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Myofascial Pain Syndrome—Trigger Points

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Myofascial Pain Syndrome—Trigger Points

Jan Dommerholt, PT, DPT, MPS

INTRODUCTION

Myofascial trigger points [TrPs] continue to attract clinicians and researchers from around the world. This review includes contributions from Brazil, Canada, Denmark, Spain, Switzerland, Taiwan, and the United States. A paper by Chang, Chen, and Chang illuminates an important aspect of the integrated TrP hypothesis and suggests that a disintegration of spinal motor neurons may contribute to the development of TrPs. Other papers investigated TrPs and whiplash injury, urinary incontinence, muscle cramps, shoulder pain, and tinnitus. The research team of Fernández-de-las-Peñas explored preliminary clinical prediction rules with trigger point therapy, which may assist in determining which patients are most likely to benefit from trigger point therapy.

RESEARCH STUDIES

Ettlin T, Schuster C, Stoffel R, Brüderlin A, Kischka U: A distinct pattern of myofascial findings in patients after whiplash injury. Arch Phys Med Rehabil 89:1290–1293, 2008.

Summary

This study from Switzerland compared the prevalence and distribution of TrPs in the neck and shoulder muscles of 47 patients with whiplash-associated disorder, 21 patients with fibromyalgia syndrome [FMS], 17 with chronic cervical syndrome, 15 with endogenous depression, and 24 control subjects [total number of patients = 100]. Patients were recruited from three treatment centers in Switzerland and Germany.

They were symptomatic for at least 6 months. Exclusion criteria were age over 60 years, insufficient knowledge of German, and significant internal or neurologic diseases.

A physical therapist specialized in the manual diagnosis and treatment of TrPs performed all examinations. The following muscles were included in the examination: semispinalis capitis, trapezius pars descendens, levator scapulae, scalenus medius, sternocleidomastoid, and masseter. The therapist was blinded to the diagnosis of the subjects. Criteria for TrPs included a palpable hardening in the muscle belly, pressure pain, referred pain with manipulation of the hardening, and recognition of the elicited pain. Three out of four criteria were required to diagnose a TrP. In addition, all subjects completed a visual analog scale for general pain level and the German version of the Beck Depression Index.

The results showed that the prevalence of TrPs in the semispinalis capitis muscle was significantly higher [85.1 percent] in the patients with whiplash-associated disorder than in any other group of patients with 53.2 percent with bilateral TrPs. The prevalence of TrPs in other muscles did not differ from the patients with fibromyalgia syndrome [FMS] or chronic cervical syndrome, but was significantly higher compared to the patients with depression and the healthy controls. Depression had no impact on the outcome. Pain levels were higher for the patients with whiplash-associated disorder and FMS compared to the other groups.

Comments

The researchers included one muscle each from the upper cervical spine, lower cervical

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spine, neck, shoulder girdle, and face, and found a distinct pattern of distribution of TrPs specifically for whiplash injuries. They acknowledged that in future studies other muscles should be examined as well. Overall, this is an excellent study documenting that TrPs are common after whiplash injury, and that there may be distinct differences between groups of patients. Patients with whiplash-associated disorder should always be examined and treated for TrPs.

Ge H-Y, Fernández-de-las-Peñas C, Madeleine P, Arendt-Nielsen L: Topographical mapping and mechanical pain sensitivity of myofascial trigger points in the infraspinatus muscle. *Eur J Pain* 12:859–865, 2008.

Summary

This study from Denmark included nineteen patients with unilateral shoulder pain. The researchers divided the area overlying the bilateral infraspinatus muscles into 10 adjacent areas of 1 cm² and measured the pressure pain threshold in each area. Next, an acupuncture needle was inserted into each area five times in different directions to elicit local twitch responses or referred pain.

The study revealed several interesting findings. First, the pressure threshold was significantly lower in the infraspinatus muscle on the painful side [$p = 0.001$]. Second, the pressure threshold was significantly lower in the midfiber region of the muscle compared to other parts [$p < 0.05$]. Third, multiple TrPs were identified in the infraspinatus muscle on the painful side. Fourth, multiple latent TrPs were identified bilaterally. Fifth, the pressure threshold of active TrPs was much lower than the latent TrPs and again much lower than the non-TrPs.

The researchers concluded that bilateral mechanical hyperalgesia is common with unilateral shoulder pain. It is likely that peripheral sensitization plays a significant role in chronic myofascial pain. Lastly, pressure pain threshold topographical mapping and dry needling are sensitive techniques to identify TrPs.

Comments

Part of the appeal of this study is its simplicity. The design of this study is not all that sophisticated, yet, the results are dramatic and

they confirm the observations of clinicians. Trigger points occur bilaterally in patients with unilateral shoulder pain and are more prevalent in the midfiber region of the muscle. The study also confirms that TrP dry needling and pressure threshold measurements can be used to identify trigger points.

