

# Myofascial Pain Syndromes— Trigger Points

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## *INTRODUCTION*

In this review, Treaster et al. explored the development of myofascial trigger points [TrPs] during computer work including a novel use of power spectrum analysis of surface electromyographic recordings related to TrPs. Fernandez et al. found that ischemic compression and transverse friction massage of TrPs were equally and statistically significantly effective. Fruth presented a remarkable case report that demonstrates eloquently close interaction between articular dysfunctions and TrPs. Her detailed history and five-year results serve as a control. This is one of the few TrP articles that have rarely appeared in the Physical Therapy journal. Other authors considered the TrP origin of symptoms from the lumbar multifidi, abdominal wall, and of chronic pelvic pain, headache, and toothache. Each article review indicates whether it is prepared by Simons [DGS] or Dommerholt [JD].

## *CLINICAL STUDY*

**Treaster D, Marras WS, Burr D, Sheedy JE, Hart D: Myofascial trigger point development from visual and postural stressors during computer work. *J Electromyogr Kinesiol* 16(2): 115-124, 2006.**

## *Summary*

This study explored the development of myofascial trigger points [TrPs] in 16 female computer operators [19-29 years of age, mean 22.8 years]. Each subject was asked to type as accurately as possible for 32 minutes using a typing program under four different visual and postural conditions. The four tests were conducted on different days. Prior to the typing task, a clinical specialist examined the subjects' bilateral trapezius muscles for the presence of TrPs. The skin overlying trapezius TrPs was marked with ink. Both the examiner and subjects rated the degree of tenderness for each TrP using a six-point scale. In addition, the examiner rated the TrPs based on muscle fiber tautness and the subject's "jump" response to manual pressure or palpation. All TrPs in the trapezius, rhomboid, levator scapula, sternocleidomastoid, scalene, and deltoid were manually released using a combination of percussion, stretch, and relaxation techniques. At the beginning of the test procedures, the subjects had full range of motion and no pain or muscle tightness. The clinical examiner was blinded to the experimental procedure.

A pair of surface electrodes was placed around the ink marks. The researchers used cyclic changes in the median frequency to determine the development of TrPs combined with a

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re-assessment by the clinical examiner and feedback from the test subjects immediately following the typing task. Changes in median frequency of at least 5 Hz, but less than 30 Hz, followed by a reversal of at least 5 Hz were classified as “cycles.” High visual stress conditions resulted in greater TrP development and sensitivity in the right trapezius muscle. The combination of high visual stress and low postural stress conditions was characterized by significantly fewer cycles in the mean frequency, when compared to low visual and low postural stress or to high visual and high postural stress conditions. Interestingly, the conditions with higher visual stress also corresponded to more TrP development and greater pain associated with TrPs. The researchers concluded that TrPs provide a useful explanation for development of pain following low level static exertions seen with computer use.

### **Comments**

This is a very interesting study that combines current work-related myalgia research with TrP research. The authors creatively applied research by McLean and colleagues on the cycling nature of median frequencies (1) and linked the results to a clinical assessment of TrPs. Median frequency cycles are thought to be related to regulation of motor unit recruitment in an effort to prevent localized muscle fiber fatigue. With low level static exertions—as seen with computer workers—it is likely that certain muscle fibers are selectively overloaded, consistent with the so-called “Cinderella hypothesis” developed by Hägg (2). According to Hägg, during low intensity tasks the normal substitution of motor units may not occur, resulting in continuous activity, which eventually may lead to damage to these motor units (2,3). When substitution does not occur, the number of median frequency cycles should decrease (1). Hägg’s Cinderella hypothesis has been confirmed in several studies, but has never before been applied to TrPs (4-6). This reviewer postulated a link between the Cinderella hypothesis and TrPs during the 2005 Focus on Pain conference in Philadelphia, Pennsylvania, United States of America.

The Cinderella hypothesis provides a seemingly excellent match with the integrated trig-

ger point hypothesis. Sustained contractures may lead to local hypoxia, which according to the integrated trigger point hypothesis would result in the development of TrPs. Myofascial trigger points are usually associated with some degree of muscle overload, which may be acute, sustained, or repetitive (7). In a recent review, it was postulated that TrPs may develop with eccentric or sub-maximal concentric contractions (8). The Cinderella hypothesis offers a likely explanation why TrPs may develop following relatively low level muscular contractions.

