REVIEW ARTICLE

Pathophysiologic and Electrophysiologic Mechanisms of Myofascial Trigger Points

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Objective: To review recent clinical and basic science studies on myofascial trigger points (MTrPs) to facilitate a better understanding of the mechanism of an MTrP.

Data Sources: English literature in the last 15 years regarding scientific investigations on MTrPs in either humans or animals.

Study Selection: Research works, especially electrophysiologic studies, related to the pathophysiology of MTrP.

Data Synthesis: (1) Studies on an animal model have found that a myofascial trigger spot (MTrS) in a taut band of rabbit skeletal muscle fibers is similar to a human MTrP in many aspects. (2) An MTrP or an MTrS contains multiple minute loci that are closely related to nerve fibers and motor endplates. (3) Both referred pain and local twitch response (characteristics of MTrPs) are related to the spinal cord mechanism. (4) The taut band of skeletal muscle fibers (which contains an MTrP or an MTrS in the endplate zone) is probably related to excessive release of acetylcholine in abnormal endplates.

Conclusion: The pathogenesis of an MTrP appears to be related to integrative mechanisms in the spinal cord in response to sensitized nerve fibers associated with abnormal endplates.

A MYOFASCIAL TRIGGER point (MTrP) has been defined as a highly localized and hyperirritable spot in a palpable taut band of skeletal muscle fibers.[1-4] There is agreement on the following common clinical characteristics of MTrPs among many authors.[3-19] (1)Compression of an MTrP may elicit local pain and/or referred pain that is similar to a patient's usual clinical complaint (pain recognition) or may aggravate the existing pain. (2) Snapping palpation (compression across the muscle fibers rapidly) may elicit a local twitch response (LTR), which is a brisk contraction of the muscle fibers in or around the taut band. Rapid insertion of a needle into the MTrP can also elicit an LTR. (3) Restricted range of stretch, and increased sensitivity to stretch, of muscle fibers in a taut band may cause tightness of the involved muscle. (4) The muscle with an MTrP may be weak because of pain, but usually no remarkable atrophy can be noticed due to the waxing and waning phenomena of the MTrP. (5) Patients with MTrPs may have associated localized autonomic phenomena, including vasoconstriction, pilomotor response, ptosis, and hypersecretion. (6) An active MTrP is one with spontaneous pain or pain in response to movement, whereas a latent MTrP is a sensitive spot with pain or discomfort only elicited in response to compression.

The reliability of MTrP examination has been strongly criticized by Bohr,[20] based on two recent studies by Wolfe and colleagues [21] and Nice and associates.[22] In these two studies, unsatisfactory kappa values (.38 and .35, respectively) were obtained. In both studies, however, MTrP examination was performed by untrained examiners. In another study, a marginal kappa value of .49 was reached by well-trained examiners.[23] Recently, Gerwin and coworkers[24] reported a study on the reliability of MTrP examination of five muscle pairs in 10 subjects by four experienced physicians who had a 3-hour training session immediately before the study. The reliability of identification of certain features of the MTrP varied among muscles. In general, agreement among examiners was highest for the reproduction of the subject's symptomatic pain (pain recognition) (kappa values .79 to 1.0). Agreement was good to excellent on the presence of referred pain (kappa values .57 to .84) and was moderate to excellent for the detection of spot tenderness or a taut band (kappa values .4 to 1.0). However, agreement varied on the presence or absence of an LTR in different muscles (kappa values from .11 to 1.0), depending on the degree of difficulty in examination. The investigators concluded that it is essential to have hands-on training to achieve a reliable MTrP examination. It appears that "spot tenderness," "pain recognition," and "taut band" are the most reliable signs and the minimal criteria needed to identify an MTrP, while "referred pain" and "local twitch response" are most useful as confirmatory signs of the MTrP.[24,25]

MTrPs are characteristic of myofascial pain syndrome. Myofascial pain syndrome can be associated with other neuromusculoskeletal disorders[10,11,14,15,26] and can be perpetuated or aggravated by conditions such as mechanical stress, metabolic inadequacies, or psychological factors.[3,7,12,27]

The pathophysiology of the MTrP has become better understood as a result of recent research studies, mainly electrophysiologic, on both human and animal subjects. Clarification of the MTrP mechanism is essential to understand MTrPs more clearly and treat them more effectively.

This article reviews clinical and basic science research in humans or animals related to the pathophysiology of MTrPs. Data sources are selected from the English literature in Medline databases for the past 15 years and references identified from bibliographies of pertinent articles and books. Only studies with appropriate controls and statistics were critically considered to have contributed to knowledge of the MTrP mechanism; others may be mentioned as supportive evidence.

ALGOMETER STUDIES ON HUMAN MTrPs

It has been suggested that a pressure algometer can assist in location of MTrPs and in documentation of their tenderness or relative sensitivity.[28-37] Previous studies have demonstrated the

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reliability and validity of the pressure algometer in measuring MTrP sensitivity.[38-40] However, it is important to place the tip of the algometer precisely on the MTrP region to avoid any major difference in readings among the consecutive measurements of the same MTrP.

