# Myofascial Pain Syndromes–
# Trigger Points

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## *INTRODUCTION*

This issue marks a major milestone in the increasingly widespread recognition of myofascial trigger points [TrPs] as a credible medical entity. Two [non-Simons] articles add substantially to our understanding of the etiology of TrPs by identifying recent research advances that increase the completeness of the Integrated Hypothesis. Gerwin and colleagues considered the histochemical milieu of a TrP, while McPartland identified genetic factors (1,2). The initial paper by Hubbard (3) triggered much of the current thinking and provided an invaluable stimulus and a new approach including a Muscle Spindle Hypothesis. However, these recent articles on the Integrated Hypothesis and the comments on the paper by Chung and colleagues (4) weigh heavily in support of the Integrated Trigger Point Hypothesis.

Several articles highlight the importance of TrPs in clinical practice with contributions from the fields of gynecology, urology, otolaryngology, acupuncture, physical medicine, physical therapy, dentistry, etc. Reviewed articles originated in the United States, Canada, Turkey, Switzerland, Spain, and Australia, which suggests that the recognition of TrPs is gaining ground not only in various medical fields, but also around the world. Each article review indicates whether it is prepared by Simons [DGS] or Dommerholt [JD].

## *CLINICAL STUDIES*

**Effect of Increased Sympathetic Activity on Electrical Activity from Myofascial Painful Areas. J.W. Chung, R. Ohrbach, W.D. McCall Jr. Am J Phys Med Rehabil 83:842-850, 2004.**

### *Summary*

The authors state initially that the mechanism leading to the electrical activity studied in this article is unclear and proceed to study electrical activity in painful areas in upper trapezius muscles that are identified as active painful areas according to the criteria of Travell and Simons (5), which describes the nature of and identification of myofascial trigger points [TrPs]. They list four hypotheses that purport to explain this electromyographic [EMG] activity: dysfunctional neuromuscular junctions [the Integrated Hypothesis], disinhibition from contralateral muscle spindle afferents, activation of the gamma motor neurons, and activation of intrafusal muscle fibers by the sympathetic nervous system. They strongly favor the fourth hypothesis because it explains the well-established influence of sympathetic effects on this electrical activity and apparently assume that none of the other three hypotheses account for this sympathetic influence. This article reports the effect on this electrical activity of increas-

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ing sympathetic outflow to muscle by breath holding.

The areas examined in six subjects met the criteria for TrPs: palpation of a painful area in a palpable taut band that reproduced the patient's adjacent neck and head-area pain. These features were absent in a control location within several centimeters of a painful site. A monopolar EMG needle was advanced at a painful and at a control site in equal small increments to the same depth. When the painful-site needle encountered the electrical activity, recordings were made during breath holding, and while breathing normally. Surface electrodes also recorded EMG activity at each needle site.

The electrical activity illustrated from a painful area corresponds to that published in a 1989 EMG textbook (6) as endplate noise and spikes. Averaged value of EMG amplitude at painful sites for normal respiration was 49.4 μV and with breath holding against the glottis [increased intra-thoracic pressure and sympathetic activity] it was 120.8 μV, a 145 percent increase [$P < 0.05$]. The corresponding EMG amplitude from control areas with the needle located along the course of the same fibers as those monitored in the control area [likely in the taut band] increased 137 percent, whereas EMG amplitude [endplate noise and spikes] from control fibers lateral to the taut band showed only a 10 percent increase with breath-holding. Surface electrode EMG activity was 25 percent greater during elevated intra-thoracic pressure. Pain pressure thresholds were significantly higher at control than at painful sites [$P < 0.01$]. The authors concluded that increased electrical activity in the painful area was due to sympathetic activity and that the EMG response to the elevated intra-thoracic pressure was local. The authors rejected serious consideration of the Integrated Hypothesis, because it does not postulate clearly how endplates are affected by the sympathetic nervous system.

### *Comments*

To this reviewer, the authors' total rejection of the term myofascial trigger point, rejection of endplate noise as the potentials being studied, and rejection of the Integrated Hypothesis, because the specific mediating substance from the neighboring sympathetic nerve fibers that induces increased acetylcholine [ACh] is inexplicable if one is acquainted with and understands the TrP and EMG literature. The criteria used to locate the painful spots were explicitly the criteria for identifying TrPs. Kimura's identification of endplate noise and spikes was based on that author's recordings and on the 1970 paper by Wiederholt (7), which established the motor endplate origin of these endplate noise potentials. This understanding of those potentials with regard to TrPs has been strongly reinforced by subsequent EMG literature (8-11). The only amendment of the Integrated Hypothesis needed is to identify the transmitting histochemical responsible. The original description of the hypothesis emphasized the importance of autonomic modulation of ACh release from the motor nerve terminal TrPs (5). One paper has identified alpha and beta adrenergic receptors in the nerve terminal that, when stimulated, increased ACh release into the synaptic cleft (12). There are now a number of recent articles that strongly endorse and help substantiate the basic concept of the Integrated Hypothesis (1,2,13). In the endplate zones of many of the human upper trapezius TrPs that we examined (11), we commonly noted that when the patient took a deep inhalation, endplate noise and endplate spikes would appear and on gentle exhalation the spikes stopped and the endplate noise subsided substantially, but not entirely. We also noticed that the control EMG needle that was located nearby but was not recording endplate noise or spikes showed a corresponding increase in background noise of considerably lower amplitude than the endplate noise. This respiratory coupling was not observed in the endplate noise of TrPs in lower extremity muscles. Apparently the nature of the non-endplate background noise activity in the respiration-coupled trapezius muscle has never been explored. A similarly unexplored noise can sometimes be observed in subcutaneous tissue [not fat]; however, we did not check it for autonomic-activity responsiveness.