Chang C-W, Chen Y-R, Chang K-F: Evidence of neuroaxonal degeneration in myofascial pain syndrome: A study of neuromuscular jitter by axonal microstimulation. *Eur J Pain* 12:1026–1030, 2008.

Summary

Researchers from Taiwan used stimulated single-fiber electromyography [SFEMG] with 23 patients with TrPs in the upper trapezius and levator scapulae muscles and with 16 controls. The SFEMG is a sensitive method to measure neuromuscular jitter, assess the functional integrity of peripheral nerves, and determine the stability of the neuromuscular transmission function. The study aimed to investigate whether neuroaxonal degeneration and degeneration of motor neurons are common in patients with myofascial pain.

Subjects had chronic myofascial pain with a disease duration ranging from 6 months to 8 years [mean 2.6 years]. Subjects with a history of diabetes, uremia, neck or shoulder trauma, cervical radiculopathy, neuritis, myasthenia gravis, myasthenic syndrome, or muscular weakness related to other neuromuscular diseases were excluded from the study. Jitter or the mean consecutive difference [MCD] was calculated as the mean of 30 consecutive interpotential intervals between stimuli and single-fiber potentials.

Subjects with TrPs had significantly higher MCD values than healthy controls with an abnormal percentage of 74.3 and 70.7 for the trapezius and levator scapulae muscles, respectively [$p < 0.01$], which may indicate instability of the neuromuscular junction, post-synaptic damage, or degeneration of motor neurons. The researchers found a positive correlation between the jitter and the duration of myofascial pain, which may support the development of progressive neuronal degradation with axonal neuropathy in more chronic cases. The authors speculated that the noted degeneration of motor neurons might

trigger sensory nerve involvement and hypersensitivity to pain.

Comments

Neuromuscular jitter is produced by fluctuations in the time for endplate potentials at the neuromuscular junction to reach the threshold for action potentials. With a dysfunctional neuromuscular junction, muscle fibers of the same motor unit may not always fire in the same sequence causing jitter. This study confirmed that patients with TrPs had a significantly increased MCD in the trapezius and levator scapulae muscles compared to controls. The integrated trigger point hypothesis is based on dysfunction of the motor endplate and this study suggests strongly that at least part of the endplate dysfunction may be the result of disintegration of spinal motor neurons. The suggested link between motor and sensory neurons is interesting and conceivable, but needs to be supported with further research. The authors made a very important contribution to the understanding of the mechanisms underlying the development of TrPs.

Fernández-de-las-Peñas C, Cleland JA, Cuadrado ML, Pareja JA: Predictor variables for identifying patients with chronic tension-type headache who are likely to achieve short-term success with muscle trigger point therapy. *Cephalgia* 28:264–275, 2008.

Summary

Thirty-five subjects with chronic tension-type headaches were included in this Spanish study, which aimed to develop preliminary clinical prediction rules with trigger point therapy. Clinical prediction rules identify those patients at the time of the initial evaluation who are most likely to benefit from a particular intervention. In addition to evaluating range of motion, head posture, pressure thresholds, and total tenderness, all subjects were examined for active TrPs in the upper trapezius, sternocleidomastoid, temporalis, and superior oblique muscles. The therapy program consisted of a combination of different trigger point techniques and a progressive exercise program.

Outcome measures were determined as at least a 50 percent reduction of at least one

headache parameter at 1 week and 1 month after discharge from therapy. Twelve potential predictor variables were entered into a logistic regression, leaving four variables with the strongest predictive value for short-term follow-up, including headache duration [<8.5 hr], headache frequency [<5.5 days/week], bodily pain [<47 on the medical outcomes study 36-item short form (SF-36)], and vitality [<47.5 , on SF-36]. For long-term follow-up, two variables with the strongest predictive value were determined, including headache frequency [<5.5 days/week] and bodily pain [<47 on SF-36].

After 1 week, the chance of a successful outcome was 80 percent with three of four variables present and 87.4 percent if all the four variables were present. After 1 month, if one of two variables was present, the probability of success was 72 percent and if both variables were present, it increased to 84 percent.

Comments

Being able to predict which patients are likely to benefit from trigger point therapy following the initial evaluation is a valuable and potentially cost-saving utility. Clinical prediction rules have been determined for other interventions for back and neck pain. This study is a step in the right direction, but is implicitly limited, because there are no studies that have defined the effectiveness of the therapy program used in this study. The authors acknowledged this limitation and did not draw definitive conclusions and labeled the outcome correctly as “preliminary” prediction rules. It was interesting to note that the number of active TrPs was not identified as a strong predictor of successful outcome.