In a recent algometry study,[41] pressure pain threshold, referred pain threshold (pressure threshold to elicit referred pain), and pain tolerance (maximal pressure that one can tolerate) were measured at three different sites in extensor digitorum communis muscles: the trigger point (MTrP site), a non-MTrP site in the same taut band (taut band site), and a control site in normal muscle tissue without taut bands. Twenty-four normal subjects with latent MTrPs and 15 patients with active MTrPs in the extensor digitorum communis muscles were studied. It was found that referred pain could be elicited from the MTrP site in all muscles with active MTrPs but only in 46.8% of muscles with latent MTrPs. Pressure over the taut band also elicited referred pain in all patients with active MTrPs, but only in 36.2% of subjects with latent MTrPs. Even from the normal muscle tissue, referred pain could be elicited in 68% of patients with active MTrPs and in 23.4% of subjects with latent MTrPs. It appears that referred pain is not a specific sign of an MTrP, but it certainly occurs more often (and is much easier to elicit) in an active MTrP region than in a latent one or a normal muscle tissue. It is likely that referred pain threshold was higher than pain tolerance at some sites. Thus, in some cases, especially in latent MTrPs or in normal muscle tissues near active MTrPs, the pain tolerance level was reached before obtaining the referred pain threshold.

It was also found that the referred pain threshold was highly correlated ($r = .88$) with the pain threshold in patients with active MTrPs.[41] Such correlation, however, was lower ($r = .62$) in subjects with latent MTrPs than in patients with active MTrPs. The difference between the pain threshold and the referred pain threshold was less in active MTrPs than in latent MTrPs or normal tissues. Therefore, the more active an MTrP (low pain threshold), the less pressure was required to elicit referred pain (low referred pain threshold). It appears that the referred pain threshold was related to the degree of *irritability* (sensitivity to mechanical stimulation) of the site being compressed.

By using a palpometer (a type of algometer), Bendtsen and coworkers[42] studied the pain intensity of myofascial tissue in response to pressure in 40 patients with chronic myofascial pain as compared to 40 controls. It was found that the stimulus-response function was linear in the 20 most tender patients, but pain intensities increased in a positive accelerating fashion with increased pressure intensities in the 20 least tender patients and in control subjects. Based on these findings, the investigators suggested that myofascial pain might be mediated by the low-threshold mechanosensitive afferents projecting to sensitized dorsal horn neurons, instead of transmitted through the high-threshold mechanosensitive afferents as in normal cases.

RESEARCH ON THE INJECTION OF MTrPs

Effectiveness of Dry Needling to Inactivate an MTrP

Trigger point injection with local anesthetics[3,5,10,11,43] or with sterile saline or water[44-47] has been recommended for inactivation of an MTrP. In a noncontrolled study, Lewit[48] reported that 271 (86.8%) of 312 painful structures were anesthetized immediately after treatment with dry needling. In 92 of those 271 structures, permanent pain relief was obtained after dry needling. In an abstract, Jaeger and Skootsky[46] also briefly reported the effectiveness of dry needling in reducing local tenderness of MTrPs based on a double-blind, controlled study.

By using an MTrP injection technique with a "fast-in, fast-out" needle movement, it was found that a significant and immediate pain relief could be achieved after MTrP injection only if LTRs were elicited during injection.[49] The rapid insertion of the needle tip into a sensitive, painful site in MTrP region may facilitate the elicitation of an LTR that may not be observed if the needle tip approaches that site slowly. Using a similar technique of MTrP injection, Hong[50] compared the effectiveness of lidocaine injection and dry needling to treat MTrPs of upper trapezius muscles under a blind, controlled study. It was found that in both lidocaine injection and dry needling, the degree of subjective pain relief, elevation of pain threshold, and increase of range of motion were significantly greater when LTRs were elicited during needle insertion than when LTRs were not elicited. The immediate effectiveness of lidocaine injection was not significantly different from that of dry needling. It appears that it is essential to elicit LTRs during MTrP injection to obtain an immediate relief of pain and tightness.[10,11,49-51]

It has been suggested that precision in needling of the site with maximal pain in an MTrP region is the major factor in MTrP inactivation.[3,5,48] Eliciting an LTR is considered an important sign of precise needle insertion into a MTrP region. Since an MTrP can be inactivated without injection of any solution, the mechanical stimulation of the MTrP may be the most important factor for pain relief. The minute sensitive site from which an LTR can be elicited by needle stimulation (such as dry needling or injection of an MTrP) has been defined as a *sensitive locus* in an MTrP region.[11]

Many authors have documented the similarity between acupuncture and dry needling in treating MTrP.[10,11,48,50,52-55] The "Teh-Chi" effect,[52] described by acupuncturists as an important sign for obtaining an optimal effect in acupuncture therapy, is similar to the feeling reported by the patient when the needle tip approaches a sensitive locus in an MTrP region during MTrP injection. The patients experiencing MTrP injection (or dry needling) may also have such a feeling at the moment when an LTR is elicited.[11] The acupuncture needle is usually moved slowly during treatment. LTRs would be less likely to be elicited by the slow needle movement of acupuncture therapy than by the rapid needle insertion of MTrP injection. In a study on the comparison between the locations of trigger points and acupuncture points, Melzack and colleagues[55] found a high degree (71%) of correspondence between these two points. Melzack[54] hypothesized that hyperstimulation analgesia to "close the gate" by the disruption of reverberatory neural circuits in the central nervous system was the mechanism of pain relief for both acupuncture and dry needling.