On the other hand, there are glaring flaws in the muscle spindle hypothesis. One is the lack of any critical substantiation of the possibility of recording intrafusal fiber action potentials with the needle the authors used, and every likelihood that it is physically not possible with that needle because of the strong capsule around the

spindle. Also the distribution of the potentials reported bear no relationship to the distribution of muscle spindles in the many muscles that have been examined for endplate noise. Spindles are distributed rather uniformly throughout the muscle and not concentrated in the mid-fiber region of the endplate zone. Some muscles with no muscle spindles exhibit this endplate noise. The spindle hypothesis makes no pretense of explaining the taut band, which is a central theme of the Integrated Hypothesis.

The value of this well-designed study is marred by what was apparently a false assumption by the authors that this study was not applicable to TrPs, or to the Integrated Hypothesis. Otherwise, it is a welcome addition to our understanding of TrPs [DGS].

**Myofascial dysfunction in the pelvis. J. Jarrell. Curr Pain Headache Rep 8(6):452-6, 2004.**

***Summary***

Puzzled by the finding that between 25 percent and 40 percent of all cases of laparoscopy done for pelvic pain do not demonstrate an identifiable visceral cause for the pain, Jarrell became interested in the contributions of myofascial trigger points [TrPs] to chronic pelvic pain syndromes. According to Jarrell, pelvic pain cannot only be due to TrPs, TrPs may also be a sign of underlying organic disease, which was the focus of this study. Fifty-five consecutive patients with pelvic pain were evaluated in a cross-sectional design. Subjects had to present with chronic pelvic pain and be found to have, as a component of their condition, evidence of myofascial dysfunction in one or more areas of the abdomen and pelvis. The specific objective was to describe the subjects with myofascial dysfunction and pelvic pain more carefully in terms of the number of TrPs and their relationship to age, parity, treatment, and underlying visceral disease. Subjects were considered to have evidence of visceral disease if they had been treated for a surgically confirmed visceral cause of pain in the past or had documented evidence of current visceral disease.

The only variable that Jarrel found to have a correlation with visceral disease was the presence of an abdominal wall TrP, which predicted evidence of visceral disease in 90 percent of subjects. If a TrP was not present, it was associated with no visceral disease in 64 percent of the subjects. Presence of TrPs in the perineum or the intrapelvic muscles was not associated with previous or existing visceral disease. The author emphasizes that, because of a strict patient selection bias, these correlations would not necessarily be observed in a more general group of subjects with chronic pelvic pain.

***Comments***

As Jarrell mentions, the presence of abdominal TrPs in patients with chronic pelvic pain may be indicative of an underlying visceral disease process, or may have resulted from previous visceral disease. By limiting the medical approach to treating the TrPs only, the underlying diagnosis could potentially be missed. This study does not suggest that abdominal muscles are the only muscles to consider when examining patients with chronic pelvic pain. We recommend examining the gluteals, quadratus lumborum, levator ani, hip adductors, obturators, and piriformis muscles as well (14) [JD].

**Urologic myofascial pain syndromes. R. Doggweiler-Wiygul. Curr Pain Headache Rep 8(6):445-51, 2004.**

***Summary***

The author is a practicing urologist associated with the University of Tennessee. In this paper, she describes that painful bladder syndrome/interstitial cystitis, chronic prostatitis, and irritable bowel syndrome are often associated with abdominal wall and pelvic floor muscle trigger points [TrPs]. Both visceral pain from pelvic organs and myofascial pain from TrPs generally are diffuse and poorly localized. Peripheral and central sensitization with resultant hypersensitivity and allodynia are common in both conditions. Referred pain can be from visceral organs to the muscles or from TrPs to visceral organs and both syndromes can trigger each other. Dr. Doggweiler summarizes several common referred pain patterns from TrPs in the low back, abdominal, and pelvic region. She emphasizes that visceral disease may

increase TrP activity as seen for example with herpes viruses and urinary tract infections.

