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TRIGGER POINTS AND TENDER POINTS

One and the Same? Does Injection Treatment Help?

Joanne Borg-Stein, MD, and Joel Stein, MD

Patients with fibromyalgia and those with myofascial pain syndrome share several features, of which chronic musculoskeletal pain is the most prominent. Treatments designed to decrease pain and improve function exist for both of these disorders, but the specific treatment strategies differ significantly. The hallmark physical findings of these two disorders, trigger points in myofascial pain syndrome and tender points in fibromyalgia, need to be understood from a phenomenologic perspective, and it is hoped ultimately from a pathophysiologic perspective as well. The myofascial trigger points seen in myofascial pain syndrome commonly are treated with local treatments, such as trigger point injections; spray and stretch; myofascial release techniques; and stretching, strengthening, and postural exercises specific to the region involved. By comparison, the more generalized tender points characterizing fibromyalgia usually are treated with systemic remedies, including oral medications, generalized stretching, aerobic conditioning, and behavioral medicine or cognitive retraining.

There appears to be some overlap between the disorders of myofas-
cial pain syndrome and fibromyalgia and between trigger and tender points. This article reviews the definitions and current state of understanding about both trigger and tender points and whether or not they can be reliably differentiated by physical examination or tissue algometry. Finally, the efficacy of trigger point injection therapy is critically analyzed.

WHAT IS A TENDER POINT?

A tender point is a localized area of tenderness in a muscle, muscle tendon junction, fat pad, or bursal region. The relationship of these points to pain and fibromyalgia has been the subject of extensive research. The diagnostic criteria for fibromyalgia have evolved, with the most recent criteria published by the American College of Rheumatology (ACR) in 1990. According to these criteria, tender points become painful (not merely tender) when approximately 4 kg of pressure is applied. This pressure can be approximated by applying pressure with the thumb or first two or three fingers to the point when the fingernail bed begins to blanch. A positive tender point count of 11 or more of 18 standardized sites, when present in combination with the history of widespread pain, yields a sensitivity of 88.4% and a specificity of 81.1% in the diagnosis of fibromyalgia. Tender points were the single most powerful way to discriminate patients with fibromyalgia from controls with other painful conditions.

It subsequently has been proposed that these strict numerical criteria need not be met for clinical purposes in selected individuals with fewer than 11 tender points, but with a clinical syndrome otherwise typical of fibromyalgia. Based on the number of tender points and other symptoms, these patients might be characterized as having probable or possible fibromyalgia.

The interrater reliability of the tender point examination has been examined. Cott et al evaluated interrater reliability of the tender point examination by digital (thumb) examination as well as dolorimetry examination. They found moderate interrater reliability (kappa = 0.51) for digital palpation of individual tender points, with similar interrater reliability (kappa = 0.62) for dolorimetry. Dolorimetry resulted in classifying significantly fewer points as tender when compared with digital palpation. The classification of subjects as having fibromyalgia versus other disorders using the overall tender point count (defined by palpation) revealed a somewhat higher interrater reliability (kappa = 0.74) than the classification of any specific point as tender or not. The necessity of algome-

References 8, 10, 13, 14, 24, 39, 41, 48, 53, 56, 58, 59

try continues to be debated.

The pathology and pathogenesis of fibromyalgia remain elusive. Early studies reported with "moth-eaten" and "ragged" muscle fibers as well as an altered cellular metabolism and energy substrate. Reduction in the concentration of high energy phosphate stores and the presence of an altered cellular metabolism provide further support for this hypothesis. The cellular energy metabolism may play an important role in the pathogenesis of fibromyalgia. Similar levels of maximum muscle contraction, and high energy phosphate stores, were found in control subjects. After contraction, the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentration of high energy phosphate stores, and the muscle fibers of the fibromyalgia group demonstrated an increase in the concentra-

WHAT IS A TRIGGER POINT?

Myofascial trigger points are hyperirritable foci located in the tissu
try continues to be debated. Fischer et al.1 reported that 90% of a group of physicians treating fibromyalgia patients could not consistently determine when they were applying 4 kg of pressure by palpation.