Referred Pain Elicited During MTrP Injection

In a recent study of patients who received MTrP injections, Hong and colleagues[56] compared the difference between the incidence of referred pain elicited by digital compression of MTrPs (before MTrP injection) and that elicited by needling during MTrP injection. It was found that needling could elicit referred pain in 223 (87.7%) of 243 MTrPs, while palpation elicited referred pain in only 131 (53.9%) MTrPs. It appears that referred pain can be elicited more frequently by needling than by palpation. It may require high pressure to elicit referred pain if the MTrP has low irritability.[26] Since the pressure induced by a small needle tip during MTrP injection is much higher than that induced by the finger tip palpation, the former method is expected to elicit more referred pain than the latter.

ELECTROPHYSIOLOGIC STUDIES ON HUMAN MTrPs

Spontaneous Electrical Activity of an Active Locus in an MTrP Region

In an electromyographic (EMG) study, Weeks and Travell[57] inserted a coaxial needle electrode into the trigger point area in a resting muscle and recorded high-frequency (10 to 12 per second) repetitive spikes with an amplitude of about 1,000 μV and a duration of 1 to 3 msec. However, there was electrical silence in the adjacent sites at the same time when the spikes were recorded from the trigger area.

In a later study, Kraft and colleagues[58] used a routine EMG technique and failed to record any EMG activity from a firm nodule at rest. Other authors[59,60] also reported absence of EMG activity at MTrP sites. Recently, Hubbard and Berkoff[61] demonstrated the presence of "spontaneous EMG activity" at minute sites ("nidus") in an MTrP region of the upper trapezius muscle and no such activity at adjacent nontender sites. Both spikes and continuous low-amplitude action potentials could be recorded from the "nidus" of an active MTrP. However, only low-amplitude action potentials could be found in latent MTrPs. In later studies by Simons and colleagues,[62-64] similar spontaneous and continuous low-amplitude action potentials (10 to 50 μV, occasionally up to 80 μV) were recorded from human MTrPs (either active or latent ones). This continuous low-amplitude activity was defined as spontaneous electrical activity (SEA) to distinguish it from the intermittent spike activity (100 to 600 μV, biphasic, initially negative) that could be recorded only from active MTrPs but not from latent MTrPs. The minute locus from which SEA can be recorded is now defined as an *active locus* of an MTrP.[25,62]

A special technique using high-sensitivity recordings and a very gentle insertion movement of the recording needle is required to record SEA. The recording needle should be moved slowly and gently (by fractions of a millimeter) during the search for SEA, because a fast movement may miss this small signal or may elicit an LTR instead. As the recording needle approaches the responsive locus where SEA can be recorded, the amplitude of SEA progressively increases and the seashell noise becomes louder during the needle movement.[62,63]

Simons and associates[62,63] found that SEA could be recorded more frequently in an MTrP region (which was always in the endplate zone) than in a non-MTrP control site. The waveforms of SEA correspond closely to previously published records and descriptions of endplate noise.[65,66] Therefore, SEA is probably one type of endplate potential, and the active loci of an MTrP are closely related to endplates.

EMG Activity of LTRs

EMG activity has been recorded from a tender spot in a palpable band, but not from adjacent muscle fibers without MTrP or taut band, in human skeletal muscle when the tender spot was stimulated by snapping palpation.[67,68] In another study on the EMG activity of LTR recorded simultaneously from the intramuscular needle electrodes and the cutaneous surface electrodes, Simons and Dexter[69] found that little or no electrical activity could be recorded from the overlying surface electrode, compared with the remarkable EMG activity recorded from the needle electrode inside the palpable taut band (containing the MTrP that was stimulated to elicit the LTR). Furthermore, after repeated stimulation, the EMG activity attenuated or even disappeared (due to the displacement of the recording needle) despite persistence of a grossly visible LTR, but could be restored by moving the needle back to the original recording site. In every case in their study, the taut band was electrically silent in the absence of MTrP stimulation and when the subject was relaxed. Based on these findings, it is reasonable to conclude that LTR occurs specifically in the muscle fibers of the taut band and specifically in response to the stimulation of the MTrP in the taut band.

In a case study, it was found that the EMG activity of LTR in the denervated muscle was remarkably reduced.[70] This finding indicates that the transmission of LTR depends mainly on the nervous system.