Ge H-Y, Zhang Y, Boudreau S, Yue S-W, Arendt-Nielsen L: Induction of muscle cramps by nociceptive stimulation of latent myofascial trigger points. *Exp Brain Res* 187(4): 623–629, 2008.

Summary

Fourteen healthy subjects received injections with glutamate or isotonic saline in latent TrPs or non-TrP muscle tissue of the gastrocnemius

muscle in this Danish study on the association between latent TrPs and muscle cramps. Latent TrPs were identified manually and confirmed with electromyography [EMG]. The presence of spontaneous intramuscular electrical activity registered with an EMG-guided injection needle and the absence of surface EMG activity was interpreted as a confirmation of a trigger point. Twenty minutes later, a second injection was delivered in the non-injected site. Pain ratings were continuously recorded on a visual analog scale. Muscle cramp was defined as a significant increase in EMG activity as measured by both the needle and surface EMG electrodes.

The pain associated with glutamate injections was rated as a 6.5 ± 0.68 mm on a visual analog scale compared to 1.8 ± 0.3 mm for the isotonic saline injections. Following the injections, 92.86 percent of subjects who were injected in latent TrPs experienced muscle cramps compared to 0 percent for non-TRP injections. None of the subjects injected with isotonic saline experienced cramps irrespective of the injection site. The researchers concluded that noxious stimulation of latent TRPs might result in muscle cramps.

Comments

Trigger points have associated with muscle cramping in previous papers. The underlying mechanisms of cramps are not well defined. This is the first paper linking latent TrPs to the development of muscle cramps. The authors suggested that activation of nociceptive muscle afferents may electrically induce muscle cramps by increasing the response of group II spindle afferents and the afferent input to motor neurons, realizing that this hypothesis does not explain the induction of muscle cramps with peripheral denervation. Another explanation suggests that noxious stimulation of latent TrPs would decrease inhibitory input to motor neurons and as a result induce muscle cramps. Therapeutically, inactivating TrPs is one possible treatment of muscle cramps.

TREATMENT STUDIES

Hains G, Hains F, Descarreaux M, Bussière A: Urinary incontinence in women treated by ischemic compression over the bladder area; a pilot study. J Chiropractic Med 6:132-140, 2007.

Summary

From Canada comes this clinical study of the effects of ischemic trigger point compression on stress and mixed incontinence. Thirty-three women with urinary incontinence were examined by a chiropractor and randomly assigned to an experimental group [$N = 24$] or a control group [$N = 9$]. Subjects in the experimental group were treated with ischemic compression therapy over TrPs located on the bladder area, or as the authors described, "deep behind the pubis." Subjects were treated three times per week for a period of 5 weeks. Trigger points were compressed starting with light pressure with a gradual increase of pressure to subjects' maximum pain tolerance levels for 15 s. Subjects in the control group received ischemic compression therapy over TrPs in the gluteus maximus, medius, and minimus muscles. Compression was maintained from 5 to 15 s per trigger point. At the conclusion of the study, the subjects of the control group were offered 15 more treatments during which trigger point TrP compression was administered over the bladder area.

Outcome measures included modified versions of the Urogenital Distress Inventory and the Incontinence Impact Questionnaire, which were administered after 15 treatments, 30 days after treatments, 6 months later. Subjects in the experimental group showed a significant decrease on the scores for the questionnaires [from 23.3 to 10.2; $p < 0.001$] compared to little change in the control group [from 25.3 to 22.2]. After 30 days the symptoms decreased further to a score of 6.9 and increased to 11.3 after 6 months. The crossover group did not experience a significant improvement in spite of receiving the same treatment as the experimental group. All subjects were evaluated and treated by the same chiropractic doctor.

Comments

This interesting study provides support for the treatment of TrPs in females with urinary incontinence. The authors speculated that they were treating TrPs in the smooth musculature of the bladder wall, a concept that has not received any support in the literature. They did not mention whether they examined the patients for abdominal wall TrPs, including the pyramidalis muscle, which have been linked to pelvic floor disorders and incontinence. The arguments in support of their assumption are highly speculative. It is doubtful that the authors actually administered ischemic compression. As TrPs are thought to be hypoxic, ischemic compression may not even be desirable.

Bezerra Rocha CAC, Ganz Sanchez T, Tesseroli de Siqueira JT: Myofascial trigger point: A possible way of modulating tinnitus. *Audiol Neurotol* 13:153–160, 2008.