The pathology and pathophysiology of the tender point remain elusive. Early studies reported muscle biopsy evidence of tissue anoxia with "moth-eaten" and "ragged-red" muscle fibers in the tender points.5,33 Reduction in the concentration of high energy phosphates suggested an altered cellular metabolism and a deficiency in the ability to generate energy substrate.3 In contrast, de Bleeckert et al.15 failed to demonstrate any change in metabolism of energy rich phosphates in the resting muscles of fibromyalgia patients using 31P MR spectroscopy in vivo. Similarly, Simms et al.16 demonstrated that fibromyalgia patients had similar levels of maximum oxygen consumption, maximum voluntary contraction, and high energy phosphates when compared with sedentary control subjects. After controlling for the level of conditioning, these findings were present in both resting and in exercising muscle. These recent studies suggest that there is, in fact, no consistent defect in local cellular muscle energy metabolism in fibromyalgic muscle. Conventional MR imaging of muscle and tender points in fibromyalgia has also failed to demonstrate any abnormalities.8 It has been hypothesized that the myalgias experienced during rest and exercise in fibromyalgia may result from central neurohormonal changes rather than local metabolic or pathophysiologic features. Needle electromyographic (EMG) evaluation of tender points in fibromyalgia revealed no evidence of ongoing denervation or focal muscle spasm, corroborating the biopsy, imaging, and spectroscopy data.18

It has been suggested that chronic muscular pain occurs owing to a combination of central and peripheral factors.21 Neurohormonal changes in the central nociceptive system may help to explain both pain at rest and the occurrence of achy, burning, postexercise pain. Mechanical factors may play an important role in maintaining increased peripheral sensitization.25 Although research has not identified any reproducible metabolic changes in muscle fibers thus far, it is possible that more sensitive techniques ultimately will demonstrate physiologic changes in the future. The distinction between normal and abnormal may be quantitative rather than qualitative.31,26 If physiologic changes do occur, they are likely to contribute to afferent nociception, which in turn has a role in the development and maintenance of the central transmission and perception of pain.31

**WHAT IS A TRIGGER POINT?**

Myofascial trigger points have been defined by Simons5 as "self-sustaining hyperirritable foci located in skeletal muscle or its associated
fascia. Another associated feature includes taut bands of muscle that produce local pain, referred pain, and a local twitch response when pressure is applied (Fig. 1). Other types of connective tissue, such as ligaments, peristome, skin, and scar tissue may also harbor trigger points but these are not commonly referred to as myofascial trigger points. These nonmuscle trigger points are outside the scope of this review. When local pressure applied to a tender area produces local pain without referred pain or pain reproduction, this area is properly considered a tender rather than a trigger point. A myofascial trigger point is, by definition, accompanied by a palpable qualitative difference in the muscle referred to as a taut band. Trigger points, like tender points, tend to occur in certain characteristic locations within each muscle. These have been demonstrated in most of the muscles of the human body, though certain muscles are much more commonly affected than others. The local twitch response is a transient involuntary contraction of muscle fibers elicited by mechanical stimulation of the taut band within the trigger point.

![Trigger Point Region](image)

**Figure 1.** Taut band in myofascial trigger points. (From Hong C-Z. Consideration and recommendations regarding myofascial trigger point injections. Journal of Musculoskeletal Pain 2:35, 1994; with permission.)

Some investigators describe trigger points as being painful upon palpation, but is not painful at rest. Functional shortening of the sarcomeres associated with a decreased ability to relax may result in an altered pattern of muscle activities, which the patient may not be aware of, but without significant atrophy of the muscle or weakness results from referred pain associated with muscle contraction.

Several studies have failed to reproduce myofascial trigger points. Durette et al. and others and found no evidence of Myotonic and wave potentials. Motor units were sampled. In contrast, a prospective study by and Berkoz examined EMG activity in individuals who failed to reproduce the taut band and characteristic needle was inserted directly into the zone and was reproduced. A similar method was used as a control area. Pain was reproducible in the trigger points if the needle moved as little activity was found in the absence of EMG activity, but of substantial potentials or positive sharp waves. The authors hypothesize that sympathetically stimulated nociceptive activity likely that the generation of pain by chemical sensitization of the muscle.

A similar technique of the authors who found active loci was performed on points. The morphology of points similar to that normally seen at a zone, trigger point, and acupoint is close proximity. The pathophysiologic nature determined. The authors speculate...
Some investigators distinguish between active and latent trigger points. A latent trigger point has the characteristics described previously but is not painful at rest to the patient. Pain is elicited only on palpation. Functional shortening of the muscle with trigger points occurs with a decreased ability to undergo passive and active stretch. This can result in an altered pattern of movement for functional or athletic activities, which the patient may perceive as incoordination. Muscle weakness without significant atrophy often occurs. It has been proposed that this weakness results from reflex inhibition developing from pain occurring with muscle contraction.

