particularly useful for clinicians to treat their own MTrPs and combines nicely with contract–release.

Figure 4 illustrates 'trigger point pressure release' that is performed by simply applying gentle persistent digital pressure against the palpable tissue barrier in the MTrP. A sarcomere is a space of constant volume. Like a balloon, if its height is reduced by compression, it becomes wider. This applies to the sarcomeres of a contraction knot. By applying digital pressure in a way that reduces their height, they must become longer, tending to normalize the length of all sarcomeres in that muscle fiber. The sticky, overcompressed titin molecules resist elongation of the short sarcomeres.

Again, if compression is applied for only a short period of time and then released, the shortened sarcomeres tend to return immediately to their previous state and little has been gained. However, if gentle compression is sustained until the clinician feels the release of tension, this corresponds to a degree of equalization of sarcomere length that can be demonstrated as an increased range of motion and reduced muscle tension. This is the essence of the 'trigger point pressure release' technique and can be continued as long as additional release of tension occurs.

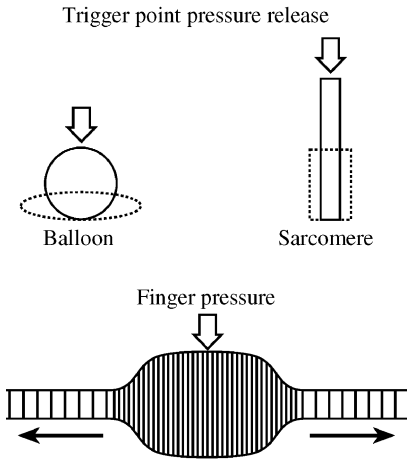

Fig. 4 Trigger point pressure release. Demonstration of the principle responsible for the effectiveness of this treatment method. Individual sarcomeres, like a balloon are constant-volume structures. Compressing either one of them in the vertical dimension cause an increase in the horizontal dimension. Thus, finger pressure applied downward on a MTrP tends to lengthen sarcomeres that are shortened for any reason and can be responsible for the tension of the taut band.

Figure 5 demonstrates combined finger pressure and voluntary contraction. The combination of applying firm, gentle, persistent TrP pressure release and sustained gentle voluntary contraction adds the impact of two effective ways of equalizing the sarcomere lengths and releasing muscle tightness. This combination is most effective when

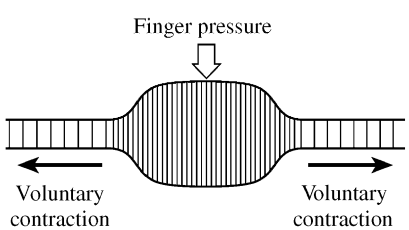

Fig. 5 Finger pressure and voluntary contraction. This figure illustrates how the effects of simultaneous application of trigger point pressure release and voluntary contraction are additive.

it begins with the muscle in a comfortably stretched position. The muscle is gently extended until no slack remains. Following each period of gentle contraction, the muscle is immediately gently extended to take up the slack that was produced by treatment. This helps the sarcomeres to retain their new lengths and establishes a new starting point for the next release cycle.

Figure 6 shows trigger point massage – a TrP-specific variation of deep massage that is particularly helpful in equalizing sarcomere length. It elongates the contraction knot sarcomeres, allowing the remaining stretched sarcomeres to shorten a bit. This massage is performed with the muscle extended to just eliminate slack. Then the clinician starts by simultaneously applying bimanual digital pressure on the middle of the MTrP, short fingernail against short fingernail, and then pulls the fingers apart while maintaining gentle, firm pressure.

Pain and tenderness

The local tenderness of the MTrP and its ability to refer pain to distant location depends primarily on the sensitization of nociceptors in the immediate region of the endplates

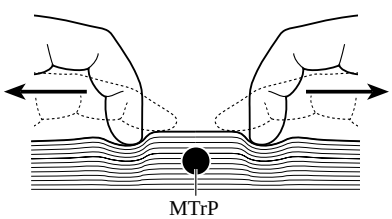

Fig. 6 Trigger point massage. This massage technique is specific for trigger points and requires two hands. It is an extension of trigger point pressure release and can be even more effective. It is designed to effectively lengthen the shortened sarcomeres of contraction knots in a myofascial trigger point and should be applied with the muscle extended to eliminate any slack. Concept by C-Z. Hong, MD.