ANIMAL STUDIES ON MTrPs

Animal Model of MTrP: Trigger Spots and Taut Band in Rabbit Skeletal Muscle Fibers

In rabbit biceps femoris muscle, taut bands similar to those in human muscle have been identified by finger palpation.[71] When certain sensitive sites in a taut band were stimulated mechanically with a blunt metal probe (snapping or tapping) or by a needle, LTRs were observed. Rabbit LTRs are similar to human LTRs both in the characteristic of visible muscle twitching and in EMG recording. The most sensitive spot to elicit an LTR in a taut band in a rabbit was defined as a myofascial trigger spot (MTrS), which is equivalent to the human MTrP in many aspects.[71] The similarities and differences between the human MTrP and rabbit MTrS are summarized in table 1. If the MTrS is squeezed when the animal is awake, the animal usually responds to this noxious stimulation as if it suffers pain or discomfort. This animal model can provide useful studies on the pathophysiology of MTrP.[71]

SEA Recorded From a Trigger Spot of Rabbit Skeletal Muscle

Using the animal model of rabbit MTrS and the same technique for human SEA recording, Simons and coworkers[72] also recorded SEA (with similar morphology as human SEA) from the active loci of MTrS in rabbit skeletal muscle. In 14 paired examinations of MTrS and control regions, SEA was searched from 24 needle advances (in 3 tracks, 8 advances per track) for each examination. SEA was observed at 70 loci in MTrS regions and at 15 loci in control regions. The difference was statistically significant. Intermittent bursts of spike activity were also observed in rabbit SEA.

In a previous rabbit study by Wiederholt,[73] electrical activity similar to SEA was recorded and was confirmed as "endplate noise" based on further histologic and pharmacologic studies. Normal endplate potentials are discrete, short, and negative monophasic miniature action potentials occurring only several times per second. However, the low-amplitude continuous discharges (similar to our SEA) have been identified as abnormal endplate potentials by neurophysiologists.[74,75] Liley[74] illustrated the conversion of the normal discrete negative monophasic potentials to abnormal continuous noiselike action potentials, similar to the SEA, by applying mild mechanical stimulation to the terminal nerve fiber or to the endplate region. Ito and associates[75] demonstrated that this abnormal pattern of endplate potentials was attributed to excessive release of acetylcholine packets. It has been suggested that the SEA found in an MTrS region corresponds to an abnormal pattern of endplate electrical activity resulting from excessive acetylcholine leakage.[25,72] It appears that the MTrP mechanism would relate strongly to dysfunction of endplates.

In a more recent study on rabbit skeletal muscle,[76] iron deposition (by applying DC current, 50 to 100 μV, for 90 sec) was performed at the active locus of MTrS where SEA was

Table 1: The Human MTrP and the Rabbit MTrS

	MTrP	MTrS
Site	Human skeletal muscle	Rabbit skeletal muscle
Identification		
Pain complaint & tender spot	Precisely located	Depends on responses to noxious stimuli when the animal is awake.
Taut band	Palpable	Palpable
Referred pain	Subjectively described	Unknown
Twitch response	LTR elicited by mechanical stimulation (snapping palpation or needling) of MTrP (but not other sites).	Rabbit LTR elicited by mechanical stimulation (snapping palpation, tapping, or needling of MTrS (but not other sites).
Electrophysiologic studies		
SEA	Recorded from active locus of an MTrP, but not from non-MTrP sites.	Recorded from active locus of an MTrS, but not from non-MTrS sites.
LTR	EMG activity of LTR recorded from taut band (containing MTrP) but not from other sites.	EMG activity of rabbit LTR (similar to human LTR) recorded from taut band (containing MTrS) but not from other sites.
	Diminished in denervated muscle	Diminished in denervated muscle
	Diminished in flaccid muscles of spinal cord injury patient during spinal shock stage (?)	Temporally diminished after spinal cord transection for 2½ hours (spinal shock period)

recorded. It was found that small nerve fibers (probably nociceptive nerve endings) were in the vicinity of iron-stain spots (the site of recording needle tip). This result is similar to that found in an earlier histologic study of a type of "insertion activity" (morphologically similar to SEA).[77] Therefore the active locus in an MTrS region is probably related to nociceptors.

EMG Activity of LTR Elicited by Mechanical Stimulation of a Trigger Spot in Rabbit Skeletal Muscle

To study the nature of rabbit LTR, a solenoid-driven device was designed to deliver tapping stimulation of MTrS. This device could synchronously trigger the oscilloscope screen and the mechanical stimulation from a metal probe. The onset latency of rabbit LTR was approximately 20 to 30msec after tapping stimulation. The mean duration was 86.6 ± 16.4msec. Based on these data, it was suggested that LTR in the rabbit was a polysynaptic reflex.

When an MTrS was stimulated, the EMG activity of rabbit LTR could be recorded from the specific taut band containing that MTrS but not from the adjacent muscle fibers.[71] From the recordings at the same site in a taut band, rabbit LTRs were best recorded when the MTrS itself was stimulated as compared to that elicited by stimulation of other sites near the MTrS.[71] These two important findings in the rabbit study are similar to those in human LTR studies.[69]

EMG activities were also recorded simultaneously from the needle for stimulation (insertion into the MTrS site) and from the needle for recording (static needle at 2 cm away from the MTrS in the same taut band).[71] The mean duration of EMG activity recorded from the static (recording) needle electrode was significantly longer than that recorded from the moving (stimulating) electrode. The morphology of EMG activity was also different between these two recordings. Therefore, rabbit LTR recorded from the static needle was not the insertional activity elicited from the moving needle. Similar to the human subject, the minute site from which an LTR can be elicited in the rabbit has also been defined as a *sensitive locus.*[71]

The EMG activity of rabbit LTRs essentially disappeared after lidocaine block or after transection of the innervating nerve.[71,78] This activity disappeared temporarily after spinal cord transection during the spinal shock period, but nearly completely recovered following spinal shock.[78] It indicated that the rabbit LTRs were mainly mediated through the spinal cord and supraspinal structures were not essential components. Therefore, LTRs are probably mediated reflexly through the spinal cord.