Several studies have been performed using needle EMG evaluation of trigger points. Duret et al. studied 21 subjects with myofascial pain and found no evidence of spontaneous fibrillation or positive sharp wave potentials. Motor unit recruitment was similar in all muscle areas sampled. In contrast, a precise needle EMG study in 1993 by Hubbard and Berkoff examined EMG activity in the upper portion of the trapezius muscle in individuals with fibromyalgia or tension headaches. In this study, trigger points were initially identified by manual palpation of the taut band and characteristic referral of pain. A monopolar EMG needle was inserted directly over the trigger point and was then advanced by 1-mm increments until the subject’s pain and classic referral zone was reproduced. A second needle was placed 1 cm away from the first as a control area. Pain complaints and characteristic referral patterns were reproduced in the trigger point region only and would disappear if the needle moved as little as 1 mm. Sustained spontaneous EMG activity was found in the 1- to 2-mm nidus of trigger points, without any comparable activity present in the control areas. The EMG activity persisted as long as the needle remained in the trigger point nidus, in one case for as long as 50 minutes. Latent trigger points were studied in asymptomatic individuals and were found to have similar spontaneous EMG activity, but of substantially lower mean amplitude. No fibrillation potentials or positive sharp waves were noted in patients or controls. The authors hypothesize that the EMG activity seen is generated from sympathetically stimulated intramuscular muscle fibers. They further speculate that the generation of pain occurs through distortion, distortion, or chemical sensitization of the spindle capsule.

A similar technique of EMG examination was used by Simons et al. who found active loci of spontaneous electrical activity in trigger points. The morphology of the spontaneous electrical activity seen was similar to that normally seen in the end plate region. The end plate zone, trigger point, and active loci of EMG activity were usually in close proximity. The pathophysiologic significance of this remains to be determined. The authors speculate that the abnormality in trigger points
may be localized to the end plate zone of muscle and related to calcium metabolism.\(^{35}\)

Laser-doppler flowmetry has been used to document decreased blood flow in the upper trapezius in patients with chronic neck pain occurring after whiplash injury.\(^{39}\) Such disturbances of the microcirculation may be too small to be detected on MR spectroscopy.

The neurobiology of the trigger point remains elusive. The mechanisms underlying allodynia and hyperalgesia as well as those producing the characteristic referral pattern pain seen with trigger points are as yet speculative. Both the pathology and the neurophysiology of the trigger point require further investigation. Most current models include an interaction between peripheral afferent nociceptors and the spinal and supraspinal central modulators of pain in the pathway from the dorsal horn neuron to the cerebral cortex. A more detailed discussion of the neurophysiology of pain as it relates to trigger point phenomena can be found in a recent review by Gerwin.\(^{25}\)

The documentation and interrater reliability of trigger point examination continues to be researched. Because trigger points can only be diagnosed by history and physical examination, a consistent and reproducible physical examination technique is critical for both clinical management and for meaningful research. The poor interrater reliability in some studies has led some to question the validity of trigger points as a meaningful descriptor.\(^{40}\) Training effects appear to be important for reliable identification of myofascial trigger points by palpation, with interrater agreement improving with uniform training. Gerwin et al.\(^{27}\) recently examined agreement among four physicians in the evaluation of muscle tenderness, taut bands, local twitch responses, referred pain, and trigger points. Interestingly, agreement was poor when first tested. Interrater reliability was re-examined after a 3-hour training session 21 months later, with good agreement for the parameters of tenderness, taut bands, pain reproduction, and trigger points. Agreement varied for different muscles. This suggests both that training influences interrater reliability and that agreement on definitions is critical. In another recent study of masticatory myofascial pain, interrater reliability of trigger point examination was highest for tenderness.\(^{44}\)

Quantification of trigger points may be facilitated by algometry. There are several different devices available with the purpose of quantitating differences in thresholds to pressure. The pressure threshold is the minimal force that induces pain.\(^{19}\) Pressure threshold measurements are both reproducible and valid.\(^{19}\) As with palpation of trigger points, these devices are still subject to user variations. The techniques and standards may be specific to individual muscles and the results depend on precisely where and at what angle the device is applied.\(^{29}\) Normative data for trigger point algometry is becoming available.\(^{46}\) The use of tenderness as the primary finding of trigger points is another facet of the classification of these painful conditions.