The authors of the current study incorporated this rationale into their study design. They made the assumption that frequency cycling is indeed indicative of TrP development. Muscle contractures at TrPs sites were assumed to fatigue associated motor neurons and therefore, reduce the number of median frequency cycles. While this is an interesting and intriguing assumption, it may also point to a weakness in the study design. At this point, there is no evidence that median frequency cycling can be linked to the development of TrPs, and this study supports this assumption only partially. To counter this criticism, the authors incorporated the skills of an expert clinician to manually examine the subjects for the presence of TrPs. In addition, they solicited the subjects’ rating of pain. However, the examiner’s subjective rating of muscle fiber tautness and the subject’s “jump” response to manual pressure or palpation does not appear to be valid measurement and it is not clear from the study to what extent this rating was applied in the final assessment.

The authors found a correlation between median frequency cycling and the development of TrPs with high visual and low postural conditions, but not with any of the other scenarios. Consequently, this study does not confirm that median frequency cycling is a reliable indicator of TrP formation. Future studies are needed to explore this fascinating area. The manual examination combined with the subjects’ subjective rating did offer support that low level static exertions may lead to the activation of TrPs. This is an important finding that should be considered in other ergonomic studies of work-related myalgia [JD].

**Cornwall J, Harris AJ, Mercer SR: The lumbar multifidus muscle and patterns of pain. *Manual Therapy* 11(1): 40-45, 2006.**

**Summary**

Fifteen healthy volunteers [11 males, four females; 24-45 years of age, mean 32 years] were included in this study of pain patterns of the lumbar multifidus opposite the spinous process of L5. Each subject received two subsequent injections into the L4 band of the multifidus muscle. One injection consisted of 0.3 ml five percent hypertonic saline, while the placebo injection consisted of isotonic saline. The injector and subjects were blinded to the injectable. The first injection was randomly assigned to one side. The second injection was performed five minutes later to the contralateral side. Following each injection, subjects were asked to describe the intensity and location of any sensations. The distribution of pain was mapped out and checked by each subjects.

None of the subjects reported local or referred pain following injections with placebo isotonic saline. Following hypertonic saline injections, all subjects reported local pain and 13 out of 15 subjects reported referred pain into either the anterior or posterior thigh. The researchers compared the findings with previously established patterns of local and referred lumbar pain and found similarities with patterns from the L3-4 interspinous ligaments, multifidi, zygapophyseal joints, the medial branches of the lumbar dorsal rami, and trigger point referred patterns. They concluded that there were many similarities between described patterns with the exception of myofascial trigger point [MTrP] referred pain patterns as reported by Simons, Travell and Simons (7).

**Comments**

This study confirms that the lumbar multifidus muscles can be a source of local and referred pain. Although the researchers did not determine much overlap between the referred pain patterns found in this study and the TrP referred pain patterns described by Simons, Travell and Simons, this study does contribute to the current knowledge base and raises ques-

tions about the accuracy of referred pain patterns in the *Trigger Point Manual*. Establishing referred pain patterns requires a detailed scientific approach as was used in this and in similar previously published studies (9-14). Lumbar multifidus muscles cannot be palpated directly and the accuracy of needle placement may be a determining factor. It is not known on many subjects the TrP referred pain patterns are based and how accurate Travell was in her needle placement when she established the lumbar multifidus referred pain patterns. It is conceivable that not all multifidus TrPs and associated referred pain patterns have been captured. To the best of our knowledge, there are no systematic studies of the lumbar multifidus TrP referred pain patterns. The referred pain illustrations of lumbar multifidus TrPs in the *Trigger Point Manual* probably only represent a few common examples and do not reflect all possibilities. Swiss authors Dejung, Gröbli, Colla, and Weissmann have described different TrP referred pain patterns of the lumbar multifidus muscles based on a total of 43 subjects (15). They determined extensive posterior thigh and leg referred pain patterns, which were very similar to the referred pain patterns in this study. However, these patterns were not derived using a systematic approach either. More studies are needed to establish all muscle referred pain patterns using a scientific methodology [DGS and JD].