that are associated electromyographically with endplate noise. Endplate noise indicates abnormal endplate function. (Simons 2001). Endplate noise is characteristic of MTrPs. (Simons et al. 2002; Couppe et al. 2001). Figure 7 outlines the basis of the energy crisis responsible for release of substances that sensitize local nociceptors. This positive feedback cycle starts (upper left corner) with resting release of grossly abnormal amounts of acetylcholine (ACh) from the nerve terminal of the motor endplate (Simons 2001). This leads to contraction knots and possibly other contractile effects that cause local ischemia and hypoxia (Brückle et al. 1990), which are combined with a greatly increased energy consumption (local maximal sarcomere contractile activity). This combination of greatly increased sustained energy demand and loss of energy supply creates an energy crisis that causes release of

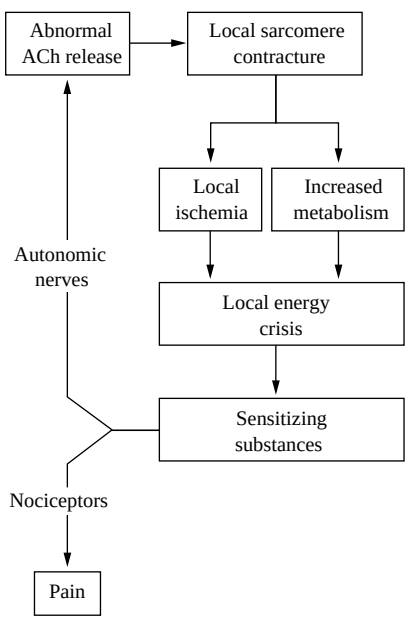

Fig. 7 Energy crisis. Schematic diagram of steps involved in the positive feedback loop of the end plate integrated hypothesis. It shows the origin and results of the energy crisis characteristic of MTPs. See text for explanation.

neurovasoreactive sensitizing substances (Mense et al. 2001, pp 37–40). Any treatment that reduces the sarcomere shortening in the region of the MTrP reduces the energy consumption, which in turn reduces the release of sensitizing substances.

The degree to which the released sensitizing substances get to actually sensitize nociceptors depends strongly on the closeness of the nociceptors to an affected motor endplate and that depends on variations in local anatomical structure. Generally the endplate and nociceptors are near each other, but not always. This is why MTrPs are primarily a motor dysfunction disease and only secondarily a pain phenomenon. The pain results secondarily from the endplate motor dysfunction. This helps to explain why there is only a general correlation between the motor expression of a MTrP (the taut band) and its degree of painfulness and why latent MTrPs are so much more common than active ones.

It is well established (Simons et al. 1999, p. 75, Lewis et al. 1994; McNulty et al. 1994; Chen et al. 1998) that variations of local autonomic activity can affect acetylcholine release at endplates. Activation of local autonomic nerve fibers by the neurovasoreactive substances would complete the feedback loop and induce continued excessive release of acetylcholine by the motor endplate. This also explains why anxiety and nervous tension that increases autonomic activity commonly aggravates symptoms caused by MTrPs.

Painful stretch range of motion

A valuable clinical indicator of MTrPs is painful restriction of stretch range of motion of the involved muscle. The pain is caused by increased tension on attachment

