

Myofascial Pain Syndromes— Trigger Points

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INTRODUCTION

Let's step back a moment for a historical look at what has happened to the quality and quantity of myofascial trigger point [TrP] literature world-wide, since the *Journal of Musculoskeletal Pain* started, including these reviews, in 1993 by comparing all of the articles that were published in the year of 1993 on the subject and eventually reviewed with the TrP articles found and reported for this typical current quarterly review [one fourth of a year]. A total of eight articles were reviewed for 1993, and 18 articles for this quarter. Of those eight articles in 1993, one was a research article on TrPs in a main-line peer reviewed journal. Three were research articles that concerned conditions characteristic of TrPs, but none of them recognized or identified the TrPs as such. Four articles explicitly dealt with TrPs, but were review articles in books or review journals with no new experimental data.

Articles reviewed in this issue explicitly identify TrPs as associated with unilateral migraine, chronic tension-type headache, symptoms of trigeminal neuralgia, low back pain, enigmatic orofacial pain, sexual dysfunction, chronic prostatitis, chronic pelvic pain syndrome, impingement syndrome, frozen shoulder syndrome, subacromial impingement syn-

drome, and hypermobility in fibromyalgia syndrome. It includes several new papers on the association of TrPs with headache. The paper by Ofluoglu et al. provides unprecedented insight into the relationship between the extremely common and incapacitating phenomenon of increased stiffness with increasing age and TrPs. Each article review indicates whether it is prepared by Simons [DGS] or Dommerholt [JD].

CLINICAL STUDIES

Fernandez C, Cuadrado ML, Pareja JA: Myofascial trigger points, neck mobility and forward head posture in unilateral migraine. *Cephalalgia* 26: 1061-1070, 2006.

Summary

The authors examined 20 subjects with unilateral migraine without side-shift and 20 matched controls for myofascial trigger points [TrPs] in the upper trapezius, sternocleidomastoid, temporalis and suboccipital muscles, and illustrated their referred pain patterns. A blinded assessor identified TrPs in these muscles by finding a hypersensitive tender spot in a taut band, a local twitch in response to snap-

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Journal of Musculoskeletal Pain, Vol. 15(2) 2007
Available online at <http://jmp.haworthpress.com>
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doi:10.1300/J094v15n02_11

ping palpation, and reproduction of referred pain typical of each with adaptations for suboccipital muscles. The TrPs were considered active if the subject recognized the referred pain as familiar. Neck mobility was measured with a cervical goniometer.

Migraine patients had significantly more active TrPs [$P < 0.001$] but not more latent ones than healthy controls. The TrPs were more likely to be located ipsilaterally in these migraine patients [$P < 0.01$]. Migraine patients were more likely to have forward head posture by goniometry than controls [$P < 0.001$] and restricted neck flexion/extension [$P < 0.01$]. The active TrPs ipsilateral to the unilateral migraine headaches may be a contributing factor to the initiation and perpetuation of their migraine attacks.

Comments

This well designed, blinded, controlled, prospective study demonstrated eloquently and convincingly that the presence of active TrPs was associated with unilateral migraine. Since this study did not include treatment results, it does not establish them as the cause of the migraine, but clinical experience indicates that, in a considerable number of patients diagnosed as having migraine, effective TrP treatment of all of the contributing TrPs improves very nearly all of them and for some completely relieves their symptoms. Treatment studies are planned next by these authors [DGS].

Göbel H, Heinze A, Reichel G, Hefter H, Benecke R: Efficacy and safety of a single botulinum type A toxin complex treatment (Dysport) for the relief of upper back myofascial pain syndrome: results from a randomized double-blind placebo-controlled multicentre study. Pain 125(1-2): 82-88, 2006.

Summary

In this prospective, randomized, double-blind, placebo-controlled study, 145 subjects [aged 18 to 70 years] were randomly assigned to one of two matched groups. Group 1 [$N = 75$] received botulinum toxin A injections into the ten most painful myofascial trigger points

[TrPs] in several cervical and shoulder muscles, while Group 2 [$N = 70$] received TrP injections with a 0.9 percent sodium-chloride solution. The TrPs were identified using a standardized method based on 1996 operational definition by Simons. They “looked for pain on palpation of cervical and shoulder muscles.” To be included in the study, subjects had to present with myofascial pain syndrome for six to 24 months with at least 10 TrPs in the neck and shoulder muscles. Exclusion criteria were evidence of other specific disorders, previous treatment with botulinum toxin, enrollment in another study, concurrent muscle disease, conditions with medication-induced bleeding, pregnancy, history of drug or alcohol abuse, a body mass index of more than 30 kg/m², or specific back disorders. Medication use was significantly restricted before and during the study.

Subjects kept a pain diary a week before the randomization and for 12 weeks following treatment. After the initial visit, subjects were scheduled every four weeks. The primary outcome measure was the proportion of subjects with mild or no pain at week 5 based on the mean score on their ordinal self-rating pain scale. Secondary outcome measures included changes in pain intensity, duration of pain, the number of pain-free days per week, duration of sleep, the number and pain intensity of TrPs, and the time to improvement in pain.