**Fernández-de-las-Peñas C, Alonso-Blanco C, Fernández-Carnero J, Miangolarra-Page JC: The immediate effect of ischemic compression technique and transverse friction massage on tenderness of active and latent myofascial trigger points: A pilot study. *J Bodywork Movement Ther* 10(1): 3-9, 2006.**

**Summary**

This comparison of two manual treatments for myofascial trigger points [TrPs] in the upper trapezius muscle in two matching randomized groups of 20 subjects was blinded, but there was no control group. Diagnostic criteria for TrPs were a tender spot in a taut band that responded to snapping palpation with a twitch response and pressure on it reproduced the typi-

cal referred pain pattern that was recognized as familiar if the TrP was active. The ischemic compression treatment involved the application of pressure on the TrP until the patient felt pressure and pain. That pressure was maintained until the sensation decreased by 50 percent when pressure was again increased to the pain threshold, and the procedure continued for 90 seconds. Transverse friction massage was applied slightly painfully across the fiber direction as recommended by Cyriax for three minutes. Both groups showed statistically significant post-treatment improvement in both decreased visual analog scale pain readings and increase in pain pressure thresholds of TrP tenderness. The results were very similar and the authors concluded that the two techniques were equally effective in reducing TrP pain and tenderness. They also noted that follow up data would be very helpful and that the lack of a control group negates an assumption that a cause and effect relationship exists between the treatments and the statistically favorable results. However, they also cite a study that found that ischemic compression results are superior to sham treatment.

### *Comments*

The authors are to be commended for a well-executed study that unfortunately lacks the critically important control group to warrant unreserved acceptance by discriminating readers. It is very unlikely that the favorable results to both treatments in this study were due to placebo effect, which is usually only 30 percent effective at most and temporary. Long term follow up helps greatly to minimize the mistake of discarding valuable findings because of the possibility of placebo effects. In clinical practice, additional placebo effect is of benefit to the patient, if the basic therapy is also effective. This paper was apparently based on patients who had been receiving appropriate treatment prior to the study. In many studies, the history of multiple ineffective treatments by multiple previous providers establishes that there is no natural healing process involved and that efficacy of the treatment being reported is more than a placebo effect because it should have been evident with the previous ineffective or only temporarily helpful treatments. The com-

mon occurrence of untreated chronic TrPs indicates that one cannot count on a natural healing process.

The authors gave equal credibility to the two possible therapeutic mechanisms. One was based on the integrated hypothesis, which is accumulating substantiating research findings (16). The other was postulated by Hou et al. suggesting that pain relief from pressure treatment may result from reactive hyperemia in the TrP region, or from a spinal reflex mechanism for the relief of muscle spasm (17). The Hou proposal of reactive hyperemia relieving muscle spasm has two major problems. We avoid the term ischemic compression, because that much pressure is not recommended and is seldom used in current research papers so that the treatments used in this paper should not have caused reactive hyperemia. The proposed relief of muscle spasm is illogical because electromyographic studies reveal no muscle spasm associated with taut bands and TrPs (18). The tension is due to non-electrogenic muscle shortening [physiological contracture] for some reason.

Although not cited by the authors, one paper does describe in detail how the pressure applied by either method lengthens shortened sarcomeres that produce the taut-band tension (19). Rather than cross fiber massage, Hong suggested that massage strokes starting at the TrP and progressing along the taut band away from it would augment normalization of sarcomere lengths [personal communication]. I have found it effective. To my knowledge no one as compared the efficacy of this technique to the other two reported in this paper [DGS].

**Nazareno J, Ponich T, Gregor J: Long-term follow-up of trigger point injections for abdominal wall pain. *Can J Gastroenterol*: 19(9) 561-565, 2005.**

### *Summary*

This retrospective study of 110 consecutive patients of a university-based gastroenterology clinic aimed to investigate the long-term outcome of trigger point [TrP] injections. The study was limited to patients with abdominal wall pain, which was defined as 1. pain fixed in

location or very localized, and 2. superficial or point tenderness less than 2.5 cm in diameter, or 3. a positive Carnett test [tenderness that increases with abdominal muscle tensing]. Of the 110 patients, 89 patients met the criteria for abdominal wall pain. Eighty-four percent of the patients were female. The mean age was 42 with a range from 12 to 88 years. The most common pain locations were the right hypochondrial [30 percent], right iliac [25 percent], and epigastric [19 percent]. Fifty percent of the subjects reported referred pain, 73 percent presented with gastrointestinal [GI] symptoms, and 39 percent had a scar in the immediate proximity of the pain location.