She continues with an overview of the components of a comprehensive urologic examination, including an assessment of bladder function, voiding diary, vaginal or rectal pelvic examination assessing tenderness, contraction, strength, and coordination of the pelvic floor muscles, assessment of perpetuating factors, a musculoskeletal evaluation, that includes gait and posture, and manual examination of TrPs. The author concludes that his article should not be interpreted as saying that painful bladder syndrome/interstitial cystitis, chronic prostatitis, and irritable bowel syndrome are always caused by TrPs but the possibility needs to be considered before planning more invasive approaches. Myofascial trigger points can be the only or concomitant cause of many debilitating pain syndromes.

### *Comments*

This article complements the paper by Jarrell reviewed above. It is another excellent example that the training in the identification of TrPs needs to extend to many different medical disciplines. It is remarkable that after Drs. Doggweiler, Jarrell, and Teachey [also reviewed in this issue] have been trained to properly identify TrPs, they each have applied the newly gained knowledge into their respective practices. Each physician found that in many cases common diagnoses within their disciplines could be attributed to TrPs. It makes us wonder how many patients with interstitial cystitis, chronic prostatitis, or other visceral disease could be managed so much better if their clinician had been trained in the identification of TrPs [JD].

## Topical lidocaine patch therapy for myofascial pain. A.S. Dalpiaz, S.P. Lordon, A.G. Lipman. J Pain Palliat Care Pharmacother 18(3):15-34, 2004.

### *Summary*

This pilot study was an open label, non-randomized, single-center study, which attempted to evaluate the efficacy of a lidocaine patch on pain and quality of life in subjects with myofascial trigger points [TrPs]. All subjects were selected from the patient population of a pain management center by the study physician. They were at least 18 years old, had pain from TrPs for at least three months, with an average score on the Brief Pain Inventory pain intensity scale of at least 4. The study physician evaluated each subject for the presence of TrPs. Subjects were excluded if they had known lidocaine sensitivity or allergy, used any lidocaine containing products, para-aminobenzoic derivative, local anesthetic, or cardiac anti-arrhythmic drug at the time of the study, had known sensitivity or allergy to an amide-type local anesthetic agent, or had a history of hepatic disease.

Each subject was instructed to place a lidocaine patch over the most sensitive TrP. Subjects were evaluated at baseline, and after 7, 14, and 28 days with the Back Pain Inventory. Quality of life was assessed with the Global Assessment of Efficacy. Statistically significant improvements were recorded for average pain, general activity, walking, ability to work, mood, relationships, sleep, and enjoyment of life with different degrees of progress for each entity. Ten percent of subjects reported no benefit, 12 percent reported "some relief" after one to two days, 52 percent "wished to continue to use the patches." The researchers concluded that topical lidocaine provided some relief and improvements in quality of life measures to almost 50 percent of patients with TrPs and that this pilot study supported the initiation of a randomized, placebo-controlled study of lidocaine patches for myofascial pain.

### *Comments*

Although the authors referenced the 1983 first edition of volume 1 of the *Trigger Point Manual* instead of the second edition (5), there is no description as to which criteria were used to determine whether subjects had TrPs, how the most tender TrPs were identified, and how familiar and experienced the study physician was in assessing TrPs. The authors included in their paper a section on limitations of the current study design with the most obvious limitations a lack of a control group, subject selection bias, lack of randomization, and the lack of blinded evaluators. Nevertheless, it is conceivable that lidocaine patches could be used in the

treatment of patients with TrPs. Future double blinded, randomized and placebo-controlled studies could determine not only the direct effect of lidocaine patches on TrP pain, but could also assess whether lidocaine patches would be useful to reduce post-injection/dry needling soreness [JD].

**Efficacy of 904 nm gallium arsenide low level laser therapy in the management of chronic myofascial pain in the neck: A double-blind and randomize-controlled trial. A. Gur, A. J. Sarac, R. Cevik, O. Altindag, and S. Sarac. Lasers Surg Med 35(3): 229-35, 2004.**

***Summary***

Low level laser therapy is gaining much ground as a valid treatment tool for myofascial trigger points [TrPs]. Several previously reviewed studies were inconclusive because of methodologically errors and misconceptions (15, 16). The current paper does not suffer from such errors. The study is a prospective, double blind, randomized, and controlled study of the effects of infrared low level 904 nm Gallium-Arsenide laser therapy [LLLT] on myofascial trigger points [TrPs]. Sixty subjects with a history of chronic myofascial neck pain were randomly assigned to two treatment groups. Group one received actual laser therapy, while group two received sham laser. All subjects were seen daily for two weeks except weekends. Outcome measures included pain at rest, pain at movement, number of trigger points, the Neck Pain and Disability Visual Analog Scale, Beck depression Inventory, and the Nottingham Health Profile. Measurements were taken at baseline, and at 2, 3, and 12 weeks.