**TRIGGER POINTS AND TENDER POINTS: ARE THEY THE SAME?**

Whereas the distinction between tender points and trigger points has been described in detail, the research may be less straightforward. Are tender points a subset of trigger points? The syndromes of fibromyalgia and the phenomenon of trigger points have many in common. Fibromyalgia points in addition to widespread stiffening were described.\(^{40}\) Assembled two groups of patients (tender or trigger points) and compared the groups with two rheumatologists, one neurologist, and one internist. There was no consensus, and the correlation between tender points and trigger points was poor. This study suggests that the two criteria are not synonymous, at least not in the presence of widespread pain. This study also suggests that the presence of widespread pain may be a more important indicator of the presence of tender points, but this is not the case. The presence of widespread pain may be a more important indicator of the presence of trigger points, but this is not the case. The presence of widespread pain may be a more important indicator of the presence of trigger points, but this is not the case.

This study found that 6 of the trigger points were identified in the three diagnostic groups for tender points identified. All three groups found that tender points were less consistent. Taut band twitches were found as cons...
tenderness as the primary criteria on algometry for both tender and trigger points is another factor in the controversy regarding the proper classification of these painful areas of muscle.

TRIGGER POINTS AND TENDER POINTS: ARE THEY THE SAME?

Whereas the distinctions between classic trigger and tender points have been described in detail, the distinction in clinical practice and in research may be less straightforward. The question "Are trigger points a subset of tender points?" continues to be debated. Overlap between the syndromes of fibromyalgia and myofascial pain as well as between the phenomenon of trigger and tender points has been proposed. As many as 72% of fibromyalgia patients may have myofascial trigger points in addition to widespread tender points. A study by Wolfe et al. assembled two groups of experts to examine the relationship between tender points and trigger points. Four experts in myofascial pain (two physiatrists, one neurologist, and one internist) as well as four experts in fibromyalgia (rheumatologists) performed a preliminary study to try to clarify the relationships between tender points and trigger points and to compare their prevalence in various disease states and in asymptomatic individuals. Three groups were examined that included patients with fibromyalgia, myofascial pain syndrome, and asymptomatic controls. Tender points and trigger points were deemed to be present when palpation with 4 kg of force yielded at least a complaint of pain (1+ on the ACR fibromyalgia criteria study). The rheumatologists examined 22 paired sites. Trigger point examinations by the myofascial pain syndrome experts scored tenderness, taut bands, referred pain on palpation, reproduction of patient's pain, and both active and latent trigger points. Interestingly, the myofascial pain syndrome and fibromyalgia experts were unable to perform equivalent examinations, as was specified in the study protocol. The authors state that the myofascial pain examinations "were very complicated and during the training sessions it became clear that the rheumatologists were unable to become proficient enough in the myofascial pain examinations. We therefore restricted the rheumatologist examinations to fibromyalgia tender points."

This study found that the rheumatologists agreed closely in the number of tender points present for all three groups. Moreover, the three diagnostic groups differed significantly in the mean number of tender points identified. Among the myofascial pain experts, results were less consistent. Taut bands, latent trigger points, and muscle twitches were found as commonly in controls as in disease groups.
Referred pain, pain reproduction, and active trigger point measures showed significant differences between disease states and controls; however, no significant difference between fibromyalgia and myofascial pain syndrome patients was seen. Rheumatologists identified taut bands, referred pain, or pain reproduction less often than their myofascial pain syndrome expert counterparts.

Myofascial pain syndrome experts were consistent in their identification of pain reproduction, referred pain and latent trigger points; however, there was not statistically significant agreement on trigger point count total scores, taut bands, muscle twitch response, or active trigger points.

Although tender points were consistently identifiable by the examiners in this and other studies, there are substantial differences of opinion regarding the findings on physical examination for trigger points, even among expert examiners. Training and experience, manifested in part by differences in medical specialty, may account for a significant portion, but not all, of these differences. Myofascial pain syndrome experts appear to identify tender points in greater number than fibromyalgia-oriented rheumatologists. In this study, only referred pain, pain reproduction, and latent trigger points (which did not differentiate controls from disease and were themselves rarely identified) were reproducible among the myofascial pain syndrome experts. The difficulty in identifying taut bands, which has long been identified as one of the classic characteristics of trigger points, is important. Loosening the definition of a trigger point by eliminating the requirement for the presence of a taut band improves agreement substantially among myofascial pain syndrome experts.