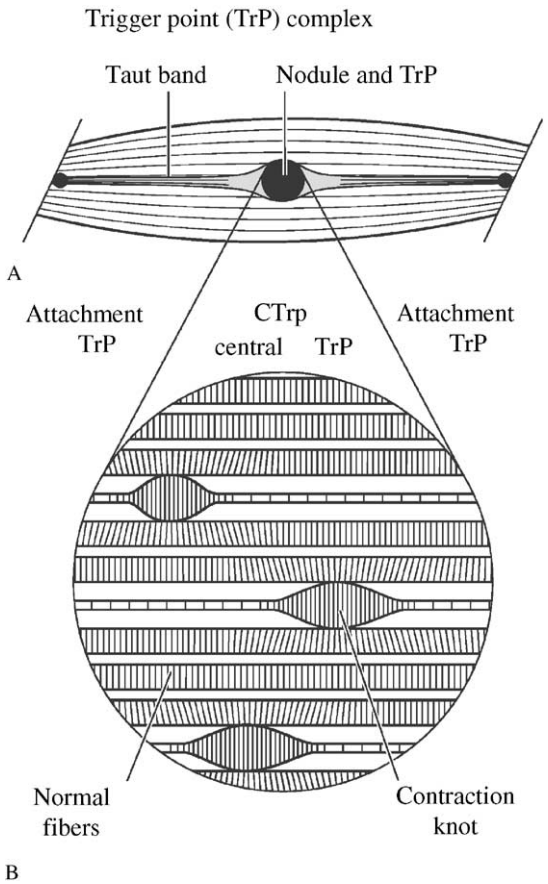

Fig. 8 Relation between central and attachment trigger points. (A) Schematic diagram looking down on a fusiform muscle such as the biceps brachii. The central trigger point (*encircled* ●) is found close to the center of the muscle fibers that form a palpable taut band (*dark fibers*). Attachment trigger points (*open circles)* are tender myotendinous attachments of taut band fibers due to enthesopathy (inflammatory reaction) caused by the sustained tension of the taut band fibers. That tension is caused by and can be relieved by normalizing, the contraction knots of the central trigger point. (B) Schematic diagram of enlargement of a microscopic part of the trigger point. The fusiform enlargements of involved muscle fibers are produced by local maximal contraction of the sarcomeres that seem to be located in the region of a motor endplate. Note that the remaining sarcomeres of the involved fiber are longer compared to the normal, uninvolved fibers.

TrPs. During examination, it is frequently more convenient to examine first for an attachment TrP rather than the central TrP. Attachment TrPs are more accessible and more readily identified than central MTrPs, especially in muscles such as levator scapulae, gluteus medius and iliopsoas.

Figure 8A explains the relation between the central TrP and attachment TrPs. The central TrP causes the tension of the taut band that produces the attachment TrP. The taut band imposes sustained abnormal tension on the myotendinous junctions where the muscle fibers attach to the muscle tendon. These attachments are complicated delicate structures that are not designed to tolerate unrelieved, constant tension. Muscles normally contract then relax, contract then relax, with appreciable periods of relief from tension between contractions. This abnormal tension induces inflammatory changes (enthesopathy) that often include some edema, release of pain-inducing sensitizing substances and eventually some degenerative changes (Fassbender 1975) that account for the attachment TrP.

Clinically, the attachment TrP can be identified by local spot tenderness in the region of the myoneural junctions, sometimes referral of familiar pain when digitally compressed, and sometimes palpable induration. Frequently the examiner can identify a palpable taut band extending toward the central TrP that is responsible for the attachment TrP.

Initiation of trigger points

Acute or chronic muscle overload initiates the excessive release of acetylcholine, which produces the local energy crises that can account for the clinical characteristics of MTrPs. The motor endplate is a synaptic junction with basically the same delicate structure (Fig. 9) as that of the myriad of synapses that conduct the business of the central nervous system with one very important difference. The central nervous system synapses are cushioned from mechanical trauma by suspensory ligaments encased in a protective bony cage. The motor endplates are exposed to all the mechanical stresses and strains that beset the muscular system, but with no special protection.

It is little wonder that, with normal use, it is only a matter of time until the muscles develop the disease caused by mechanical trauma, myofascial trigger points. The pioneer muscle physiologist, Liley, in 1956 observed that almost any kind of mechanical stimulation of the endplate induced the same kind of electrical activity that nearly 20 years later was proven by another physiologist team to be caused by excessive release of acetylcholine

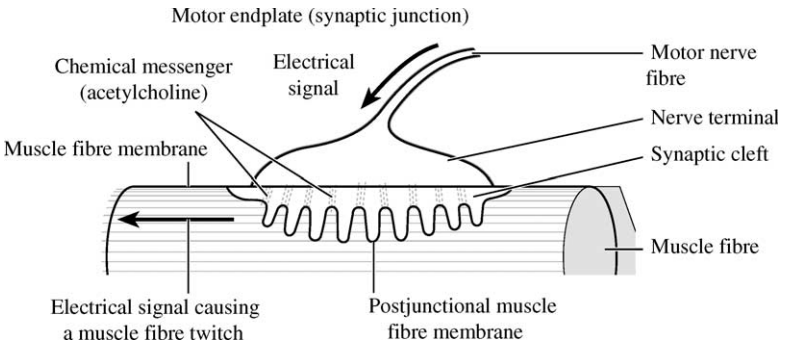

Fig. 9 Motor endplate synaptic structure and function. Schematic cross-section of the terminal nerve fiber, synaptic cleft, and postjunctional muscle fiber membrane. It also shows how the electrical signal of the motor nerve is normally converted to a chemical signal and then back to an electrical signal that is propagated along the muscle fiber membrane in both directions. The original electrical signal (propagated action potential along the nerve membrane) causes the presynaptic nerve terminal to simultaneously release *many packets* of acetylcholine that diffuse across the synaptic cleft. On arrival at the postjunctional membrane of the muscle cell, this chemical messenger is converted back to an electrical signal when the acetylcholine activates acetylcholine receptors located in the postjunctional membrane. Normally just a few packets per second of acetylcholine are released randomly under resting conditions.