At the five-week assessment point, 51 percent of the botulinum toxin group reported mild or no pain, compared to 26 percent of the placebo group. The greatest positive effects were noted in between four to six weeks following the injections. Several of the secondary outcome measures were also favorable for the botulinum toxin group, including the change from baseline in pain intensity and the number of days without pain. There were no significant differences between the two groups for duration of daily pain, duration of sleep, and the number of TrPs. However, the mean pain intensity scores for all TrPs were significantly lower in the botulinum group. The most common adverse event was temporary muscle soreness, which occurred in both groups, but more in the botulinum toxin group, which was attributed to the drug itself.

Comments

This study is a welcome addition to the growing number of publications investigating the effects of botulinum toxin A on TrPs, but provided an inadequate identification of the diagnostic TrP criteria used. The authors included a comprehensive review of other botulinum toxin studies and they managed to avoid the most common methodological errors. The authors used Dysport in this study, which does not have the same bioequivalency as Botox. Ranoux et al. suggested a dose relationship of Botox and Dysport of 1:3 for cervical dystonia (1). The authors did not indicate when to use botulinum toxin in relation to other interventions such as manual trigger point therapy, or trigger point injections or dry needling. Botulinum toxin may be reserved for those cases that do not respond favorably to manual therapy and dry needling or injections with an anesthetic. The authors considered the anti-nociceptive and muscle relaxing effects of botulinum toxin and its ability to modulate both motor endplates and muscle spindles [JD].

Fernandez-de-Las-Penas C, Alonso-Blanco C, Quadrado ML, Gerwin RD, Pareja JA: Myofascial trigger points and their relationship to headache clinical parameters in chronic tension-type headache. Headache 46(8): 1264-1272, 2006.

Summary

Twenty-five volunteers with tension-type headache at least 15 days per month and 25 headache-free controls were examined by blinded assessors for myofascial trigger points [TrPs]. Forward head posture was determined by photography. The upper trapezius, sternocleidomastoid, and temporalis muscles were examined bilaterally. A TrP was identified by tenderness in a hyperirritable spot within a palpable taut band, a local twitch response elicited by snapping palpation, and elicited referred pain with palpation. The TrP was considered active if the referred pain was recognized as familiar headache and as latent if not. A headache diary was kept by each patient for four weeks.

Subjects with chronic tension-type headache [CTTH] had an average of 3.9 active TrPs per subject but the control subjects had none [$P < 0.001$], as would be expected. On the other hand, CTTH subjects had a total of 53 latent TrPs, whereas the controls had only 35, which was not a statistically significant difference, but is clinically important. Eighty percent of the CTTH subjects had an active TrP in the right temporalis muscle, 76 percent in the right upper trapezius, and 64 percent in the left sternocleidomastoid muscle. Significantly increased headache characteristics compared to controls was found for headache intensity and duration with TrPs in the right upper trapezius and left sternocleidomastoid muscles. For headache intensity, the left temporalis muscle had more TrPs than controls, and for headache duration, the right temporalis muscles had statistically significantly more TrPs than controls.

Comments

This blinded controlled study helps greatly to scientifically substantiate the clinical experience that CTTH is usually caused by active TrPs in a number of head and neck muscles. It emphasizes the importance of the clinician examining for head and neck TrPs in every patient seen for acute or chronic tension-type headache.

The greater number of latent TrPs in CTTH subjects suggests that those individuals were more prone to have TrPs than control subjects, lending credence to the clinical impression that latent TrPs are more prone to become active TrPs than for active TrPs to develop de novo.

This paper presents quality prevalence data that active trigger points are associated with CTTH: temporalis > upper trapezius > sternocleidomastoid muscles. This finding is enhanced by the control data and provides guidance as to which muscles are the most important to examine first.

This study establishes the strong association between active TrPs and CTTH, but does not establish them as the cause of the headache. Fortunately, subsequent treatment papers are planned to serve that purpose [DGS].

Ofluoglu D, Gunduz OH, Kul-Panza E, Guven Z: Hypermobility in women with fibromyalgia syndrome. Clin Rheumatol 25(3): 291-293, 2006.

Summary

This controlled and apparently unblinded clinical study on fibromyalgia syndrome [FMS] by physiatrists in Turkey serendipitously includes myofascial trigger point [TrP] data. They assessed 93 women with FMS and 58 healthy women without FMS using the Beighton score that increases with more extensive hypermobility.

The FMS patients had much higher Beighton scores than normal controls [$P < 0.0001$] that decreased with age. The patients became stiffer. Also with increasing number of TrPs, the Beighton scores decreased. The patients' stiffness increased with increasing numbers of TrPs, which may also have been a function of age. The authors did not mention the diagnostic criteria used to identify TrPs, or what muscles were examined for them.

Comments

Since study of TrPs was not a stated purpose of this study, it is understandable that the information on them is sketchy, and therefore, the results need to be considered only indicative of the findings. One would expect increasing stiffness and less flexibility with increasing numbers of TrPs. Unambiguous, reliable data have not been reported as to the prevalence of TrPs with increasing age for any age group so the effect of that variable is unknown.

