

■ Myofascial Trigger Points Show Spontaneous Needle EMG Activity

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Monopolar needle electromyogram (EMG) was recorded simultaneously from trapezius myofascial trigger points (TrPs) and adjacent nontender fibers (non-TrPs) of the same muscle in normal subjects and in two patient groups, tension headache and fibromyalgia. Sustained spontaneous EMG activity was found in the 1–2 mm nidus of all TrPs, and was absent in non-TrPs. Mean EMG amplitude in the patient groups was significantly greater than in normals. The authors hypothesize that TrPs are caused by sympathetically activated intrafusal contractions. [Key words: myofascial trigger points, fibromyalgia, EMG, muscle spindles]

Fifty years after Travell first described myofascial trigger points (TrPs),⁴⁵ and 10 years after Travell and Simons published their Trigger Point Manual,⁴⁶ the underlying pathophysiology of TrPs, a common cause of chronic muscle pain, remains unknown.

Travell and Simons define specific criteria for TrPs^{40,46}:

1. Palpable firm area of muscle, referred to as the taut band.
2. Within the taut band, a localized spot of exquisite tenderness to manual pressure, the TrP.
3. A characteristic pattern of pain, tingling, or numbness in response to sustained pressure on the TrP within the taut band.
4. A local twitch of the taut band when the TrP is distorted transversely.

Travell and Simons have reported that although the taut band may be several centimeters long, the TrP itself is only a few millimeters in diameter.⁴⁶

Travell and Simons distinguish between active TrPs that cause clinical pain syndromes, and latent TrPs, which are painless, not associated with clinical pain syndromes. Latent TrPs are, like active ones, identified by manual palpation of taut bands, tenderness, and characteristic referral pattern of pain in response to sustained manual pressure. Fifty percent of normal asymptomatic persons

have latent TrPs on examination of the shoulder-girdle musculature.⁴¹

Simons and Travell also distinguish between TrPs and Tender points (TePs). Tender points are areas of tenderness that may or may not be in muscle tissue, do not have palpable taut bands, and do not refer pain to adjacent areas.⁴⁶

Simons further distinguishes between two clinical syndromes: TrPs localized to one or two muscles, which he defines as the myofascial pain syndrome, and multiple or generalized TrPs or TePs, which he and others believe are in the spectrum with fibromyalgia.^{11,18,39,43}

Muscle biopsy studies of TrPs have searched for areas of tissue damage,⁴⁹ local hypoxia,^{7,10,12,29,39} or sympathetic hyperactivity,⁹ but have not shown consistent abnormalities by light microscopy, histochemistry, or electron microscopy.⁵⁰

Needle electromyography (EMG) of painful muscle syndromes has produced variable results. The first was published by Buchthal and Clemmesen in 1940,¹⁶ who concluded that the spontaneous EMG activity they identified arose in proprioceptive receptors. Since then a number of studies have reported on patients with lumbar disc disease,²¹ tension headache,³⁶ fibrositis,^{2,28} fibromyalgia^{20,30,51} and myofascial taut bands.^{19,23} In 1957, Travell described high-frequency firing from TrPs,⁴⁴ but in the 1983 TrP Manual Travell and Simons concluded that TrPs showed no resting activity and that any activity seen was either insertional or motor unit activity.⁴⁶

In an attempt to resolve these issues, in this study we recorded needle EMG simultaneously from the 1–2 mm nidus of TrPs and from adjacent nontender muscle fibers of the same muscle.

■ Methods

Patients presenting to the Neurologic Centre for Headache and Pain with TrPs on physical examination and who agreed to undergo needle EMG were studied. Procedures followed were in accord with the standards for human experimentation. Informed consent was obtained after the nature of the procedure had been fully explained to each subject.