Inclusion criteria were 1. age 17-55 years; 2. pain from the neck and shoulder-girdle lasting at least one year, affecting the quality of work or daily living; 3. between one and 10 tender points in the shoulder-girdle, tender points that on palpation induced reproduction of the reported symptoms [note: even though the authors used the term "tender point," from the study it is clear that they used Simons, Travell and Simons criteria for TrPs (5)-JD]. The researchers excluded nearly all other possible pathologies and conditions.

The study showed significant improvements in the laser group in all studied parameters, including levels of pain, number of TrPs, depression scores, and functional and quality of life measures. In addition, subjects in the treatment group scored significantly higher on a test for self assessed improvement of pain [63 percent vs. 19 percent]. The authors conclude that LLLT can be an important adjunct in the treatment of patients with TrPs.

***Comment***

Even though the exact mechanisms of LLLT are not known, this study eloquently illustrates that LLLT should be considered in the treatment of TrPs. It is likely that the effects of LLLT are due to a combination of anti-nociceptive, anti-inflammatory, collagen proliferation, and circulatory effects. LLLT is an excellent choice of treatment, especially for those patients with adverse effects to medications or needling procedures. The treatment is painless and has very few, if any, negative side effects [JD].

## *REVIEW ARTICLE*

**Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. D.G. Simons. J Electro Kinesiol 14:95-107, 2004.**

***Summary***

In this review article, Simons explores the impact of myofascial trigger points [TrPs] on work-related musculoskeletal pain. After a comprehensive historical overview dating back to the early 1900s, the author reviews the clinical features of TrPs, comments on various treatment options, and most importantly, provides a critical and reflective analysis of the Integrated Hypothesis. In taking a patient's history, common features suggesting the presence of clinically relevant TrPs include a complaint of regional pain, onset of pain whether following sudden muscle overload, sustained muscular contraction, or repetitive activity. Acceptance of the concept of TrPs is hampered by at least

five reasons: 1. TrPs lack a generally recognized etiology; 2. there is no diagnostic gold standard; 3. TrPs are underexamined by research investigators; 4. TrPs are complex, interactive, and often coexist with other conditions; and 5. relatively few practitioners have received adequate training to diagnose TrPs.

The second half of the paper reviews various aspects of the Integrated Trigger Point Hypothesis and points to several areas of future research to validate the underlying assumptions. For example, Simons expresses that "a study is needed that examines the prevalence of TrPs at sites identified as endplate noise during routine electromyography," or "biopsies including longitudinal sections of human TrPs are urgently needed." The paper concludes that TrPs are indeed a likely source of musculoskeletal disorders, especially in the workplace.

### *Comments*

It is always refreshing when an author is able to question his own work. In this paper, Simons reviews with much clarity and honesty what is and what is not known about the pathophysiology of TrPs. Many peer-reviewed research studies on TrPs are difficult to obtain, as the journals that have published them are not necessarily included in the database of the National Library of Medicine [Medline, PubMed and Gateway]. In spite of these often excellent publications, there is a tremendous shortage of research that incorporates the clinical experience of practitioners worldwide familiar with the identification and management of TrPs. Further studies are needed that explore the pathophysiologic mechanisms underlying TrPs. The articles by Gerwin and colleagues and McPartland (1,2) may signal a significant step in that direction and combined with this article form a solid basis for future research efforts [JD].

## An expansion of Simons' Integrated Hypothesis of trigger point formation. R.D. Gerwin, J. Dommerholt, J.P. Shah. Curr Pain Headache Rep 8: 468-475, 2004.

### *Summary*

Based on new experimental data and established muscle pathophysiology, the authors propose an expansion of Simons' Integrated Hypothesis (5) as to the etiology of myofascial trigger points [TrPs]. They consider the event that activates a TrP to be an acute or repeated muscle overload such as eccentric or strong concentric contraction with the contractile forces distributed irregularly through hypoperfused muscle. Focal areas of muscle injury and ischemia cause low tissue pH and hypoxia. These in turn induce local histochemical changes that release substances that stimulate muscle nociceptors that cause pain. The histochemical changes also facilitate resting acetylcholine [ACh] release at the myoneural junction, inhibit ACh breakdown, and inhibit removal of ACh from its receptor. As these changes wind up and become self-sustaining they induce local muscle contracture. This increased muscle-fiber tension is responsible for the palpable taut band characteristic of TrPs.

Normally, the nerve terminal releases quantal [packets of] ACh by exostosis into the synaptic cleft, at various rates continuously, and in large quantities in response to a motor nerve action potential that originates in the motor neuron. In addition, the nerve terminal can spontaneously release quanta occasionally or leak ACh continuously at various rates as non-quantal ACh. The acetylcholine esterase in the synaptic cleft limits ACh passage to the acetylcholine receptors [AChR] in the postjunctional membrane of the muscle cell and also helps to terminate ACh activation of the receptor. These functions of the esterase are inhibited by the acidic milieu observed in the region of an active endplates (13). Activation of many of these ACh receptors due to the simultaneous arrival of a large number of quanta induces an action potential that eventually causes a muscle contraction [twitch].