The older patients were stiffer and had more TrPs. I know of no other paper published that provides this much insight into the relationship between the extremely common and incapacitating phenomenon of increased stiffness with increasing age and TrPs. It is hoped that future papers will explore this relationship with a blinded and another controlled study that includes a full description of methodology. That this stiffness apparently relates strongly to TrPs is suggested by the fact that a common and effective strategy for minimizing the stiffness of advancing age is daily gentle rhythmic stretching exercises that inhibit development and activation

of TrPs. Specific research addressing this issue could be of great benefit. Changes in the thixotropic properties of the muscles with age may also be an important factor. This factor has yet to be investigated at all [DGS].

Chung JW, Ohrbach R, McCall, Jr., WD: Characteristics of electrical activity in trapezius muscles with myofascial pain. Clin Neurophysiol: 117(11): 2459-2466, 2006.

Summary

This paper aimed to examine muscle action potentials from tender areas in the trapezius muscle based on the "integrated hypothesis." The authors outlined that several previous studies appeared to provide inconsistent data about the electrical activity recorded at tender areas due to methodological differences and omissions. The study was performed on seven subjects between 22 and 38 years old with myofascial pain in the trapezius muscles. The tender area was identified by palpating the muscle for a nodule and a taut band as described in the *Trigger Point Manual* (2). Pressure-pain thresholds were measured with an algometer. The electromyography [EMG] was performed with insulated monopolar electrodes. One electrode was inserted into the skin directly overlying the tender area. The second electrode was placed into the skin overlying a non-tender area. The needles were slowly advanced in 1-3 mm increments until the maximum insertion depth of 37 mm was reached. The EMG was observed continuously. Whenever electrical activity was observed at a certain insertion depth, the researchers would continue measuring the activity for four- to 30-minute periods to determine the stability of the signal. In each 1.6 second-period, the amplitude of the EMG signal was measured by calculating the root mean square value.

Every tender area was characterized by moderate to severe pain on palpation, with referred pain to the adjacent neck and head areas. Control areas did not feature any of these characteristics. The EMG activity, interpreted as motor action unit potentials, were recorded from all seven subjects. To examine the repeatability of the recordings, the researchers compared EMG recordings taken while advancing the needle

with recordings taken while withdrawing the needle. Five subjects presented with less EMG activity on withdrawal, while the remaining two subjects had no EMG activity at all. The authors speculated that this may have been due to needle penetration would have provided “some measure of therapy.” The main finding of this study was that EMG recordings were consistently measured from tender areas in the trapezius muscle at discrete depths and over prolonged periods of time. The authors mentioned several hypotheses to explain the data. While they seemed to be leaning toward the integrated trigger point [TrP] hypothesis as a likely explanation, they were reluctant to make any definitive statements, because of “often lacking direct and convincing evidence” other than acknowledging that the action potentials originated in “the tender area, which has a relatively discrete spatial organization and has temporal stability.”

Comments

As the authors concluded, this study supports previous studies of specific electrical activity recorded at TrPs, however, without ever mentioning the word “trigger point.” At every occasion, where others would have used the term “myofascial trigger point,” the authors consistently avoided this term, although they quoted the Trigger Point Manual as their source to identify myofascial tender areas. The criteria used to identify these tender areas matched the criteria for TrPs. When they referred to the integrated TrP hypothesis, they even omitted the words “trigger point.” This unusual pattern reminded this reviewer of outdated sexual education manuals, where much attention was paid to bees and birds without ever mentioning any essential information. Apparently, to these authors the term “trigger point” is a term to be avoided in public. In a previous article by the same group, also reviewed in this journal, the authors displayed the same pattern also for no obvious reasons (3,4). While the authors were a bit more accepting of the integrated TrP hypothesis in the current article, they still considered other outdated and flawed concepts, which we already rejected in the review of their previous article (4). While we encourage scientists to be skeptical, these authors display a level of

skepticism that seems out of touch with many published studies, even though they quoted many of these studies.

Nevertheless, this paper is an important contribution to the literature confirming that TrPs have a specific electrical activity consistent with endplate noise. The authors carefully planned the study to avoid some shortcomings they perceived in other studies [JD].

Itoh KY, Katsumi Y, Hirota S, Kitakoji H: Effects of trigger point acupuncture on chronic low back pain in elderly patients—a sham-controlled randomized trial. *Acupunct Med* 24(1): 5-12, 2006.

Summary

This double-blinded, cross-over, clinical trial from Japan by two orthopedic surgeons and two acupuncturists randomly assigned 26 qualified consecutive outpatients with low back pain [LBP] to the real [A] or the sham [B] group for acupuncture treatment of their myofascial trigger points [TrPs]. During the first three-week treatment phase, Group A received real treatments and group B sham acupuncture treatments. After a three-week wash-out period, the treatments were reversed for a second treatment phase. A follow-up period of three weeks followed. The TrPs were identified by a tender taut band, patient recognition of elicited pain, and a local twitch response except for the inaccessible suboccipital muscles, which were identified by tenderness and pain recognition with manual pressure or needling. For real treatment, a disposable stainless steel needle 0.2 mm × 50 mm was inserted into a TrP to a depth of 10 to 40 mm as indicated and a sparrow-pecking technique applied to elicit a local twitch response. The needle then remained in place for 10 minutes. Sham acupuncture employed the same procedure with a blunted needle that did not penetrate the skin, and a generally successful effort was made to give the subject the impression that the procedures were identical.