A recent histologic study on rabbit skeletal muscle demonstrated that a small nerve fiber was frequently found near the sensitive locus from which a rabbit LTR was elicited.[79] Similar to the active locus, the sensitive locus in an MTrS region is probably also related to nociceptors.

Animal Studies on Referred Pain Mechanism

Hoheisel and coworkers[80] reported a study of the referral of muscle pain. The receptive field properties of single dorsal horn neurons of Sprague-Dawley rats were assessed electrophysiologically. The mechanical threshold of a high-threshold mechanosensitive dorsal horn neuron was determined by recording the electrical activity of the dorsal horn neuron in response to the mechanical stimulation of its original receptive field in the biceps femoris muscle. Noxious deep-pressure stimulation to the biceps femoris muscle was required to activate this high-threshold neuron. Bradykinin was then injected into the tibialis anterior muscle (outside the original receptive field). New receptive fields at the injection sites or at distal sites could be induced about 5 minutes after injection. It required noxious stimulation on the new receptive fields to activate the dorsal horn neuron corresponding to the receptive field of biceps femoris muscle. Fifteen minutes after injection, the mechanical threshold of the original receptive field in biceps femoris muscle also reduced. Therefore, innocuous stimulation to the original receptive field could activate the dorsal horn neuron. In the control studies (without injection of bradykinin), repeated searching for receptive fields with noxious stimuli did not change the size or number of receptive fields. This study demonstrated the development of new receptive fields outside

the original receptive field in muscle tissues. It could be the neurophysiologic basis of referral muscle pain (referred pain from muscle to muscle).[80-85]

NEW UNDERSTANDING OF MTrP

Based on the studies reviewed above, we now have better understanding of MTrPs. Some important new conclusions are discussed below.

Multiple Active and Sensitive Loci in an MTrP Region

It has been recommended that during MTrP injection the needle should be inserted into multiple sites in the entire region in order to eliminate tenderness in the entire MTrP region.[3] An LTR can be elicited each time the needle tip encounters a sensitive locus of the MTrP region.[10,11,49,50] When an LTR is elicited during MTrP injection, it is always associated with a sharp pain or discomfort and frequently associated with referred pain with patterns similar to those elicited by snapping palpation of the MTrP.[10,11] Based on observations during MTrP injections, a model of multiple small sensitive loci in an MTrP region has been proposed.[10,11] It appears that these sensitive loci are the sites from which pain, referred pain, or LTR can be elicited. These loci could be sensory receptors or sensory nerve fibers, as shown in a recent histologic study.[79]

Recent human and rabbit studies also have shown that SEA can be recorded from multiple minute sites in an MTrP (human) or an MTrS (rabbit) region.[61-63,72] These SEA loci are related to dysfunctional endplates ("motor structures"), and they are defined as active loci to distinguish them from the sensitive loci, which are "sensory structures" (table 2). Either SEA or LTRs, or both, can be observed at different loci in an MTrP region during the search for SEA, and both SEA and LTR are often associated with a sharp pain sensation that is similar to the patient's usual complaint. Therefore, a sensitive locus is probably in the immediate vicinity of an active locus, and both structures together may form an *MTrP locus,* a basic unit of an MTrP (fig 1). It is possible that the sensitive loci are widely distributed in the entire muscle but are concentrated in the MTrP region, since referred pain can also be elicited in "normal" muscle tissue (high pressure, however, is required to elicit it).[41] When a sensitive locus is associated with an active locus, an MTrP locus could develop.

Spinal Cord Mechanism of MTrP

Recent studies on referred pain and LTR[42,70,71,78,80] have supported the concept that the MTrP mechanism is closely related to spinal cord integration. When the input from nociceptors in an original receptive field persists (pain from an active MTrP), central sensitization in the spinal cord may develop and the receptive field corresponding to the original dorsal horn neuron may be expanded (referred pain).[81-84,87] Through this mechanism, new MTrPs, or "satellite MTrPs,"[3,4] may develop in the referred zone of the original MTrP.

The clinical observation of "key trigger point,"[10,11,88] "primary trigger point,"[3,4] or "gateway muscles"[14,15] has been described in the literature. The key MTrP is the original MTrP produced shortly after injury (or overloading). If the original pathologic lesion is not appropriately treated, MTrPs may propagate to other sites of the body (usually the referred zone). Injection into a key MTrP may suppress satellite MTrPs.[10,11,88] For a long-standing untreated active MTrP, the irritation from the peripheral nociceptors may be persistent, and the expansion of receptive field may increase progressively. Finally, spontaneous pain may spread to many distant regions in addition to the original reference zone through the mechanism of central sensitization in the spinal cord.