Summary

Ninety-four patients with constant unilateral or bilateral tinnitus during at least 3 months and an equal number of controls were examined for the presence of TrPs in the superficial masseter, splenius capitis, sternocleidomastoid [sternal head], anterior temporaiis, upper trapezius, posterior digastric, scalenus medius, levator scapulae, and infraspinatus muscles using the criteria suggested by Simons, Travell, and Simons and by Gerwin et al. (1, 2). The researchers examined whether palpation of TrPs would alter the nature of tinnitus in loudness or changes in the sound. The evaluation of tinnitus loudness was assessed with a visual analog scale. Palpation was performed once with progressive and sustained deep single-finger pressure up to 10 s.

Trigger points in at least one muscle were much more common in the patient group [72.3 percent] than in the control group [36.2 percent: $p < 0001$]. Laterality was observed in 56.5 percent and modulation of tinnitus occurred in 55.9 percent of subjects in the experimental group. Changes in tinnitus were mostly presented as changes in loudness. Palpation of TrPs in all the muscles included muscle-modulated tinnitus, but much more pronounced in the masseter, followed by the

splenius capitis, sternocleidomastoid, and temporalis. All examinations were performed by the same physical therapist who was not blinded. The authors concluded that both active and latent TrPs are associated with tinnitus.

Comments

Two of the authors have previously published some of their findings (3), but this is their first solid study of the association between TrPs and tinnitus. A next logical study would be to examine the effect of different trigger point-treatment approaches on tinnitus, including manual TrP therapies, dry needling, and injection techniques or stretching, muscle energy, and strain-counterstrain. This is a very encouraging study from Brazil published in an audiology and neurotology journal. Hopefully, medical specialists involved in the diagnosis and treatment of tinnitus will take notice and incorporate TrPs in practices.

REVIEWS & COMMENTS

Malanga G. Wolff E: Evidence-informed management of chronic low back pain with trigger point injections. *Spine J* 8(1): 2008.

Summary

This paper provides a comprehensive review of the TrP literature with an emphasis on TrP injection for the treatment of patients with chronic low back pain. The authors included a brief historical section, general descriptions of injection techniques, some interpretations of insurance companies, and a summary of possible mechanisms of action. To support the use of TrP injections for chronic low back pain, the authors included the findings of several Cochrane reviews.

Comments

It was encouraging that the North American Spine Society sponsored a special issue of the Spine Journal, which included this reasonably well-written article on TrPs as a nonsurgical approach to chronic low back pain. Although the authors advocated the use of TrP injections, they

excluded many recent studies, which substantiate the trigger point concepts and the use of injections. They mentioned that “a few states allow physical therapists to perform dry needling.” Dry needling is within the scope of physical therapy practice in 11 states in the United States, and in many countries around the world, including Australia, Canada, Ireland, the Netherlands, New Zealand, Norway, South Africa, Spain, and the United Kingdom, among others.

The authors noted the limits some insurance companies have imposed on the administration of TrP injections, but they did not comment on the significant discrepancies between arbitrary insurance policies and clinical utility. One of the insurance companies allows up to three injections per year, which for most patients with TrPs and chronic pain conditions is very inadequate. The authors suggested that trigger point injections should be considered when patients with chronic low back pain have failed to respond to medications or a course of active physical therapy. This reviewer would argue that TrP injections or dry needling should be considered early in the treatment of patients with chronic low back pain and not just after other remedies have failed.

Simons DG: New views of myofascial trigger points: Etiology and diagnosis. Arch Phys Med Rehabil 89:157–159, 2008.

This article is a commentary on several studies published in the *Archives of Physical Medicine and Rehabilitation*. Dr. Simons emphasized the importance of the recent studies by Shah et al. of the chemical environment of active TrPs for the integrated trigger point hypothesis (4, 5). The finding of multiple pro-inflammatory inflammatory cytokines, serotonin, and bradykinin, among others, offers support for the notion that noxious chemicals in the immediate vicinity of motor endplates may indeed stimulate nociceptors, contributing to pain from TrPs. The other study by Chen et al. was highlighted for its support of the presence of taut bands using magnetic resonance elastography (6, 7). Although this technique does not have direct clinical utilization, it is nevertheless the first magnetic resonance elastography study of trigger point phenomena. All papers already have been reviewed in this column.

Clark GT: Classification, causation and treatment of masticatory myogenous pain and dysfunction. Oral Maxillofacial Surg Clin N Am 20:145–157, 2008.

What a pleasant surprise to come across this article published by an American dentist, who actually mentioned TrPs in this overview article of masticatory muscle pain! As we have commented many times earlier in this column the dental literature frequently defines myofascial pain as “muscle pain with or without limited mouth opening” following the 1992 temporomandibular disorder criteria by Dworkin and LeResche (8). Clark included basic information about TrPs and briefly discussed the treatment options for trigger points therapy. Hopefully, this paper will contribute to expanding the TMD criteria and include the current knowledge base of TrPs.

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