Group A scored significantly lower visual analog scale [VAS] readings than Group B during Phase 1: 6.5/10 to 2.7/10 compared to no change in Group B [P < 0.001]. The Roland Morris function-questionnaire-responses in

Group A improved from 8.6 to 3.3 compared to no change in Group B [$P < 0.01$]. For the second phase, Group A retained considerable benefit from previous treatment despite the three weeks of washout and were not a suitable control. However, Group B improved during treatment in VAS scores from 6.9 to 2.7 [$P < 0.01$] and reported a similar improvement in function. The lack of Group B responses to sham treatment indicates that there was very little placebo effect in this study. The authors stated that the beneficial effects of treatment were not sustained in Group A. However, their tabular data indicate that during three weeks of washout Group A's VAS scores went from 2.7/10 to a steady 4.6/10 region for the next three weeks of sham treatment. This sustained improvement is clinically significant compared to their initial VAS score of 6.5/10.

Table 1 lists how commonly TrPs were observed in which muscles in this cohort of LBP patients, which can be used to indicate the prevalence of TrPs that are associated with LBP in various muscle. This listing reported: quadratus lumborum—nine muscles; iliopsoas, gluteus medius, and gluteus maximus—seven; piriformis—five; Iliocostalis lumborum and hamstrings—three; other—four. This totals 45 muscles that had active TrPs in 26 LBP subjects. The authors concluded that trigger point acupuncture might have greater short-term effects than sham acupuncture for the treatment of LBP.

Comments

This is one of the few scientifically credible papers that specifically addresses the identification and effective treatment of TrPs in relation to LBP. The authors are to be highly congratulated on such a well-designed, much-needed study that demonstrates clearly that treatment of TrPs is a very appropriate consideration for patients with LBP. The authors avoided the common mistake of preselecting a limited number of muscles for examination and included most of the muscles that are likely to contribute to LBP. The levator ani and rectus abdominis were conspicuous for their absence, but may have been included in the "other" category, the content of which was not reported. The authors identified a total of 45 muscles with

TrPs in 26 subjects. It is clear that some patients had more than one muscle involved and that different muscles were contributing to the LBP in different patients. Their TrP treatment technique was clinically very effective and employed an TrP dry needling technique using an acupuncture needle. A paper recently reviewed in this journal demonstrated that every one of a Chinese acupuncture site known for relieving pain was found to also be the site of an TrP (5).

To this reviewer, sustained improvement from a VAS of 6.5 to 4.6 is clinically significant and deserves recognition, especially since no follow-up program of home exercises was mentioned, and is frequently so important for sustained clinical results. This is more improvement than some authors report who are delighted with statistical significance, but with less improvement clinically. It just takes more subjects or more uniform results. Another treatment paper reviewed in this issue emphasized the importance of a follow-up home program approach (6).

The authors made one statement that needs reconsideration. They indicated that there is serious doubt as to the site of origin of local TrP tenderness saying, "In particular, sensitized nociceptors at the fascia might be possible candidates for localized tenderness." The fact that Shah et al. reported significantly, sometimes very significantly, more nociceptive histochemicals in the milieu of active TrPs than at control sites clearly supports the concept that nociceptors accompanying the motor nerves supply endplates of TrPs are responsible for local TrP tenderness, as was previously clearly described by the Integrated Hypothesis (7-9). Shah et al. reported histochemical factors in TrPs that enhance nociception including: pH, substance P, calcitonin gene-related peptide, bradykinin, serotonin, norepinephrine, tumor necrosis factor-alpha, interleukin [IL]-beta, IL-6, and IL-8 [8].

This paper shows that acupuncture [along with numerous others] treatments of TrPs can be effective, and that TrPs are an important factor in LBP. It reminds us that every patient seen for LBP deserves to have the numerous muscles that common contribute to LBP examined for TrPs [DGS].

REVIEWS

Müller W, Fiebich BL, Stratz T: New treatment options using 5-HT₃ receptor antagonists in rheumatic diseases. *Curr Top Med Chem* 6(18): 2035-2042, 2006.

Summary

This review of three previously published papers with 6 graphs by 2 German rheumatologists and a psychiatrist analyzes in depth the histochemistry and immune system effects of the 5-HT receptor antagonist tropisetron. The original paper reporting its pain-relieving effect on myofascial trigger points [TrPs] for a few days was reviewed in this journal in 2004 (10). Tropisetron has numerous major effects that include blocking a number of nociceptive substances identified in the myofascial trigger point TrP milieu by Shah et al. (8). It provided effective pain relief for only about half of the fibromyalgia syndrome and half of the TrP subjects.