Taut Band Formation

The pathophysiology of a taut band is now much clearer. Based on studies on SEA recorded in an MTrP region, Simons[25] has proposed an updated hypothetical mechanism of taut band formation. Intracellular calcium in certain muscle fibers may be excessively released in response to trauma or abnormal stress. The abnormally increased calcium may cause uncontrolled shortening activity and increased metabolism. The muscle fiber shortening also impairs local circulation, which causes a loss of oxygen and nutrient supply to the region. This completes a vicious cycle; thus, an energy crisis occurs, and taut bands form. This hypothesis has been supported by studies that showed a low oxygen tension in an MTrP region[89] and a significant decrease in high-energy phosphates coupled with an increase in low-energy phosphates and creatine in a tender muscle site.[90] To date, no scientific data invalidate the "energy crisis" hypothesis, although this hypothesis has been challenged.[20]

The Nature of Active Loci—Endplates Versus Muscle Spindle

Recently, Hubbard[91] reported one biopsied specimen from the upper trapezius muscle of a 35-year-old patient with MTrPs. The specimen was marked with methylene blue at the site where spikes (and SEA) were recorded. In this specimen, a single muscle spindle with two nuclear bag fibers and four nuclear chain fibers were found near the marked site. Therefore, he proposed that the "TrP-EMG activity" (spikes and SEA) is

Table 2: Sensitive Loci (LTR Loci) and Active Loci (SEA Loci) in an MTrP Region

	Sensitive Locus (LTR Locus)	Active Locus (SEA Locus)
Definition	A minute site to elicit LTR when it is mechanically stimulated if the stimulating needle is moved rapidly	A minute site from which SEA can be recorded if the recording needle approaches this site slowly and gently
Probable structure	Sensory structure, sensitized nerve fiber (nociceptor)	Motor structure, endplate (abnormal endplate)
Histologic study on rabbit	A nerve fiber found at an immediate vicinity of the LTR locus	A nerve fiber found at an immediate vicinity of the SEA locus
Distribution	Mostly in the MTrP region (human and animal studies)	Mostly in the MTrP region, always in the endplate zone (human and animal studies)
Associated with pain	Always	Sometimes
Associated with referred pain	Frequently	Sometimes

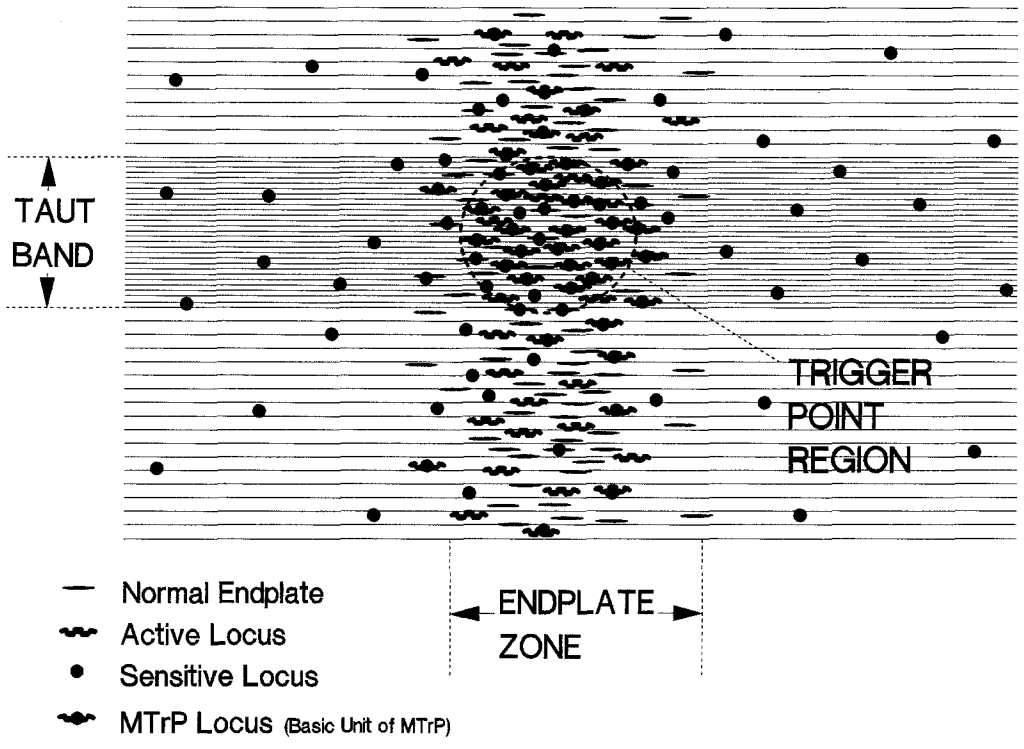

Fig 1. Sensitive loci and active loci around an MTrP region. It is hypothesized that a sensitive locus (nociceptor) may be associated with an active locus that is dysfunctional endplates. Sensitive locus (sensory component) and active locus (motor component) may cause the formation of the basic unit (MTrP locus) of an MTrP.

recorded from contracting intrafusal muscle fibers of muscle spindle and that MTrP pain arises in the spindle capsule because of increased pressure, initiated by traumatic and/or repetitive hyperextension of the spindles and sustained by sympathetically mediated factors. This uncontrolled single-case study, however, contradicts other histologic studies.[73,76,77,79]