Eighty-eight percent of the subjects received abdominal wall TrP injections with a combination of either bupivacaine [0.25 percent to 0.50 percent] or lidocaine [one percent to two percent] and betamethasone. The remaining 12 percent were injected with either bupivacaine or lidocaine. A maximum of 15 ml was injected at any one time. While most subjects received only one injection, 36 percent of the subjects received repeated TrP injections ranging from one to 27 over variable amounts of time. The mean length of follow-up was 25 months with a range of 0.5 to 146 months.

Eighty-nine percent reported some or complete early relief [within two weeks of the injection]; 77 percent reported some or complete relief at follow-up. Of interest is that only 35 percent of the subjects who did not meet the criteria for abdominal wall pain experienced some or complete relief. The authors concluded that the presence of abdominal wall pain was a strong predictor of long-term relief. The presence of GI symptoms had a significant negative impact on pain relief. Ninety-five percent of subjects without GI symptoms reported pain relief compared to only 71 percent of subjects with GI symptoms. There were no differences between males and females. Patients with surgical scars experienced the same degree of relief, but needed significantly more injections. Furthermore, the addition of a steroid did not change the degree of pain relief.

### **Comments**

The authors are commended in undertaking this study. Abdominal wall TrPs have been

shown to be relevant in the etiology of low back, abdominal, and pelvic pain syndromes (20-23). At first glance, this retrospective study appears to confirm the immediate and long-term effectiveness of TrP injections in abdominal trigger points. A reported 89 percent and 77 percent experienced immediate or long-term relief respectively. However, the true effects of the injections are difficult to interpret. The authors did not report how they defined TrPs or TrP injections. What criteria did they use to palpate abdominal TrPs? Did they determine the presence of a taut band and the most tender region within that taut band as suggested by Simons, Travell and Simons (7)? The authors do not mention whether they attempted to elicit local twitch responses or whether they infiltrated the general vicinity of a point of tenderness. Intramuscular TrP injections are only effective if the needle elicits local twitch responses (24). The number of follow-up injections and the time line of follow-up appointments varied greatly. With such a wide range, retrospective chart reviews do not provide sufficient assurances that reported long-term relief is primarily due to any particular intervention.

The authors expressed surprise about the lack of effectiveness of adding a steroid to local anesthetics. This may indicate a lack of familiarity with the integrated trigger point hypothesis (7). Steroids are most effective when treating inflammatory conditions and there is no current evidence that TrPs are associated with local inflammation. The use of steroids is not recommended for the injection of TrPs and may actually lead to destruction of muscle fibers. There is also no evidence that using long-acting anesthetics are more effective than short acting anesthetics in the treatment of TrPs. Long-acting anesthetics may actually be more myotoxic. The authors reported using a one percent to two percent lidocaine solution. A recent study indicated that using a 0.25 percent lidocaine solution is more effective (25), while reducing its myotoxicity. The observation that subjects with a surgical scar required significantly more TrP injections is consistent with Lewit and Olsanska's notion that scars are a common source of myofascial pain (26). Prospective studies on the effectiveness of treat-

ments directed at TrPs are very much needed [JD].

### **CASE REPORT**

**Fruth S: Differential diagnosis and treatment in a patient with posterior upper thoracic pain. *Phys Ther* 86(2): 254-268, 2006.**

#### **Summary**

A 35-year-old youth minister had pain in the posterior upper thoracic region for four months beginning two days after sitting on the bleachers for three hours at an ice hockey game. The pain localized between the right scapula and the spine, increasing during the next six weeks. Cyclobenzaprine HCL and naproxene and physical therapy [PT] elsewhere did not help. Two months later, three weeks of different PT treatment that included exercise, modalities, spine mobilization, and massage did not help. His physician increased cyclobenzaprine dosage, ordered radiographs of the thoracic and cervical spines and of the right shoulder, and referred patient to the author for another try at physical therapy. Radiographs were negative.