Comments

The authors are to be complimented on this thoughtful summary of and commentary on three previously published papers. Because this drug blocks effective activity of a number of nociceptive substances present in TrPs, the investigators wondered why only half of the subjects with TrPs experienced good short term relief. They explained it for TrPs on the basis that the injection was misplaced, which could be possible since they made no mention of eliciting a twitch response to confirm accurate placement of the needle. Also this drug blocks only one of 14 different serotonin receptors. This specificity of the usual analgesic pain medications and the multitude of nociceptive substances in TrPs is probably one basic reason why the usual analgesic medications are so ineffective for TrP pain. In addition, the fact that only half of their fibromyalgia syndrome patients got little relief may be because approximately half of the muscles of fibromyalgia syndrome patients have active TrPs, making a significant contribution to their clinical pain and the partial effectiveness of this drug in these patients may have been due to treatment of their

TrPs without effect on their fibromyalgia syndrome. They also advance a number of other possible reasons. Knowledgeable interpretation of studies like this helps us to better understand the nature and appropriate treatment of TrP pain [DGS].

Resteghini P: Myofascial trigger points: pathophysiology and treatment with dry needling. *J Orthop Med* 28(2): 60-68, 2006.

Summary

This paper reviews in much detail the pathophysiology of myofascial trigger points [TrP], especially with regard to indications for dry needling. Resteghini weaves a wide variety of perspectives into a well-written essay about the current state of affairs of TrP research. Pertinent acupuncture references are combined with Gunn's notion of radiculopathy and TrPs, Simons' integrated TrP hypothesis, Baldry's superficial dry needling, and studies on sensitization and neural plasticity. Taken all together, Resteghini managed to offer a refreshing and inclusive perspective on myofascial pain and TrP dry needling. The author concluded the paper with a brief review of select dry needling studies.

Comments

With the rapidly growing number of physical therapists and other clinicians practicing the technique of dry needling, this article is one of the most comprehensive reviews to date. In the United States, nearly 20 percent of state boards of physical therapy have already concluded that TrP dry needling falls within the scope of physical therapy practice. While Resteghini provided much up-to-date information about the pathophysiology of TrPs, he only included a few studies on dry needling. Several other studies have been published that are supportive of TrP dry needling, such as DiLorenzo et al.'s paper (11) on the effectiveness of dry needling in patients with hemiplegia, Lucas et al.'s paper (12) on dry needling and stretching for the treatment of latent TrPs, or the Cochrane collaboration's meta-analysis (13), which concluded that dry needling may be a useful adjunct to other therapies for chronic low back pain. The

introduction of dry needling to the United States physical therapy community has many similarities with the introduction of manual therapy in the sixties, when academia, state boards of physical therapy, the American Physical Therapy Association, and even Dr. Janet Travell were very reluctant in endorsing manual therapy for physical therapists (14). Resteghini has provided much support for including trigger point dry needling in clinical practice [JD].

Lotaif AC, Mitrirattanakul S, Clark GT: Orofacial muscle pain: new advances in concept and therapy. Journal Calif Dent Assoc 34(8): 625-630, 2006.

Summary

This brief eclectic review by dentists summarizes myogenic pains of many well-known origins that are characteristic of the orofacial region. Myofascial trigger points [TrPs] were included, but with a number of misleading statements with regard to its nature and with a lack of awareness of recent advances in this field. The authors conclude that appropriate treatment depends on understanding the etiology and mechanism underlying the pain, with which I wholeheartedly agree.

Comments

The statement that myofascial pain is not associated with any histologically evident tissue damage overlooks four papers (15-18) that present many consistent histological changes and damage, and one eloquent histochemical study of TrPs that places a firm foundation for understanding the painfulness of TrPs. The histological studies include biopsies of dog TrPs, of experimentally induced TrPs in rats, and two biopsy studies of human TrPs. The histochemical study reported numerous strong stimulants of nociception in surprisingly significant amounts in TrPs compared to normal muscle (8). It strongly substantiates the basic concept of the integrated hypothesis and contributes greatly to our understanding of the pain characteristics of TrPs. For some unfathomable reason, the authors of this review of advances in the field made no mention of the integrated hy-

pothesis that was first published in 1999 and subsequently substantiated by three peer-reviewed electrodiagnostic papers that were summarized in conjunction with an updated presentation of the integrated hypothesis in 2004 (2,9) [DGS].

Bron C: The subacromial impingement syndrom [in Dutch: Het subacromiaal-impingementsyndroom]. Tijdschrift manuele therapie 3(3): 20-26, 2006.

Summary

Non-traumatic shoulder pain is commonly attributed to primary or secondary subacromial impingement syndrome [SAIS], which is characterized by pain in the antero-lateral aspect of the upper arm with referred pain into the elbow and radial aspect of the hand. Pain can be present at rest and increases with abduction and flexion of the arm, and the combined movement of the arm into extension, internal rotation, and adduction when bringing the hand to the low back. Patients may present with a painful arc between 60E-120E of flexion and abduction. The usual orthopedic tests, such as the Painful Arc Test and the Empty Can test, are not very reliable in making the diagnosis of SAIS. One of the differences between primary and secondary SAIS is the lack of objective radiological findings in secondary SAIS. In this article from the Netherlands, physical therapist Bron described in detail the various stages of SAIS and reviewed several etiologic aspects and contributing biomechanical considerations involving the gleno-humeral joint and rotator cuff muscles. A recent study identified a proprioceptive role of the coraco-acromial ligament, while others valued the role of the infraspinatus, teres minor and subscapularis muscles in the etiology of SAIS (19). The author quoted several relevant imaging studies indicating that as many as 35 percent of asymptomatic volunteers had a partial or full rupture tear, while in another study over 50 percent symptomatic patients and in healthy controls had at least one rupture and not necessarily in the painful shoulder! Bron concluded that imaging studies are not reliable either.