Twenty-nine patients had chronic tension-type headache with pericranial muscle tenderness, diagnosed by a board-certified neurologist according to the criteria of the International Headache Society.²⁷ These patients had mild to moderate daily fluctuating bilateral frontal and occipital pain and had normal neurologic examinations except for the presence of

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TrPs. Twenty-five patients met the American College of Rheumatology criteria for fibromyalgia.⁴⁸ These patients had mild to moderate daily fluctuating bilateral neck or shoulder and low-back or buttock pain and had normal neurologic examinations except for the presence of TrPs. No patients had myopathies, neuropathies, radiculopathies, or other significant medical disorders.

We also examined eight normal subjects without history of significant head, neck, or back pain but with latent TrPs on palpatory examination. Normals with latent TrPs were chosen so that needle placement in a TrP could be compared with needle placement in non-TrP muscle fibers. For all subjects and patients, ages ranged from 18 to 80, mean 38.6, mode 36. Eighty percent were women.

Trigger points were identified by finger palpation for localized (1–3 cm diameter) muscle firmness (“taut band”), tenderness to steady pressure with the thumb or first two fingers, and referral of pain in characteristic patterns as described by Simons and Travell.⁴⁶ Trigger points in the upper trapezius were chosen for needle EMG examination because these could be identified in all patients, regardless of site of pain, and in all normal subjects (latent TrPs).

Tenderness rating is as follows: 0, no tenderness; 1, mild tenderness without grimace or flinch; 2, moderate tenderness plus grimace or flinch; 3, severe tenderness plus marked flinch or withdrawal; 4, unbearable tenderness, patient withdraws with light touch. Patients were also asked to rate their level of pain before testing, according to the following scale: 0, no pain; 1, ignorable pain; 2, moderate pain; 3, pain that interferes with one’s activity; 4, incapacitating pain requiring bed rest or cessation of activity.

Monopolar TECA disposable EMG needles were inserted through the skin directly over the TrPs. A second needle was placed 1 cm away in nontender fibers of the same muscle. Both needles were referenced to the same equidistant surface electrode. High and low cuts were 10,000 and 30 Hz, gain was 100 μ V per division, sweep speed was 100 milliseconds per division, displayed on a 2-channel Cadwell Quantum 84 machine, Kennewick, Washington. After ascertaining that the

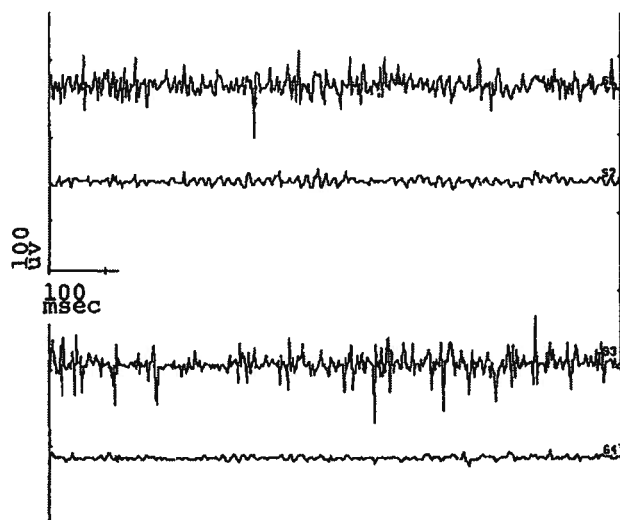


Figure 1. Spontaneous EMG activity recorded in trapezius trigger point (S1 and S3) and an adjacent nontender site 1 cm away in the same muscle (S2 and S4), in two normal subjects.