The patient complained of constant shoulder-area pain, limited ability to play with and care for his children, participate in softball, and disturbed sleep due to pain when he changed position during the night. Specific daily functions were tested with a comprehensive, simple questionnaire using a five-point scale for each function: 0-4 [full normal function]. Initially rating of functions by the patient: use your hand with arm at shoulder level 1; dress yourself 2; sleep 0; use arm overhead 1; throw ball overhead 2; perform child care 1; perform normal sport 0; etc. Total score 36 /72.

Initial examination revealed that manual muscle testing for strength was not feasible because of limitation by pain. There was painful limited mobility of the right costovertebral and costotransverse joints at ribs 3 through 6, and trigger points [TrPs] in the right middle trapezius and rhomboid muscles. Subsequent examination identified TrPs also needing treatment in the right pectoralis major, serratus posterior superior, serratus anterior, and lower

trapezius. Treatment began with digital pressure applied to the middle trapezius and rhomboid TrPs that provided sufficient pain relief that she could then start to release of the joint restrictions. After seven PT sessions in four weeks that focused on releasing articular dysfunctions and inactivating all painful and function-inhibiting TrPs, the patient had return of full function [total function score, 71/72—only sleep was slightly disturbed occasionally] and remained that way through the next five years. The author recommended attention to the lack of research on the causes of pain and dysfunction in the thoracic area, on the reliability of detecting TrPs, and on the efficacy of joint mobilizations and TrP release.

#### **Comments**

This is the first article specifically addressing TrPs to appear in the PT journal for over five years—a most welcome appearance. It is very well and knowledgeably written with extensive older references. This case report eloquently demonstrates the critically important interaction between articular dysfunctions and TrPs and demonstrates how important it is to address each with appropriate diagnostic techniques and treatment. Shoulder pain problems like this commonly involve many of the shoulder-girdle muscles, and usually respond only when all of them are included in the treatment program, as demonstrated in this case. The function evaluation instrument used is novel but looks very practical and effective. It nicely verified the effectiveness of the author's diagnosis and treatment, especially after the history of repeated failure of routine physician and PT approaches to this common but usually overlooked type of musculoskeletal pain and dysfunction.

The history of multiple ineffective treatments by multiple previous providers establishes that there is no natural healing process involved and that efficacy of this kind of treatment is more than placebo effect, which if important, should have been evident with the previous treatments. The favorable five-year follow up confirms the remarkable effectiveness of these treatments compared to the pre-treatment history.

The TrP source reference was the 1983-first edition of volume 1 of the *Trigger Point Manual*, not the much-updated 1999-second edition (7). As a result, the diagnosis of TrPs included the jump sign, which is redundant with the tenderness test, and a crude measure of painfulness compared with the well-established visual analog scale measure. I strongly endorse the author's concluding recommendations [DGS].

### BRIEF REVIEWS

**Samraj GPN, Kuritzky L, Curry RW: Chronic pelvic pain in women: Evaluation and management in primary care. *Comp Ther* 31(1): 28-39, 2005.**

In this article, the authors provide an excellent overview of the prevalence and pathophysiology of chronic pelvic pain. They emphasize that chronic pelvic pain is a syndrome with a complex multi-faceted etiology. Myofascial pain is included in the list of most common pathologies along with endometriosis, interstitial cystitis, irritable bowel disease, and pelvic adhesions. Myofascial pain may present as vulvar vestibulitis, vaginismus, levator ani syndrome, pelvic floor tension myalgia, or pudendal neuralgia, among others. As part of a comprehensive medical history and examination, the authors recommend a detailed physical examination of the low back, spine, abdomen, and groin, as previously outlined by Prendergast and Weiss (23,27) [JD].