Instead, he recommended considering myofascial trigger points [TrP] as the referred pain

patterns of several shoulder muscles match the pain patterns seen in SAIS. Furthermore, limitations in range of motion present with SAIS are consistent with movement restrictions caused by TrPs in for example the deltoid and infraspinatus muscles. Bron speculated that chronic TrPs may eventually result in structural damage of the subacromial region, which would be diagnosed as a primary SAIS. He concluded the article with a succinct review of the characteristics and treatment options for TrPs.

Comments

This article once again underscores the notion that TrPs should be included in the differential diagnostic considerations and management of common orthopedic injuries. The author, who is a specialist in the physical therapy management of patients with head, neck and shoulder pain and dysfunction, made this important contribution in the Dutch manual therapy journal. Judging by the very limited number of TrP publications in the worldwide manual therapy literature, manual therapists have not been overwhelmingly impressed with, or exposed to current TrP concepts. Hopefully, this article will contribute to a better appreciation of the important role TrPs play in most, if not all, orthopedic ailments [JD].

Hong C-Z: Treatment of myofascial pain syndrome. *Curr Pain Headache Rep* 10: 345-349, 2006.

Summary

Following a brief introduction, summarizing the evidence and nature of myofascial trigger points [TrPs], Hong emphasized that treating the cause of myofascial pain therapy is the most important aspect of any clinical management strategy. The author stated that active TrPs are caused by facet joint dysfunction and that as soon as the underlying pathology has been eliminated, TrPs will become inactive. According to Hong, treatment of active TrPs is mainly indicated when the underlying etiology cannot be determined and should consist primarily of conservative, non-invasive therapy using deep pressure massage for superficial TrPs, and ultrasound, laser, acupressure, acupuncture, or

local injections for deeper TrPs. Unless pain from acute TrPs becomes intolerable, Hong does not recommend treating TrPs. For TrPs caused by complex regional pain syndrome [CRPS], Hong suggested to treat distal, satellite TrPs before proximal TrPs. The article concludes with a review of various interventions, including therapeutic exercise, thermotherapy, and dry needling, and injection therapy. Hong cautioned against using botulinum toxin because of lack of cost effectiveness.

Comments

Hong is possibly the most prolific TrP researcher and has contributed many outstanding studies and articles to the growing myofascial pain literature. Yet, in this article, he makes many statements that do not appear to be supported and seem contradictory to current thinking. For example, Hong expressed that TrPs are secondary to facet joint dysfunction, which is indeed a possible mechanism, but there are many other possible causes, such as visceral disease, eccentric exercise, muscle overload, etc. His suggestion that active TrPs do not need to be treated, unless the cause cannot be determined, is not consistent with clinical practice. Treating active TrPs has always been the main objective of trigger point therapy. Hong also stated that TrPs never disappear and are only converted from an active to a latent state. This statement contradicts common knowledge and implies that TrPs uncontrollable and irrespective of treatments or self-management. Addressing other contributing factors, such as joint dysfunction, is very important, but does not necessarily eliminate the need to treat active TrPs. To justify his statement that trauma-induced active TrPs will disappear without any direct treatment, Hong inserted two of his own references. However, both references are review articles rather than scientific studies, and they do not support his assumption. Hong basically states that there is a direct causal relationship between facet joint dysfunction and the presence of active TrPs. Although there are no studies that support any causal relationship between joint dysfunctions and TrPs, there is good clinical evidence and one paper establishing a correlation. It seems that Hong confuses

cause and correlation in his rather definitive statements.

There are several other unsupported statements throughout the article. Hong's suggestion to treat distal TrPs with CRPS seems at odds with the typical presentation of patients affected by this pathology. Most patients with CRPS have symptoms in the distal part of their limbs, which suggests that treatment of TrPs should commence in the paraspinal region in those neurological segments that are linked to the symptoms (21). His repeated recommendation to use ultrasound in the treatment of TrPs is not supported by the literature either, other than by using high intensity static ultrasound (22,23).

The last part of the article seems to contradict the guiding principles expressed earlier. Initially, Hong recommended against using invasive procedures, while later he devoted several paragraphs to invasive procedures. Hong quoted a study by Graboski et al., which in a previous review in this journal, was criticized for a poor understanding of TrPs (23,24). There are many outstanding studies on botulinum toxin that do support its use for the treatment of persistent TrPs as reviewed in a recent consensus statement (25). [JD]

Sorrell MR: The physical examination of migraine. *Curr Pain Headache Rep* 10(5): 350-354, 2006.