Spikes are propagated along the length of the taut band as single muscle-fiber action potentials but not as motor-unit potentials.[64] The spike potentials are propagated at least 2.6cm along the length of the taut band, which is far beyond the maximum 1cm length of a muscle spindle.[64] These potentials are therefore propagated by an extrafusal muscle fiber instead of an intrafusal one as proposed by Hubbard.[61,91] Therefore, the MTrP is more likely located at the site of dysfunctional endplates.[25] The fact that botulinum toxin is so effective clinically for inactivating MTrPs strongly supports the endplate location.[92-94]

Pathogenesis of MTrPs

Simons[25] has suggested that the taut band is the necessary precursor to the development of MTrPs based on the fact that taut bands commonly exist in pain-free individuals[21,23] and that those who are more prone to develop taut bands are also more likely to develop MTrPs.[95] A latent MTrP may develop in a taut band in response to stressful life events and abnormal muscle stress combined with genetic predisposition.[25] Further mechanical stress or other aggravating (perpetuating) factors may cause a latent MTrP to become active. The active MTrP may recover spontaneously (if further overload is avoided), may persist without progression, or may be aggravated (increased pain intensity and spread to other sites, if perpetuating factors are present or if they are not treated appropriately).

The pathogenesis of MTrPs is probably related to an integrative mechanism in the spinal cord in response to sensitized sensory nerve fibers (nociceptors) associated with dysfunctional endplates. LTR or referred pain is probably mediated through the spinal cord in response to the stimulation of a sensitive locus that is in the immediate vicinity of an active locus. Figure 2 illustrates a proposed pathogenesis of MTrPs that is modified from two sets of hypothesis proposed by Simons[25] and Hong.[26]

Association of Sympathetic Activity With MTrP

The clinical observation of autonomic phenomena associated with active MTrP has been well documented.[3] MTrPs have been found to be a complication (or a manifestation) of reflex sympathetic dystrophy (RSD).[96] Trigger point injection could be an effective way to treat muscle pain in some RSD patients.[97]

Hubbard[91] injected phentolamine, either intramuscularly into the site of active loci or intravenously, and found that the amplitudes and the number of spikes recorded from an MTrP region were significantly reduced after injection. A similar result was obtained in another preliminary study on rabbit MTrS.[98]

Trigger Points in Fibromyalgia Patients

In clinical practice, approximately 70% of fibromyalgia syndrome (FMS) patients have MTrPs.[99,100] FMS patients have diffuse tender points caused by reduced pain threshold at many sites, including muscle, skin, subcutaneous tissues, and even nonpainful sites.[101,102] Tender points at certain sites (muscular or nonmuscular) are characteristics of FMS.[103] The muscular tender point is considered to be different from an MTrP in that no referred pain, LTR, or taut band is required to define a tender point.[103] In clinical practice, referred pain or LTR cannot be consistently elicited in some MTrPs[3,4,21,24] and is not frequently noticed in the muscular tender points of FMS patients.[21,100] This is basically because of the difficulty in the skill of palpation. High pressure may be required to elicit referred pain or LTR in some MTrPs.[41,56] FMS patients have generalized low pain threshold and cannot tolerate the high-pressure palpation that is actually required to elicit referred pain or LTR. Furthermore, they have diffuse pain in the areas of referred zone of MTrPs