Njoo and Van der Does studied the interrater reliability of myofascial trigger points. Of the commonly used criteria for identifying a trigger point, only local tenderness showed good interrater reliability (kappa greater than 0.5). Referred pain, palpable taut bands, and muscle twitch were not reliable. Pain reproduction and the presence of a “jump sign” were two criteria that did have good interrater reliability in this study. The conclusions of this study are somewhat weakened by the limited training possessed by some of the examiners. A substantial portion of the examinations was performed by medical students rotating for 3-month periods.

The absence of well designed studies demonstrating good interrater reliability of trigger points is perhaps the most pressing research issue in the study of myofascial pain at present. All intervention studies and inquiries into basic mechanisms depend on a well validated and generally accepted definition of trigger points. The development of the ACR criteria for fibromyalgia and the tender points seen in this disorder can serve as a model for these studying trigger points and myofascial pain.

The fact that interrater reliability on such reliable clinical features as selected and trained groups to the currently accepted features of the disease.

**DOES INJECTION TREATMENT FOR TRIGGER POINTS HAVE ANY ROLE?**

Trigger point injection management of myofascial pain syndrome, especially in the setting of recurrent injections in patients with chronic pain, can be very effective. However, the long-term benefits of trigger point injection management are not yet fully understood. Further research is needed to determine the optimal treatment approach and the long-term outcomes of injection management for trigger points.

**Table 1. SUMMARY TABLE: TRIGGER POINT MANAGEMENT**

<table>
<thead>
<tr>
<th>Distribution</th>
<th>Synthes</th>
<th>Wide Spread</th>
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<tbody>
<tr>
<td>Gender Incidence</td>
<td>Present</td>
<td>Not Present</td>
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<tr>
<td>Characteristics</td>
<td>Tenderness</td>
<td>Tenderness</td>
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<tr>
<td>Physical Features</td>
<td>Pain on movement</td>
<td>Pain on movement</td>
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<tr>
<td>Algometry</td>
<td>4 kg pressure</td>
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<tr>
<td>Inter-rater reliability</td>
<td>Good</td>
<td>Good</td>
</tr>
<tr>
<td>Pathology</td>
<td>Neurologic</td>
<td>Neurologic</td>
</tr>
<tr>
<td>MR Imaging</td>
<td>No imaging</td>
<td>No imaging</td>
</tr>
<tr>
<td>Needle electromyography</td>
<td>No needle</td>
<td>No needle</td>
</tr>
<tr>
<td>Allodynia/hyperalgesia</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Skin fold tenderness</td>
<td>Treatment with local injection</td>
<td>Treatment with local injection</td>
</tr>
<tr>
<td>Uncertain</td>
<td>Uncertain</td>
<td>Uncertain</td>
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</table>
The fact that interrater reliability can improve with training suggests that such reliable clinical criteria can be developed with a carefully selected and trained group of examiners. Table 1 summarizes some of the commonly accepted features of both tender points and trigger points.

**DOES INJECTION TREATMENT HELP?**

Trigger point injections commonly are performed in the management of myofascial pain syndrome, with widespread clinical acceptance.

<table>
<thead>
<tr>
<th>Table 1. SUMMARY TABLE: TRIGGER POINTS VERSUS TENDER POINTS</th>
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<tr>
<td><strong>Tender Points</strong></td>
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<tr>
<td>Distribution</td>
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<tr>
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<tr>
<td>Gender incidence</td>
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<td>Characteristic referral pattern</td>
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<td>Physical features</td>
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<tr>
<td>Algometry</td>
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<tr>
<td>Interrater reliability</td>
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<tr>
<td>Pathology</td>
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<tr>
<td>MR imaging</td>
</tr>
<tr>
<td>Needle electromyography</td>
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<tr>
<td>Allodynia/hyperalgesia</td>
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<tr>
<td>Skin fold tenderness</td>
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<tr>
<td>Treatment with local injection</td>
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Analysis of the medical literature examining the efficacy of these injections is hindered by difficulties in definitions, as well as variations in the technique of injection.

It is commonly stated that the best therapeutic response to trigger point injections is seen when a local twitch response is elicited at the time of injection and the injection leads to immediate relief of pain.\textsuperscript{51, 53} Needle diameters used in various case series have varied from 21 gauge to 30 gauge. In clinical practice, a 25 or 27 gauge 1.5-inch needle is satisfactory for most patients. Injection of certain deep muscles and injection of obese individuals may require longer needles. A 5- to 10-mL syringe is adequate for most purposes. The usual volume injected varies from 2 to 10 mL depending on the size of the muscle. Because of the characteristic location of many trigger points, reference texts are useful in facilitating identification of these points for injection or other local treatment.\textsuperscript{54} Once a taut band has been identified by palpation, the trigger point should be located within the band. Injection of the needle in the appropriate location should usually generate a twitch response. Repeated injections are often needed, with repetition of the procedure until a twitch response can no longer be elicited. Postinjection protocol should include a home program of local cooling and stretching exercises. A few days of relative rest is helpful if there is significant postinjection soreness.