Following muscle overload, sufficient [abnormal] continuing steady activation of individual receptors due to spontaneous ACh release from the motor nerve terminal depolarizes the postjunctional membrane and produces endplate noise, but rarely induces a propagated action potential. Such occasional threshold responses are identified as spontaneous endplate spikes. A key feature of the hypothesis is this increased effectiveness of ACh with resulting endplate noise and occasional endplate spikes. The initial injury-induced muscle fiber ischemic

hypoxia and tissue acidity induces release of adenosine triphosphate, substance P, calcitonin gene related peptide, bradykinin, cytokines, and other substances that sensitize and activate muscle nociceptors (13,17). These substances are well known to cause local edema and pain and can produce central neuroplastic changes that lead to allodynia, hyperalgesia, and enlargement of the pool of activated dorsal-horn nociceptive neurons (17).

Calcitonin gene related peptide 1 [type 1] is produced in the anterior horn motor neuron body and goes by axoplasmic flow to the nerve terminal. Its production is up regulated by neuronal blockade. Calcitonin gene related peptide increases the effectiveness of ACh by enhancing spontaneous release of ACh from the nerve terminal; by down-regulating all forms of ACh esterase and their activity at the synapse; and up-regulating ACh receptors in the postjunctional membrane by increasing their phosphorylation; by increasing the rate of AChR desensitization; by prolonging the mean open time of AChR channels; and by increasing the concentration of ACh receptors on the postsynaptic membrane.

In addition to the nociceptor sensitizing substances identified by Shah and colleagues (13) in the region of involved endplates, the presence of an acidic pH alone strongly initiates and perpetuates muscle pain in rat muscle without damaging muscle tissue. This rat model demonstrates that secondary mechanical hyperalgesia is maintained by neuroplastic changes in the central nervous system. Mechanical hyperalgesia is characteristic of TrPs.

The authors conclude that these new findings support the main thesis of the Integrated Hypothesis and point to areas needing further investigation.

### *Comments*

This tour de-force of the histochemical and activity changes that can occur in motor endplate regions within an TrP fit beautifully the clinical characteristics of TrPs and strongly reinforces the Integrated Trigger Point Hypothesis. The addition of calcitonin gene related peptide effects to our thinking is of fundamental importance for a better understanding TrPs. The remarkable study by Shah and colleagues (13) establishes clearly that the substances described so fully in this paper are present and active in TrPs. Together they open a new chapter in the TrP saga. Another article by McPartland [McPartland 2004] that is also reviewed in this issue approaches this same subject from the genetic point of view and makes a number of additional valuable contributions to our understanding of this hypothesis. Together, these reports open the road to additional research that can further refine the hypothesis [DGS].

## Travell Trigger Points–Molecular and osteopathic perspectives. J.M. McPartland. JAOA 104:244-249, 2004.

### *Summary*

McPartland effectively summarized how the Integrated Hypothesis added substantially to our understanding of the pathophysiology of myofascial trigger points [TrPs]. The hypothesis, first published in the 1999 *Trigger Point Manual* (5), identifies the core dysfunction with the effect of increased release of acetylcholine [ACh] in involved myoneural junctions [endplates] of skeletal muscle. McPartland presents in detail how genetic effects producing presynaptic, synaptic, postsynaptic, and acquired dysfunctions could do this. The genetic effects involve defects in the L-type and N-type voltage-gated $Ca^{2+}$ channels.

PRESYNAPTIC: internet sources list 695 reports of L-type and 57 reports of N-type $Ca^{2+}$ channel mutations that would increase release of ACh from the nerve terminal. SYNAPTIC: known genetic defect can impair cholinesterase inactivation of ACh within the synaptic cleft.

POSTSYNAPTIC: the five subunits of nicotinic ACh receptors depend on at least 16 gene codes that combine in a variety of ways that can produce gain-of-function defects, making them particularly susceptible to genetic defects.

ACQUIRED: dysregulated expression of the 16 gene codes for nicotinic ACh receptors can substitute the central nervous system form [activated by nicotine] for the muscle form that is not responsive to nicotine [may be important to smokers]; single genes expressing splice variants; and by simple upregulation of L-type

and N-type $Ca^{2+}$ channel receptors by psychological, physiological, and chemical stressors.

The author then describes how these effects interact with and reinforce other features of the Integrated Hypothesis including segmental central nervous system effects [somatic dysfunctions of Korr] and with biomechanical factors like postural disorders. He details how the most recent clinical treatments of the *Trigger Point Manual* relate closely to osteopathic manual techniques and emphasizes the common interactions between TrPs and articular dysfunctions.