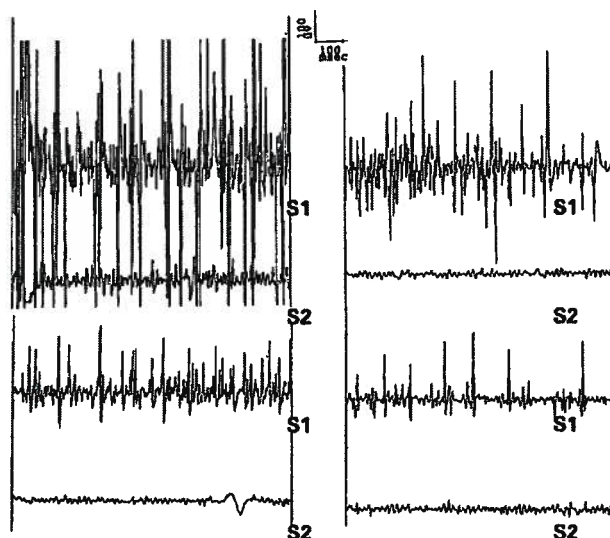


Figure 2. Spontaneous EMG activity recorded in trapezius trigger point (S1) and an adjacent nontender site 1 cm away in the same muscle (S2), in four patients.

second site was electrically silent and elicited no pain (hereafter called the non-TrP), the TrP needle was advanced in 1-mm increments until the subject reported experiencing the same pain and referral pattern experienced during manual palpation—the TrP. Typically this occurred at a depth of about 2 cm, with the patient or subject reporting a deep steady ache or squeezing sensation that radiated to the ipsilateral cervical, occipital or temporal areas as described by Travell and Simons.⁴⁶ If necessary, the needle was withdrawn and redirected until the precise point could be identified. The needles were left in place for 15–50 minutes.

All EMGs were saved on disc and printed. The Cadwell Quantum 84 Area software was used to calculate the absolute (both negative and positive deflections from the baseline) area-under-the-curve and the mean amplitude for each 1-second interval (the full screen width at a sweep of 100 msec).

■ Results

No fibrillation potentials or positive sharp waves were seen in any normal subjects or patients. Brief insertional activity could be seen when the needle was first inserted or the subject moved, in which cases both the TrP and the non-TrP needles recorded typical motor unit potentials, which either disappeared spontaneously or could be readily eliminated by relaxing the muscle.

Spontaneous EMG activity was recorded from the TrPs of all normal subjects and patients. The EMG activity disappeared when the TrP needle was moved by as little as 1 mm. No spontaneous activity was recorded from non-TrPs. In all normal subjects and patients, the appearance of the spontaneous EMG activity corresponded to the report of pain, which was described as a deep aching or squeezing sensation, typically associated with a referral of pain upward into the cervical, occipital, or temporal areas, and often associated with autonomic symptoms such as light-headedness, diaphoresis, or

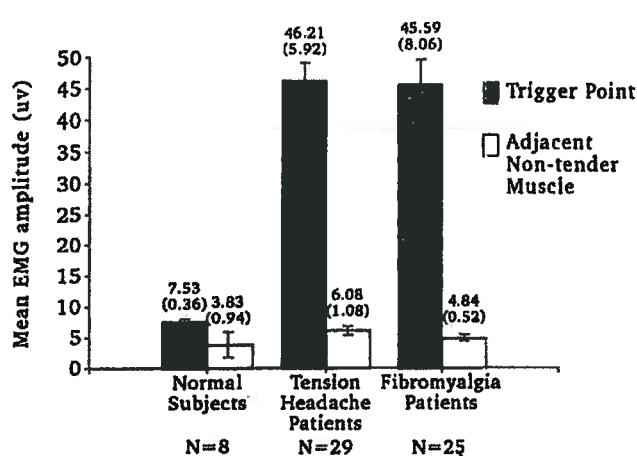


Figure 3. Mean EMG amplitudes and standard errors (SE) for TrPs and adjacent non-TrPs in trapezius.

nausea. The spontaneous activity was present for as long as the needle was not moved, up to 50 minutes.

Examples of the spontaneous EMG activity are presented in Figures 1 and 2.