Summary

With this paper, Sorrell outlined the role of the cervical musculature in migraine headaches. After a brief illustrative case report, he described how the location of the headache pain should direct the physical examination to the splenius capitis, splenius cervicis, semispinalis, sternocleidomastoid, temporalis, trapezius, levator scapulae, suboccipitalis, and multifidi muscles. Myofascial trigger points [TrPs] in these muscles and their typical referred pain pattern frequently are responsible for cervicogenic headaches, irrespective of how the headaches are labeled. Pressure applied to cervical TrPs can provoke the patient's typical headache. Sorrell mentioned that usually, treatment of the responsible TrPs will reduce or eliminate the headache, and associated

symptoms of nausea, vomiting, and sound and light sensitivity, etc.

The author considered several other aspects of migraine headaches, including forward head posture, abnormal cervical spine alignment, nerve dysfunction, allodynia, and temporomandibular joint dysfunction. Sorrell recommended having patients return to their physician while experiencing a headache if the clinical examination does not reveal which muscles are contributing to the headaches. He also emphasized the importance of discussing the findings with the patient and outlining a comprehensive treatment plan that may include posture corrections, ergonomic adjustments, and physical therapy. Lastly, he argued that emergency room physicians should be familiar with TrPs and the physical examination.

Comments

Sorrell has prepared an excellent review article that calls on physicians to become familiar with TrPs and learn how to examine patients with migraine headaches. He included several current and pertinent references, which support the points he makes. We can only hope that clinicians who routinely see persons suffering from headaches will take his advice. As with many other pain problems, considering TrPs in the differential diagnosis will only improve patients' lives and functioning [JD].

CASE STUDIES

Anderson RU, Wise D, Sawyer T, Chan CA: Sexual dysfunction in men with chronic prostatitis/chronic pelvic pain syndrome: improvement after trigger point release and paradoxical relaxation training. *J Urol* 176(4 Pt 1): 1534-1538, 2006, with Editorial Comment, 1538-1539.

Summary

Of 146 men referred to the urology clinic at Stanford University Hospital with refractory chronic pelvic pain for at least one month, 92 percent reported one or more sexual dysfunctions that included ejaculatory pain, decreased libido, erectile dysfunctions, and

ejaculatory difficulties. They reported initial median pain scores of 12 [range 5 to 18]. In all patients, traditional therapy with antibiotics, anti-inflammatory drugs, and Alpha-blockers had failed. The urologist examined each patient's the prostate, external genitalia, and the external and internal pelvic muscles to identify myofascial trigger points [TrPs].

The physical therapist treated the TrPs with applied pressure weekly for four weeks and then biweekly for eight weeks. The authors concomitantly gave the patients pelvic relaxation training and supervised practice in paradoxical relaxation exercises of specific breathing patterns to quiet anxiety followed by daily home practice sessions.

At baseline, 92 percent of the 145 patients reported one or more sexual dysfunctions and over half had pain or discomfort with ejaculation in the past month. Eighty-one percent reported sexual dysfunctions in the absence of pain, which was surprisingly high for men in their mid-fifth decade of life. Results of treatment were assessed by two established questionnaires, the Global Response Assessment and the Pelvic Pain Symptom Survey. Results were graphed and described in tertiles of percentage improvement compared with severity at baseline level for each individual.

The three clinical conditions tested were pain, sexual dysfunctions, and urinary problems. At the end, response to treatment was identified by patients as markedly improved, moderately improved, slightly improved, no change, or worse. The last three were lumped into one category, as an unsatisfactory response. Half of the patients with sexual symptoms improved 50 percent or greater with a mean percentage score decrease of 77 percent to 87 percent. Ejaculatory pain decreased 83 percent and five of 43 became symptom-free.

Comments

This multi-patient prospective case report incorporated no placebo or control group (or blinding), but statistical analysis of the patients' estimates of benefit said that there was less chance than one in a thousand that this result could have occurred by chance. This does not eliminate the possibility of an enormous placebo effect. However, comparing each pa-

tient's long history of previous unsuccessful traditional therapies by equally dedicated and concerned therapists, these results are clinically very significant. The fact that these authors were addressing the causes of the patients' symptoms and not primarily just the symptoms per se very likely made the difference. These authors were the first clinicians in these patients' experiences to examine and treat pelvic region TrPs that were strongly associated with their symptoms and, based on treatment results, were causing much of them.

This way of looking at the research design would have been greatly strengthened by a table that listed for each patient in detail both their previous history of expensive diagnostic procedures instead of than a pelvic physical examination of the muscles and also the various treatments received by what professionals. If the estimated cost of each of these is added up for almost no results and compared to the cost of this effective treatment, the noteworthy dollar savings to the health care industry, to say nothing of the patients' avoidable frustration and suffering, would become more apparent. This experimental design would be a useful alternative to control groups that are impractical and inordinately difficult in a private practice setting, which is a major potential source of this kind of much needed research.

The authors are to be commended on dividing their results into tertiles and presenting the graph with numbers that shows all three categories of pretreatment symptom scores for all three conditions. All nine graphs showed that between 51 percent and 88 percent of the patients showed an improvement rate of 25 percent or greater. Between 33 percent and 50 percent off the patients, generally about 40 percent, showed an improvement of 50 percent or greater. For patients often with multiple conditions that had been refractory to other treatments for so long, these are impressive results.

Generally, to me, presentation of only the mean or median treatment response values are important, but a frustration, because more important is to also know how many subjects got excellent results and how many got poor results and the authors' explanation as to why the difference. This is where the seeds of progress can be found [DGS].