(Heuser & Milelli 1971; Ito et al. 1974). This endplate noise is the same kind of electrical activity that is characteristic of MTrPs (Simons et al. 2002). Liley (1956) reported that once it was initiated, this dysfunction persisted, which is also characteristic of MTrPs. The classical history of onset of myofascial pain caused by MTrPs is either an acute muscular overload (sometimes due to an accident, poor coordination when fatigued, or an awkward movement) or chronic overload caused by sustained muscle contraction or by monotonous repetition of the same movement.

Box 4 Additional ways of treating MTrPs

- Techniques to augment release (exhalation, eye movement)
- Reciprocal inhibition (voluntary contraction of antagonist muscle)
- Dry needling (acupuncture technique)
- Therapeutic ultrasound
- Strain–counterstrain
- Microcurrent application
- Laser irradiation
- Injection

Additional treatments

Box 4 lists additional treatments that some clinicians find effective in treating MTrPs. Augmentation techniques are well described by Lewit (1986,1991) and by Simons et al. (1999). Reciprocal inhibition is described as such by Simons et al. (1999) and by Lardner (2001). The value of dry needling has been reported by Hong (1994) and Baldry et al. (2001). The value of therapeutic ultrasound, especially with regard to patients with fibromyalgia was reported by Lowe et al. (1999). Although the mechanisms described to explain their clinical effectiveness are not convincing, many clinicians find strain–counterstrain, microcurrent application, and laser irradiation useful forms of treatment for MTrPs (Simons et al. 1999). Injection of the MTrP itself with an analgesic medication such as procaine or lidocaine are well-established techniques commonly used by physicians (Simons et al. 1999).

Occasionally, as pointed out by Thompson (2001) the taut bands of MTrPs may be serving a useful function as a stabilizing mechanism. In this case it is wise to correct the underlying cause of instability *before* releasing the MTrP tension. In fact, correcting the underlying instability often results in spontaneous resolution of the MTrP (Thompson 2001).

Fibromyalgia and trigger points

Table 2 summarizes features that distinguish MTrPs from fibromyalgia. Recent advances now make it clear that myogenic MTrPs and central nervous system fibromyalgia syndrome (FMS) are etiologically and pathophysiologically distinct conditions (Mense et al. 2001, Chapters 8 & 9). The regional-only pain of MTrPs can appear confusingly like the widespread pain of FMS when there is multiple regional involvement in one patient. The palpable taut band is a reliable distinguishing feature of MTrPs, when it is accessible (See Box 2).

Table 2 Distinguishing MTP MTrPs and fibromyalgia

MTrPs	Fibromyalgia
Peripheral muscular origin	Central nervous system origin
Female to male ratio 1:1	Female to male ratio 4–9 : 1
Local or regional pain	Widespread, general pain
Focal tenderness	Widespread tenderness throughout
Muscle feels tense (taut bands)	Muscle feels soft and doughy
Restricted stretch range of motion	Commonly hypermobile
Examination for MTrPs anywhere	Examination for prescribed tender points
Immediate response to MTrP injection	Delayed response to MTrP injection
May also have fibromyalgia	Nearly all also have MTrPs

Restriction of stretch range of motion is characteristic of MTrPs, not FMS, but most FMS patients also have MTrPs. Totally different examinations are required to make each diagnosis (Mense et al. 2001). MTrP injections respond differently in patients who do or do not also have FMS (Hong & Hsueh 1996). It is becoming increasingly clear that most, if not nearly all, FMS patients also have pain-generating MTrPs.

One important study of FMS patients (Donaldson et al. 1998) and two case reports (Starlanyl & Seffrey 2001; Donnelly in press) give remarkable insight into the close relationship between the two distinctly different conditions. The Donaldson paper calls attention to the importance of electroencephalogram (EEG) changes in FMS patients and raises the likely possibility that a software-type brain dysfunction is the critical dysfunction in FMS patients that disturbs normal functioning of all organ systems. Donnelly (in press) documents the repeated observation that some FMS patients are able to clearly distinguish between pain of fibromyalgia and that caused by MTrPs. FMS produces a generalized pain relatively independent of movement. MTrPs produce a regional deep aching pain and are more strongly related to muscular activity. The author personally knows a number of medically trained patients who describe convincingly their ability to distinguish the two sources of pain and then successfully initiate different remedial action for each.

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