McPartland presents a detailed review of how Quotane, capsaicin, dry needling, Botulinum toxin injection, quinidine, diltiazem hydrochloride, and herbal medicines that are used to treat TrPs affect $Ca^{2+}$ or $Na^{+}$ channel function. He concludes that with a better understanding of its molecular basis, the TrP approach will continue to co-evolve with osteopathic concepts.

### *Comments*

The appearance of this article and that of Gerwin and colleagues (1), which is also reviewed in this issue, indicate that the Integrated Hypothesis has stimulated further research and integrative thinking in support of the Hypothesis as the most credible concept of the etiology of TrPs. This paper is a gold mine of support for that hypothesis and shows a remarkable depth of understanding of the hypothesis and current knowledge of genetic effects. Especially propitious is the enthusiasm for this concept and clinical approach shown by an osteopathic physician. The osteopathic literature has had few reviewed articles on TrPs for many years. Hopefully this is a breakthrough that will quickly gather momentum [DGS].

## Manual therapies in myofascial trigger point treatment: a systematic review. C. Fernández de las Peñas, M. S. Campo, J. F. Carnero, J. C. M. Page. J Bodywork Movement Ther 9:27-14, 2005.

### *Summary*

The seven studies that were included in this review of results of manual therapy treatment of myofascial trigger points [TrPs] were found by searching seven databases and were evaluated for quality on a 10-point scale. Two blinded reviewers examined each article for inclusion and exclusion criteria, design, randomization, description of dropouts, blinding, outcome measures, details of the intervention used, and results. Two studies rated 6 points, two-5 points, and one each 3-, 2-, and 1-point. The results of this examination were tabulated for each article. Treatment was aimed at reducing pain and restoring normal function and most treatments were targeted at deactivating TrPs. Treatments that were reported included spray and stretch, soft tissue massage, and pressure release [misnamed ischemic compression] in two studies each. Occipital release, active head retraction and retraction/extension [per McKenzie], strain/counterstrain, and myofascial release were studied in one study each. Only two studies examined efficacy beyond placebo and found no difference.

The authors reported finding: 1. that few randomized controlled trials analyzed manual therapy of TrPs; 2. that neither retraction/extension exercises nor ultrasound with massage and exercise were better than placebo; 3. an urgent need for research that establishes efficacy of treatment beyond placebo; and 4. that "no reported treatment had been more efficacious than control intervention," and that some [three] trials confirmed that "TrP treatment is effective in reducing the pressure pain threshold and visual analog scale [VAS] scores." [To this reader, the two statements in item 4 can be interpreted as self-contradictory.] They noted the established value of outcome measures including pressure pain threshold measures by algometry, VAS measures, and range of motion, since TrPs characteristically restrict it.

The authors expressed serious concern about the lack of general agreement as to appropriate diagnostic criteria for identifying TrPs by examination and quoted five studies that questioned the reliability of all of the examinations that have been commonly recommended. They concluded that efficacy of manual therapy beyond placebo has been neither established nor refuted and that it is effective in reducing pressure pain sensitivity [of TrPs].

### *Comments*

This thoughtful review of carefully selected literature provides valuable insight into where we now stand and what is most urgently needed. There are just two points that may need some clarification. The authors' tabulated results of the Gam and colleagues (18) indicated that treatments caused significantly less tenderness than no treatment in controls. More specifically, the authors of that study concluded that massage of the TrPs and a home stretching program was effective in reducing the number and intensity of treated TrPs, whereas ultrasound treatment made no difference. The fact that this effect on TrPs did not result in significant reduction in clinical pain complaint [VAS scores] may be at least partly due to the fact that over half of the treated patients had more than the five active TrPs [could be 10] that were selected for treatment in this study. The remaining untreated TrPs would be likely to be aggravated and cause more pain because of the absence of the treated TrPs. This would obscure the clinical benefits of treatment. Clinical experience generally is that when the TrPs that are causing the pain become less tender the pain complaint decreases. If research studies do not substantiate this, it is important to determine why.

The other point concerns reliability of examination of TrPs. The author's tabulated results of four studies clearly indicate that some examinations are consistently more reliable than others. Some of the studies cited had significant weaknesses that would account for much of their poor results. The tabulated results of the Gerwin and colleagues study (19) make it clear that the three examinations they recommended were highly reliable with high kappa scores of: palpable taut band-0.85, tender spot in taut band-0.84, pain recognition-0.88. They specifically did not recommend the local twitch response-0.44 as a diagnostic criterion. The other outstanding study, not included in that table, was Sciotti and colleagues (20) with even better results under more demanding conditions. Evaluation of all of the studies published and clinical training experience indicate that it takes innate ability with adequate training and practice to develop a high degree of reliability in the examination of TrPs and that some muscles are consistently more reliably examined than others [DGS].