Efficacy has been demonstrated with sterile water, lidocaine (1\% and 2\%) without epinephrine, bupivacaine, dexamethasone, and prednisolone.\textsuperscript{5, 7, 11, 16, 17, 23, 34} As an alternative to injection, dry needling of trigger points has been found to be effective.\textsuperscript{30, 34} Given the variety of injected substances that have been found to be effective, it appears that the source of the injected substance is not a critical factor. Most authors do not feel that there is any additional benefit of steroid preparation unless there is an associated bursitis, tendinitis, epicondylitis, or scar or neuroma. The use of a local anesthetic has clinical benefits inasmuch as it reduces the postinjection soreness when compared with dry needle. The presence of a twitch response with needle manipulation is an important predictor of therapeutic efficacy.\textsuperscript{34}

Interestingly, it appears that patients who have both fibromyalgia and myofascial trigger points get no immediate relief from trigger point injection and exhibit more severe postinjection soreness.\textsuperscript{55} They do have both immediate and sustained improvement in range of motion and improvement in pain intensity after 2 weeks, but the response is less compared with the patients with active trigger points but not fibromyalgia.

There are several hypotheses as to the apparent efficacy of myofascial trigger point injections but no mechanism has been proved experimentally. It has been proposed that the mechanical disruption caused by the repeated needlepoint disrupts the trigger point mechanism. A self-reinforcing cycle with both peripheral and central compo-

FUTURE TRENDS AND RESEARCH

Trigger Point Definition and Criteria

Meaningful clinical research requires a reliable manner with good reliability and validity that defines trigger points. One possible definition of trigger points is the standardization of examination and the defining characteristics. There are major and minor criteria that can achieve better reproducibility and interrater reliability defining the definition:

**Major Criteria**
- Regional pain complaint
- Focal pain to palpation
- Pain referred to a region
- Reproduction of pain complaint

**Minor Criteria**
- Presence of a taut band
- Local twitch response on palpation
- Decreased range of motion
ments has been proposed, with several variants. In this model, local muscle pain and spasm leads to increased nociceptive input to the central nervous system, which is amplified centrally. This causes increased efferent output from the central nervous system, which may directly or indirectly increase muscle contraction and pain. Interruption of the peripheral nociceptive input to the central control mechanism has been proposed to break the "vicious cycle" of pain. This bilateral interaction between peripheral and central factors is central to most theories of trigger point injection efficacy.

Although there are numerous case series demonstrating therapeutic benefit after trigger point injection, there are few well-controlled studies. This conspicuous absence of control groups limits the generalizability of most studies of trigger point injection. The potent placebo effect associated with injections needs to be controlled for properly. Blinded outcome analysis with strict outcome criteria is equally important in designing the definitive study of trigger point efficacy. The results of outcome studies on trigger point injections are summarized in Table 2.

FUTURE TRENDS AND RESEARCH

Trigger Point Definition and Validity

Meaningful clinical research on trigger points requires a precise definition of this phenomenon. Such a definition must be applicable in a reliable manner with good test-retest and interrater reliability. The problem of defining trigger points may relate more to the need for standardization of examination technique and interpretation rather than the defining characteristics. The ultimate definition may not differ substantially from the current working definition if the physical examination techniques can achieve better reliability. Based on the research on trigger point interrater reliability described previously, we propose the following definition:

Major Criteria
- Regional pain complaints
- Focal pain to palpation
- Pain referred to a regional site
- Reproduction of pain complaints

Minor Criteria
- Presence of a taut band
- Local twitch response on needling of the trigger point
- Decreased range of motion of the involved muscle
Table 2. Efficacy Studies of Injection Therapy