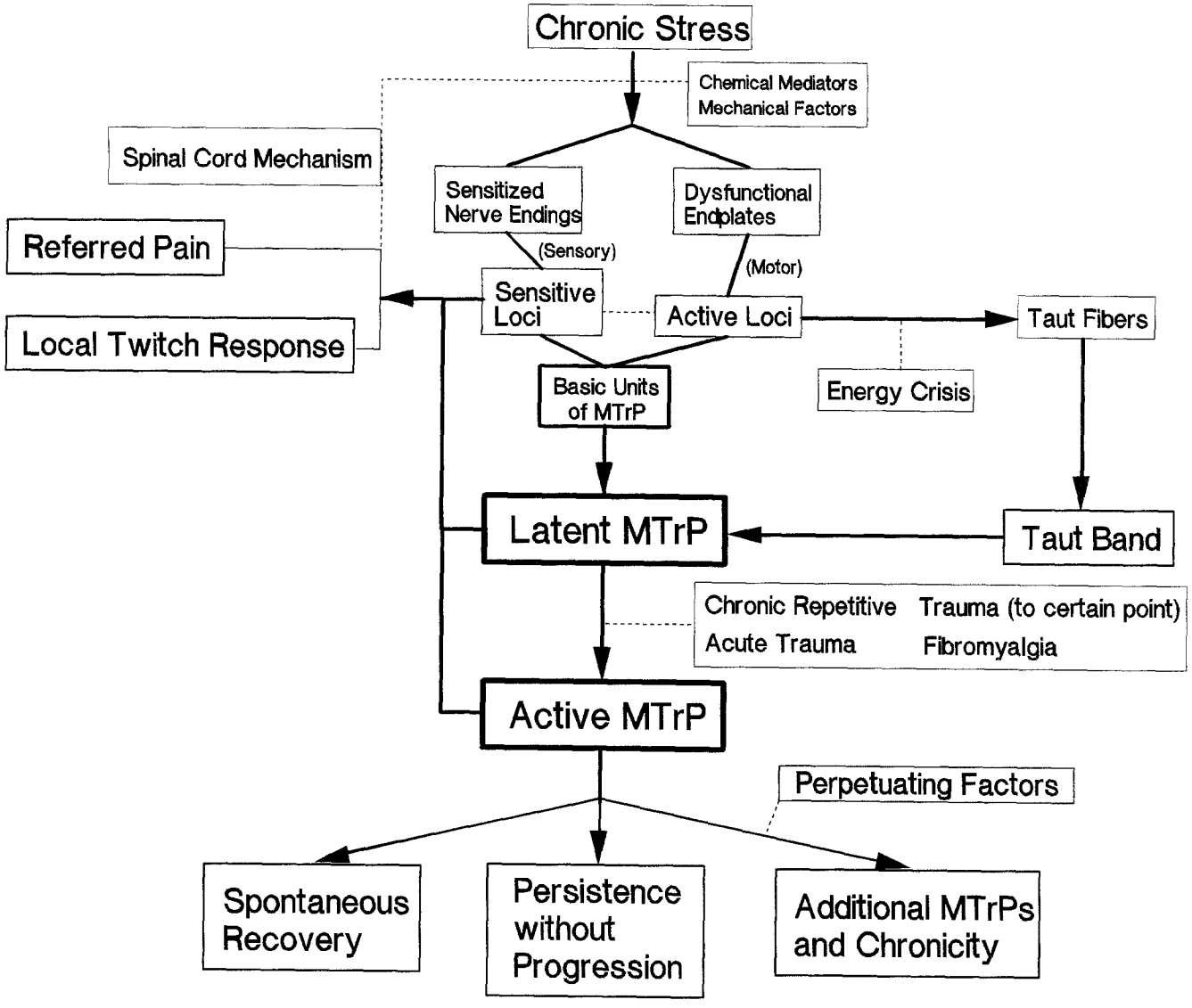

Fig 2. A proposed pathogenesis of MTrPs.

and may have difficulty in distinguishing a referred pain from a local pain. It is likely that muscular tender points in FMS patients are actually MTrPs.

The mechanism of reduced pain threshold in an FMS patient apparently results from biochemical disorders in the central nervous system.[104,105] In such a situation, latent MTrPs of FMS patients may become active because of reduced pain threshold at the central nervous system level (central sensitization). Before the onset of symptoms, an FMS patient can have many latent MTrPs in many muscle groups, like any person with no pain complaint. Latent MTrPs in an FMS patient may be more likely to be activated (because of reduced pain threshold) in response to trauma or any other factor. Some FMS patients may initially present with a regional pain syndrome that spreads to become a more generalized pain typical of fibromyalgia.

FMS patients develop diffuse pain, and when they develop MTrPs they have a poor response to MTrP therapy.[26,106] Therapy should attempt to increase their pain threshold, usually by giving medicine to modify the biochemical mechanism of the central nervous system. Patients with FMS are likely to experience significant but delayed and attenuated pain relief after MTrP injection, compared with patients who have only myofascial pain syndrome (with similar MTrPs but without FMS).[106] It appears that active MTrPs in FMS patients are more irritable and less responsive to treatment than those in non-FMS patients.

UNRESOLVED PROBLEMS AND FUTURE DIRECTIONS FOR RESEARCH

Basic Science Studies

1. The interaction between an active locus and a sensitive locus is unclear. Future studies should be directed either peripherally (on the muscle) or centrally (in the spinal cord). Peripheral studies should include the studies on the morphologic nature and the distribution of sensitive loci and active loci in the muscle, along with their correlation to the location of endplates, nerve endings, and taut bands. Study in the spinal cord should include both morphologic and electrophysiologic studies of the dorsal horn neurons in the spinal cord to identify the neural circuits related to

the MTrP mechanism. It is also necessary to clarify the mechanism by which dry needling inactivates an MTrP.

2. The nature and the formation of the taut band are becoming much clearer. Light microscopy of the responsible contraction knots[107] and ultrasound imaging of the LTR[108] have been demonstrated and need further exploration. Studies should also include biochemical study of the effects of calcium ions or calcium block agents on the consistency of muscle fibers and the electrical activity (SEA) of an MTrP locus.

Clinical Studies

1. The natural course of latent MTrPs in a normal subject is unclear. Both longitudinal and cross-sectional studies on the prevalence and activity of latent MTrPs in certain muscle groups are important to clarify the hypothesis we have proposed.
2. Studies on the association of active MTrPs with various types of injury and sites of injury may help to improve our understanding of the central, neurophysiologic mechanism related to the activation of MTrPs.
3. Experimental studies on the effects of various types of muscle activity or various postures of inactivity on the activation of latent MTrPs (to become active MTrPs) are required to confirm the "traumatic hypothesis" of MTrP activation.
4. The reflex inhibition or facilitation of muscles by MTrPs is a clinically and theoretically important subject that is now beginning to be investigated. Inactivation of MTrPs may be an effective way to reduce spasticity, if MTrPs indeed are major factors in aggravating spasticity. Scientific data are required to clarify this issue.

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