Figure 3 shows the means and standard errors for mean EMG amplitudes for the TrPs and adjacent non-TrPs in normal subjects (latent TrPs) and patient groups (active TrPs). Because unequal sample sizes can exacerbate violations of statistical assumptions, both parametric and nonparametric analyses were run. With either method of analysis, the TrP mean EMG amplitudes for normals were significantly lower than for the two clinical groups, which were not significantly different from each other. In fact, the distributions were nonoverlapping. No normal subject had a mean amplitude greater than 10 uv, and no patient had less than 10 uv.

Pearson correlations between mean EMG amplitude and age, self-reported pain, and tenderness to palpation, and Point Biserial correlations between mean EMG amplitude and sex, were calculated. Correlations were as follows: age: $r(62) = 0.07$, not significant (NS); sex: $r(62) = 0.12$, NS; pain: $r(59) = 0.14$, NS; tenderness: $r(58) = 0.43$, $P = 0.0007$.

Discussion

Spontaneous EMG activity was found in the 1–2-mm nidus of myofascial trigger points (TrPs). The activity disappeared if the recording needle was advanced or withdrawn as little as 1 mm. When the needle reached the TrP, the patients consistently reported the onset or aggravation of pain and the characteristic referral pattern of pain. This activity was sustained for as long as we recorded, up to 50 minutes. The second monopolar needle 1 cm distant showed no spontaneous activity, further indicating that the TrP activity was limited to the small area and that no motor unit activity was occurring in adjacent muscle fibers. The TrP EMG mean amplitudes were significantly greater in the two groups of muscle pain patients than in normals. There was no significant

difference between the muscle tension headache patients and the fibromyalgia patients. Mean EMG amplitude was significantly correlated with tenderness to palpation of the TrP, but not with self-reported pain, age, or gender.

What is the source of this activity? Its extremely localized nature appears to rule out splinting, "spasm," or a metabolic disorder of muscle. Conversely, the activity is not localized enough to be generated in an end-plate,^{17,22} nor does it have the expected location³⁸ or waveform morphology³³ for end-plate activity. We theorize that the TrP EMG activity is generated from sympathetically stimulated intrafusal muscle fiber contractions.

The traditional teaching that there is no sympathetic innervation of muscle²⁴ is no longer correct.⁶ The sympathetic nervous system innervates not extrafusal fibers, but intrafusal fibers. This unmyelinated axon had been recognized in muscle spindles but were not known to be of sympathetic origin or were assumed to be destined for blood vessels, not muscle fibers.³⁻⁵ Using 5-hydroxydopamine (5-HDA), however, which accumulates in vesicles of the terminals of postganglionic sympathetic axons, an electron microscopy study in cats demonstrated sympathetic endings in close proximity (a few microns) to intrafusal neuromuscular junctions and muscle fiber membranes. These 5-HDA-containing fibers were not associated with blood vessels.³⁷

In 1985, Passatore and Grassi^{34,35} performed the first of several physiologic studies on rabbits, rats, and cats paralyzed with curare-type neuromuscular blocking agents and artificially ventilated. The cervical sympathetic nerve was isolated in the neck, cut, and its peripheral stump stimulated. Muscle tension was measured by an isometric force transducer attached to the animal's jaw. Blood flow was also monitored. Sympathetic nerve stimulation produced an immediate increase in jaw tension and cutting it reduced the tonic jaw tension. The response to sympathetic stimulation was not blocked by curare, indicating that it was not due to alpha-motoneuron activation of extrafusal muscle fibers, nor to gamma-motoneuron activation of intrafusal muscle fibers. The response was totally blocked by the sympathetic alpha-blocker phentolamine. Jaw muscle temperature was not affected, and occluding the external carotid artery had little effect, indicating that the response was not due to vascular changes. Jaw tension was proportional to the size of the compound action potential of the sympathetic nerve as well as to the discharge frequency recorded in the spindle afferent nerve.^{34,35}