**Jensen S: Neck related causes of headache. *Aust Fam Pract* 34(8): 635-639, 2005.**

From Australia comes this brief introductory review of common causes and a basic assessment of cervicogenic headaches. Jensen, a senior lecturer in musculoskeletal medicine, suggests that because of a lack of musculoskeletal skills in the medical community, cervicogenic headaches frequently go undiagnosed and are confused with migraine or tension-type headaches. Each structure innervated by any of the first three spinal nerves may be a source of cervicogenic headaches, including the median and lateral atlanto-axial joint,

the atlanto-occipital joint, C2/3 and C3/4 zygapophyseal joints, and myofascial trigger points [TrPs] of the suboccipital, upper posterior cervical, upper prevertebral muscles, trapezius, and sternocleidomastoid muscles. The author summarizes key components of the examination, starting with an inspection, followed by a movement assessment, and palpation.

Unfortunately, the article does not include much detail about how to perform the physical examination of the various spinal structures and cervical muscles and it is doubtful that the average family practitioner will be able to implement the recommended approach in clinical practice. Jensen maintains that "the clinical skills necessary to evaluate the neck and surrounding structures are relatively easy to grasp [. . .] and well within the realm of a busy GP." However, in our experience learning and mastering manual therapy skills and techniques, including joint assessments and TrP examinations, does take considerable training and practice. Until family practitioners have completed such training, it is unlikely that they will develop the necessary musculoskeletal skills to properly identify and differentiate the articular dysfunctions and TrPs, and therefore, cervicogenic headaches from migraine and tension-type headaches [JD].

**Timmermans F: Aktueller Stand der Überlegungen zum myofaszialen Schmerzsyndrom [in English: Current thoughts on myofascial pain syndrome]. *Physioscience* 2: 1-8, 2006.**

Timmermans contributed this general overview of the current thinking about myofascial trigger points [TrsP] based on a fairly extensive review of the literature. After a brief overview of the most common TrP hypotheses, the author continues to describe the neurophysiology of local and referred TrP pain, the examination techniques, and common treatment options, including dry needling. The article attributed the motor endplate hypothesis incorrectly to Hubbard and Berkhoff as the author did in a Dutch version of a similar paper (28). Otherwise, this is a reasonable comprehensive basic review of the current thinking about TrPs [JD].

**Kim ST: Myofascial pain and toothaches. Aust Endod J 31(3): 106-110, 2005.**

This review, also from Australia is well written, insightful, and thoughtful, but some critical references are anachronistic. The most serious is the extensive dependence on the 1983 edition of the *Trigger Point Manual* instead of the 1999-second edition. The author defined myofascial pain in terms of myofascial trigger points and noted that myofascial pain information is fragmented and poorly understood in dentistry. A major reason for this is the 1992 seminal dentistry article that defined myofascial pain only as tenderness of multiple masticatory muscles and eliminating any palpatory findings of a taut band or related spot tenderness that are distinguishing characteristics of trigger points (29). The dental literature has been crippled by this oversight ever since. Clinical features are well described, but the pathophysiology is well described only as of our 1983 understanding of it. The pathophysiology can now be identified by the integrated hypothesis (18) with noteworthy support from basic research (30) and is still debated, but substantiated with impressive research (16). In this review, diagnosis was well described and illustrated, but management missed all of the effective manual treatment methods covered in subsequent publications. Patients can be taught to use many of these treatments for themselves [DGS].

**Visser B, van Dieën JH: Pathophysiology of upper extremity muscle disorders. Electromyog Kinesiol 16: 1-16, 2006.**

This communication from The Netherlands focuses an extensive review of injury mechanisms on the possible causes of observed clinical characteristics of upper extremity disorders. Myofascial trigger points [TrPs] are mentioned along with selective recruitment and overloading of type I motor units, intracellular  $Ca^{2+}$  accumulation, impaired blood flow, reperfusion injury, blood vessel-nociceptor interactions, myofascial force transmission, intramuscular shear forces, and impaired heat shock response. None of these showed complete proof of being a cause. A succinct

summary of TrP clinical characteristics and of the integrated hypothesis expressed uncertainties as to the causal role of muscle activity and task requirements in relation to abnormal endplate activity (18). In addition to the proposed influence of an autonomic central process we now have evidence of significant immune system involvement in pain production (16).

Of the eight mechanisms, the authors favored continuous low or medium intensity muscle activity with  $Ca^{2+}$  accumulation, then intermittent high intensity activity, and finally localized forceful sarcomere lengthening, all of which have been identified clinically with activation of TrPs [DGS].

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