## *CASE REPORT*

**Electrical twitch obtaining intramuscular stimulation (ETOIMS) for myofascial pain syndrome in a football player. J. Chu, I. Takehara, T.C. Li, I. Schwartz. Br J Sports Med 38(5):E25, 2004.**

### *Summary*

A 21-year-old male college football wide-receiver with a three year history of progressive bilateral chronic lower back pain and tight hip muscles was treated with "electrical twitch obtaining intramuscular stimulation" [ETOIMS]. Electrical twitch obtaining intramuscular stimulation is a treatment technique developed by Dr. Chu that incorporates electrical stimulation with a monopolar electromyographic needle electrode with a diameter of 0.41 mm. The patient presented with slight range of motion limitations in the trunk, a pain score of 8/10 on a visual analog scale, inability to perform a full squat, and myofascial trigger points [TrPs] in the T7-S1 paraspinal muscles and lower limb muscles. Symptoms started after a bilateral fracture of the L5 pars interarticularis associated with heavy weightlifting. Prior to the ETOIMS treatment, the patient had been treated with activity restriction, bracing, physical therapy, and acupuncture. The authors described that electromyography revealed no spontaneous activity, but an increased percentage of polyphasic motor unit action potentials was found at the left L2 and bilateral L5 myotomes.

The patient underwent ETOIMS with manual insertion of a monopolar needle electrode into multiple motor points at 2 Hz for two seconds, using a unipolar negative wave of 20 mA and 0.5 ms pulse duration, supplied by the electromyogram machine. The muscles treated were primarily bilateral lower paraspinals, gluteus maximus, tensor fascia latae, adductor magnus, and rectus femoris. Outcome measures included serial visual analog scale and the ability to flex, abduct, and externally rotate the hips before each treatment. The patient was treated weekly for a total of 23 sessions. After

10 treatments, he was pain-free. Interestingly, hip range of motion improved dramatically in the first two treatments, but never fully recovered. The patient returned for two additional treatments during a period of 2.5 years after the treatment series. The patient became a professional football player after college. He still receives "treatments for continued pain relief and to improve his agility, speed, and endurance."

### *Comments*

Chu and colleagues presented an interesting case of a high-level athlete with persistent pain and limited range of motion, in spite of previous treatments. Clinicians familiar with the treatment of TrPs are probably not surprised by the relief of pain following treatment. However, the lack of further improvement in hip range of motion brings up the question whether other non-muscular structures could be responsible for the persistent limitations, such as arthrogenic or neurodynamic restrictions. The authors did not indicate whether they considered other causes of restricted range of motion.

In the discussion section of this paper, the authors suggested that deeper tissues can only be reached by using a relatively thick [diameter 0.41 mm] needle instead of commonly used acupuncture needles. In the clinical experience of this reviewer, nearly all deeper tissue harboring TrPs can be treated with standard acupuncture needles with a diameter of 0.30 mm. Combining dry needling of especially deeper TrPs with electrical stimulation is supported by many studies on electro-acupuncture and percutaneous [intramuscular] neuromuscular electrical stimulation [JD].

## Otolaryngic myofascial pain syndromes. W.S. Teachey. Curr Pain Headache Rep 8(6): 457-62, 2004.

### *Summary*

The author of this paper is an otolaryngologist, who has integrated the diagnosis and treatment of myofascial trigger points [TrPs] in his practice. He describes that over a period of five months, in 106 of 257 consecutive new patients [41 percent] with complaints of pain, headaches, or ear, nose, and throat symptoms, the chief complaint was attributed to TrPs. Many of these patients had already received multiple ineffective treatments over a long period of time by a variety of medical disciplines. The worst cases included multiple dental extractions, multiple varying types of dental splints, dental occlusal therapy, or temporomandibular joint adjustments.

Teachey reviews many diagnoses commonly seen in any otolaryngic practice. In his experience, sinusitis unresponsive to antibiotics is frequently due to TrPs in the masseter, pterygoids, zygomaticus, or sternocleidomastoid muscles. Patients with ear aches, a foreign body sensation in their ear, "blocked" ears, hyperacusis, hypoacusis, hearing loss, tinnitus, or dizziness with normal otolaryngic and audiometric studies often have active TrPs in the pterygoids, masseters, or the clavicular division of the sternocleidomastoid muscle. Teachey includes a long list of several other common diagnoses such as headaches, nasal pain or congestion, pain or pressure in or behind the eyes, blurred vision, reddening of the conjunctiva, chronic/recurrent "tonsillitis," dysphagia, odynophagia, burning sensation, throat "congestion"; throat "drainage," voice irregularities, chronic and recurrent pain in the area of the parotid or submaxillary glands, parotitis, and submaxillary sialadenitis, among others.

The paper includes five pertinent case studies that further illustrate the importance of considering TrPs in the differential diagnosis. Teachey warns that in spite of the impressive number of patients with myofascial dysfunction, true disorders suggesting ear, nose, throat, and sinus pathology must first be considered.