In another study, these same investigators found that cervical sympathetic nerve stimulation produced similar increases in jaw tension with intravenous injection of noradrenaline (a mixed alpha-1 and -2 agonist) and phenylephrine (a selective alpha-1 agonist). They also found that phenoxybenzamine (an alpha-1 blocker) inhibited the tension effect of sympathetic stimulation

by approximately 50% and that the combination of this blocker and a selective alpha-2 blocker eliminated it.²⁵

In a review article, they concluded "that the physiological as well as pathological states during which the sympathetic output is modified are known to be accompanied by changes in muscle tone. The peripheral sympathetic actions upon muscle spindles could then be in part responsible for these changes. In this context those clinical syndromes characterized by pain symptomatology often associated to muscle hypertonia, should also be mentioned."²⁶

The authors implied that the jaw tension response to sympathetic stimulation was produced by intrafusal muscle fiber contractions.

In recent years, the afferent fibers responsible for nociception in muscle have been studied and comprehensively reviewed by Mense at the University of Heidelberg.^{31,32} Unmyelinated C and thinly myelinated A delta fibers with cell bodies in the dorsal root ganglia and with synapses in the dorsal horn respond to pressure, chemical, and nociceptive stimuli in muscle. Although it is widely assumed that the sensory endings are diffusely and randomly distributed throughout muscle tissue, this is in fact not known. One likely site for these endings is the spindle capsule, which contains unmyelinated fibers thought to carry pain sensation,⁴ is pressure sensitive,¹⁵ and contains a substance-P-related enzyme.⁴²

Pharmacologic studies also have demonstrated a direct adrenergic effect on muscle. Bowman^{13,14} has reviewed this evidence extensively in several comprehensive papers. In summary, adrenalin causes a 10–20% increase in tension in nonfatigued, fast-contracting mammalian skeletal muscles and increases muscle spindle excitability and spindle afferent discharge. Adrenalin also enhances physiologic tremor as well as Parkinsonian tremor. These effects are not dependent on the classic alpha or gamma motoneuron pathways because they occur in fully curarized or chronically denervated muscle. The effect is also independent of muscle blood flow. Because adrenalin does not cross the blood–brain barrier, these effects are not due to central nervous system activity. The adrenalin doses necessary to produce increases in muscle tension and spindle activity are high (3 µg/kg intravenously) and therefore have been thought to be of questionable physiologic significance. Bowman concluded that the adrenalin effects on the muscles represented "spill-over" from the vascular system.

Sympathetic activation of intrafusal muscle fibers has not been studied in humans. There has been one study, however, that evaluated the effect of sympathetic blockade on TrPs. Eight patients with primary fibromyalgia were studied before and after sympathetic blockade via stellate ganglion injection. The study reported a significant pain reduction in six of the eight patients, and a significant reduction in the number of palpated TrPs.⁸

■ Conclusion

It is now recognized that there is direct sympathetic innervation to the intrafusal fibers of muscle spindles, and that sympathetic stimulation causes muscle tension in curarized animals that is blocked by alpha-adrenergic antagonists. Furthermore, the density of muscle spindles is greatest in the neck muscles.^{1,47}

These anatomic, physiologic, and pharmacologic findings have generated little clinical interest to date. In the latest edition of *Myology*, Barker and Banks simply state that their "present position is thus that the significance of intrafusal autonomic innervation remains to be elucidated."⁶

We found sustained spontaneous electrical activity localized to the 1–2 mm nidus of TrPs. We hypothesize that sympathetically stimulated intrafusal contraction causes an involuntary low-grade but symptomatic muscle tension. Prolonged or chronic spindle tension becomes painful by distending, distorting, or chemically sensitizing the spindle capsule. Sympathetic activity explains the autonomic symptoms associated with TrPs and provides a mechanism by which local injury and nociception causes local tension, and by which emotional factors cause widespread tension and pain. For chronic muscular pain, this theory offers a mechanism of pathogenesis, an objective method of diagnosis and evaluation, and the potential for improved treatment.

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