<table>
<thead>
<tr>
<th>Author and Year</th>
<th>Purpose</th>
<th># Subjects</th>
<th>Methods</th>
<th>Trigger Point Criteria</th>
<th>Needle Size and Solution</th>
<th>Outcome Measures</th>
<th>Follow up</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hong et al. 1990</td>
<td>Compare TPI in MPS vs MPS + FMS</td>
<td>NA</td>
<td>Blinded</td>
<td>Point tenderness, tail band, lateral paraspinal</td>
<td>NA</td>
<td>PI, PT, ROM</td>
<td>Immediate and 2 weeks</td>
<td>MPS improved, ROM, PI, and PT improved immediately and after 2 weeks</td>
</tr>
<tr>
<td>Hong et al. 1996</td>
<td>1) Assess efficacy of TPI with monosynaptic vs. polysynaptic</td>
<td>58</td>
<td>Double blind</td>
<td>Controlled</td>
<td>Tail band, lateral paraspinal</td>
<td>Restricted ROM, point tenderness, tail band, lateral paraspinal</td>
<td>PI, PT, ROM</td>
<td>Immediate and 2 weeks</td>
</tr>
<tr>
<td>Dain et al. 1994</td>
<td>Efficacy of TLP injection in reducing pain</td>
<td>132</td>
<td>Case series</td>
<td>No controls</td>
<td>Sacral tenderness</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Bove et al. 1994</td>
<td>Efficacy of TLP injection in reducing pain</td>
<td>199</td>
<td>Analysis of factors</td>
<td>No controls</td>
<td>VAS # Trigger points</td>
<td>NA</td>
<td>4 weeks post injection</td>
<td>100% cure</td>
</tr>
<tr>
<td>Drosed et al. 1999</td>
<td>Efficacy of TLP injection in reducing pain</td>
<td>53</td>
<td>Double blind</td>
<td>No controls</td>
<td>Papillary TLP + lateral paraspinal</td>
<td>NA</td>
<td>NA</td>
<td>Risk factors for treatment failure</td>
</tr>
<tr>
<td>Carlsen et al. 1999</td>
<td>Study relationship between muscle pain and pain relief</td>
<td>20</td>
<td>Open</td>
<td>No controls</td>
<td>Regional pain score</td>
<td>0-10 visual analog scale</td>
<td>0-10 visual analog scale</td>
<td>Pain reduced in 94% of patients</td>
</tr>
<tr>
<td>Byrom et al. 1999</td>
<td>Study relationship between muscle pain and pain relief</td>
<td>40</td>
<td>Uncontrolled</td>
<td>No controls</td>
<td>Tender points and trigger points (lateral paraspinal)</td>
<td>PI, PT, ROM</td>
<td>Immediate and 3, 6, and 12 months</td>
<td>PI, PT, ROM</td>
</tr>
</tbody>
</table>

Sallam 1992 | Efficacy of TPI vs. TENS | 20 | Uncontrolled | No controls | Papillary TLP | 0.5% lidocaine | 0-10 visual analog scale | 20 minutes and 3 days | Pain significantly reduced |
<p>| Bove et al. 1999 | Efficacy of TLP injection in reducing pain | 53 | Double blind | No controls | Papillary TLP | 0.5% lidocaine | 0-10 visual analog scale | 20 minutes and 3 days | Pain significantly reduced |
| Karim et al. 1997 | Efficacy of TPI in TMD | 63 | Prospective | Double blind | Papillary TLP | 0.5% lidocaine | 0-10 visual analog scale | 20 minutes and 3 days | Pain significantly reduced |
| Bove et al. 1994 | Efficacy of TPI in chronic neck pain | 57 | Randomized | Single blind | Papillary TLP | 0.5% lidocaine | 0-10 visual analog scale | 20 minutes and 3 days | Pain significantly reduced |
| Gunter 1990 | Efficacy of motor point injection in chronic neck pain | 30 | Randomized | No controls | Muscle motor points | 0.5% lidocaine | 0-10 visual analog scale | 20 minutes and 3 days | Pain significantly reduced |</p>
<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Intervention</th>
<th>Outcome Measure</th>
<th>Comparison</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salerno et al. 1992</td>
<td>92</td>
<td>Efficacy of TPI vs. TENNS</td>
<td>Pain</td>
<td>Palpation</td>
<td>0-10 verbal pain scale</td>
</tr>
<tr>
<td>Ghezzi et al. 1998</td>
<td>98</td>
<td>Efficacy of TPI in LBP</td>
<td>Pain</td>
<td>Palpation</td>
<td>0-10 verbal pain scale</td>
</tr>
<tr>
<td>Pappas et al. 2007</td>
<td>07</td>
<td>Efficacy of TPI in TMS</td>
<td>Pain</td>
<td>Palpation</td>
<td>0-10 verbal pain scale</td>
</tr>
<tr>
<td>Rount and 2004</td>
<td>04</td>
<td>Efficacy of TPI in chronic back pain</td>
<td>Pain</td>
<td>Palpation</td>
<td>0-10 verbal pain scale</td>
</tr>
<tr>
<td>Guarnieri 1990</td>
<td>90</td>
<td>Efficacy of motor point dry needling in chronic LBP in whiplash workers</td>
<td>Pain</td>
<td>Palpation</td>
<td>0-10 verbal pain scale</td>
</tr>
<tr>
<td>Lowery 1973</td>
<td>73</td>
<td>Efficacy of dry needling</td>
<td>Pain</td>
<td>Palpation</td>
<td>0-10 verbal pain scale</td>
</tr>
<tr>
<td>Cooper 1961</td>
<td>61</td>
<td>Efficacy of TPIs</td>
<td>Pain</td>
<td>Palpation</td>
<td>0-10 verbal pain scale</td>
</tr>
</tbody>
</table>