### *Comments*

To the best of our knowledge, this is the first paper that describes in detail how common clinically relevant TrPs are seen in an otolaryngic practice. Based on personal communication with the author, his practice has changed considerably since he has included TrPs in his list of common diagnoses. After completing extensive training in the diagnosis and treatment of myofascial pain, Teachey uses a multidisciplinary approach as described in this important paper [JD].

## *BRIEF REVIEWS*

**Myofascial pain: diagnosis and management. S.B. Graff-Radford. Curr Pain Headache Rep 8(6):463-7, 2004.**

This article succinctly reviews the pathogenesis of myofascial pain with emphasis on both central mechanisms with peripheral clinical manifestations. The author includes a description of an integrated six-week management approach that involves stimulating central inhibitory mechanisms through pharmacology and behavioral techniques and simultaneously reducing peripheral inputs through physical therapies including exercises and trigger point-specific therapy. Patients experienced a 90 percent reduction in pain and a 90 percent reduction in their analgesic use [JD].

**Myofascial pain syndrome and its treatment in low back pain. P.P. Raj, L.A. Paradise. Semin Pain Med 2(3):167-174, 2004.**

Several reasons make this review article important. First, it is written by a well-known pain specialist, who has published several authoritative text books on pain management (21,22). Second, it is included in an excellent and up-to-date issue on low back pain of a respectable pain management journal, which may indicate that the recognition of myofascial pain [MPS] and trigger points [TrPs] is gaining ground.

The authors provide a detailed overview of the diagnostic features of MPS and TrPs, the physical examination, and treatment with special emphasis on low back pain. They emphasis that MPS is commonly overlooked, and advocate, that when TrPs are properly recognized, patients with low back pain can be treated effectively.

At times the article seems a bit outdated, especially in the sections on diagnostic criteria and pathogenesis. In the current thinking, the essential criteria for identifying TrPs include the presence of a taut band, exquisite spot tenderness in that taut band, the patient's recognition of the pain complaint, and a painful limit to full stretch (5). The section on pathogenesis is fairly generic and does not review the updated integrated trigger point hypothesis (5). Overall, it is encouraging that the article is included in a prominent pain management journal [JD].

**Nichtmedikamentöse Therapie myofaszialer Schmerzen [in German: Non-pharmacological therapy of myofascial pain]. C. Gröbli, B. Dejung. Schmerz 17:475-480, 2003.**

This paper describes in detail a systematic manual therapy approach for both acute and chronic myofascial pain conditions and myofascial trigger points [TrPs] developed in Switzerland by the co-author Dr. Beat Dejung. The authors suggest that especially in chronic pain conditions special attention must be paid to connective adhesions associated with TrPs, which they speculate may form as a result of local edema in the region of TrPs. The manual therapy approach combines TrP compression with active contractions of the muscle treated at that time, localized stretches in the direction of the muscle fiber directly in the vicinity of TrPs, myofascial release and muscle play techniques, and superficial and deep dry needling. The paper concludes with the results of a non-randomized, non-blinded, single center study of 84 consecutive patients with low back pain who were treated with the outlined manual therapy approach. Patients had chronic pain ranging from six months to 30 years with an average of 4.4 years. The main outcome measure consisted of a pain score on a visual analog scale. As a result of the treatments, the average pain score went down from an initial 6.6 to 3.7. The authors concluded that manual trigger point therapy is effective. Of course, the lack of a control group, lack of randomization, and lack of blinded evaluators make this conclusion a bit speculative. We hope the authors will take this pilot study as an incentive to undertake a more rigorous scientific study evaluating the effects of their manual therapy approach [JD].

**Points détente et acupuncture: approche neurophysiologique [in French: Trigger points and acupuncture: a neurophysiologic approach]. S. Cardinal. Montreal, Centre collégial de développement de matériel didactique, 2004.**

Although the Trigger Point Manuals have been translated into French (23,24), this new

book is a welcome up-to-date addition to the limited number of publications about myofascial trigger points [TrPs] available in the French language. The author is an accomplished acupuncturist with great familiarity of the current thinking about TrPs. The book aims to introduce acupuncturists to the concepts of myofascial pain and TrPs as a complimentary approach in the treatment of their patients suffering especially from chronic pain problems.

The book is divided into four sections. In the first section, Cardinal succeeds in accurately explaining the Integrated Trigger Point Hypothesis in much detail. Common symptoms of myofascial pain are reviewed, followed by the principles of examination and diagnostics. The second section features current neurophysiologic insights. Cardinal skillfully integrates TrP referred pain patterns and nociceptive pathways with known acupuncture theory and meridians. In the third section, the author reviews TrPs with other musculoskeletal pain syndromes, including fibromyalgia, tendonitis, neuropathies, etc. The last section provides an outstanding review of various needling approaches according to Travell, Baldry, Gunn, and Seem.

This excellent book deserves to be translated into other languages to make it accessible to the non-French speaking among us [JD].

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