P1 = pain intensity; PT = pain threshold; ROM = range of motion; NA = not available; TPI = trigger point injection; MPS = myofascial pain syndrome; NMS = nonmyofascial LTR; LTR = local twitch response; TPI = trigger point; ACR = American College of Rheumatology; VAS = visual analog scale.
A trigger point is defined by the presence of all of the major criteria. The minor criteria are supportive, particularly in cases where there is uncertainty regarding the classification of a painful area as a tender point versus a trigger point. The concept of the latent trigger point is excluded from this definition by the requirement for a complaint of pain by the patient. These proposed criteria must be prospectively tested in a blinded fashion to establish their interrater and test-retest reliability before they can be adopted as a definitive definition for trigger points.

**Trigger Point Injection**

The methods used for trigger point injection need to be described carefully in studies of their efficacy to allow for meaningful comparisons of one study with another. Given the strong placebo value of injection therapy, control groups are essential for true assessment of trigger point injection efficacy. Blinded, placebo-controlled trials of myofascial trigger point injections with long-term follow-up are needed to determine the true efficacy of this procedure. An ideal placebo control might be injections in a control area within the involved muscle located outside the identified trigger point, with a blinded assessment by a second examiner to establish efficacy.

The overlap in the definitions and symptoms of tender points and trigger points raises the possibility that injections may be beneficial in the management of tender points. This needs to be studied in a controlled fashion, with particular attention to long-term outcomes in these individuals with a chronic disorder.

The underlying mechanisms responsible for the efficacy of trigger point injections remain unclear. This issue is tied closely with the pathology and pathophysiology of both trigger and tender points. Understanding effects of injections and other treatments may lead to a broader understanding of the pathophysiology that is responsible for these disorders.

**CONCLUSIONS**

1. Tender points and trigger points are defined as distinct clinical entities but with the overlapping feature of local tenderness.
2. Trigger points and tender points may coexist in the same individual.
3. Interrater reliability of tender point examination is well established.
4. Interrater reliability studies of trigger point examination reveal that tenderness and findings, with genetic bands, the twitch response.
5. The clinical distinction has important therapeutic strategies are substantiated by often the most syndrome, whereas bursitis, myalgia.
6. Myofascial trigger point pain and improved reduced through the response with twitch effect in conjunction with must include structural and respiratory.
7. Further research is needed of trigger points' role in diagnostic techniques and the true efficacy of this treatment.

**References**

10. Campbell SM, Clark S, Tindall "Blinded" controlled study of sy...
that tenderness and pain reproduction are the most reproducible findings, with generally poor results for the presence of taut bands, the twitch response, and the jend sign.

5. The clinical distinction between trigger points and tender points has important therapeutic implications, because the management strategies are substantially different at present. Local treatments are often the most effective approach to the myofascial pain syndrome, whereas systemic approaches are better used for fibromyalgia.

6. Myofascial trigger point injections have been shown to decrease pain and improve range of motion. Postinjection soreness is reduced through the use of local anesthetics for injection. A twitch response with needle manipulation appears to be an important predictor of therapeutic efficacy.

7. Trigger point injections appear to be effective but must be used in conjunction with a comprehensive program. This program should include stretching exercises, aerobic exercise, and functional and vocational restoration.

8. Further research is essential to define better the clinical features of trigger points, the reliability of physical examination and other diagnostic techniques, the pathophysiology of these disorders, and the true efficacy and mechanisms underlying injection treatment.

References


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