Ceneviz C, Maloney G, Mehta N: Myofascial pain may mimic trigeminal neuralgia. Cephalalgia 26: 899-901, 2006.

Summary

A 56-year-old man was referred to the dental Tufts Craniofacial Pain Center with a preliminary diagnosis of neuropathic pain suggestive of trigeminal neuralgia [TN]. For two months he had up to three episodes a week of brief attacks of left-sided sharp, aching, burning pain starting in the lower aspect of the jaw at 4/10 intensity and shooting up to his ear reaching an intensity of 10/10 which lasted from 10 to 15 seconds. The only triggering factor identified was fatigue. This pain met the criteria of the International Association for the Study of Pain for TN. A panoramic radiograph revealed no bony or dental pathology, but he had an Angle's Class I occlusion with a total of 12 missing posterior teeth bilaterally. Left-sided myofascial pain from the left lateral pterygoid muscle and temporal tendon was diagnosed based on tenderness of those structures to palpation that reproduced the patient's pain, which identified active TrPs. Fortunately, based on this diagnosis, an unnecessary brain magnetic resonance imaging was not ordered.

The patient was treated with the insertion of a flat lower orthotic appliance with bilateral posterior contacts on premolar and molar teeth to be removed only when eating. In one week, pain attacks subsided to 3.5/10 intensity of slight twinges of pain confined to the jaw. Two weeks later, and again at one month later, he was pain-free and was wearing the prosthesis only at night. The lateral pterygoid muscle and temporal tendon were no longer tender to palpation. At the three- and 10-month phone follow-ups, he remained pain free and continued to wear the prosthesis only at night.

Comments

This is an outstanding example how TrPs can mimic other well-established diagnostic categories and would be misdiagnosed, mistreated, and trigger needless expensive testing if the muscles were not examined for TrPs. According to this report, there was no specific indication that TrPs were the treatable cause of the

pain and was detected only because the authors' craniofacial pain examination included routine palpation of masticatory muscles and their attachments. Patients would benefit greatly if TrPs were routinely investigated on the initial examination for any musculoskeletal pain. The authors are to be highly congratulated [DGS].

Jankovic D, van Zundert A: The frozen shoulder syndrome. Description of a new technique and five case reports using the subscapular nerve block and subscapularis trigger point infiltration. Acta Anesthesiol Belgica 57(2): 137-143, 2006.

Summary

The anesthesiologist authors of these three case reports live in Germany and the Netherlands. The five subjects ranged in age from 38 to 68 years. Three of them experienced a traumatic onset with symptom durations of two to six weeks. The hemiplegic patient had symptoms for seven weeks and the rheumatoid arthritis patient for nine years. Four of the five cases had subscapularis myofascial trigger points [TrPs] identified by serious shoulder and arm pain, tenderness of the subscapularis muscle and of its attachment with restricted abduction and/or external rotation of the arm. The hemiplegic patient had tenderness at the humeral attachment of the subscapularis and TrPs in the supraspinatus, trapezius, and serratus muscles based on tenderness to palpation. None of the patients had responded to conventional therapies that included oral oxycodone and diclofenac, non-steroidal anti-inflammatory drugs, local anesthetic infiltration, analgesic infusion, oral buprenorphine, ibuprofen, diazepam, transcutaneous electrical nerve stimulator application, muscle relaxing drugs, low dose prednisone, and amitriptyline. Symptoms in all patients had been aggravated by physical therapy. One patient reported an initial visual analog scale [VAS] pain rating of 9/10, two reported 8/10, and two reported 7/10—all intolerable, disabling levels of pain.

In four cases the first one or two treatments combined an injection of analgesics ropivacaine 0.375 to 0.75 percent or bupivacaine 0.25 to 0.5 percent with the addition of a corticosteroid, dexamethazone 4 to 8 mg, or tri-

amcinolone acetate 20 to 40 mg. The subsequent five to 20 nerve block analgesic injections were without the steroid. The hemiplegic patient received no corticosteroid. Injection of the subscapularis muscle was accomplished by inserting a 7 cm [2.8 inch] 20-G needle beneath the scapula from the mid-vertebral border along the course of the middle fibers of that muscle toward their humeral attachment. The authors note that for them, trying to locate specific TrPs in this muscle with a needle is futile. Routinely injection was followed by pain-free physiotherapy to increase range of motion and to encourage return of muscle strength. Patients were also instructed to perform these exercises at home also, after treatments were concluded.

The 51-year-old diabetic patient with a four-month history of shoulder pain responded to the first treatment with VAS reduction from 8/10 to 4/10 in 24 hours. Six treatments without steroid in two weeks that were followed immediately by pain-free routine physical therapy and stretching exercises resolved her pain. She was still pain-free four months later.

The 68 year old lady with shoulder pain following sudden stress overload six weeks before responded to the first injection with VAS 9/10 becoming 5/10 in 24 hours, now with restful sleep. With five more steroid-free injections in the next two weeks she was pain-free and remained that way for at least eight months.

The 38-year-old man had 7/10 shoulder pain following trauma two weeks earlier. The shoulder had been totally immobilized during that two weeks. He responded to 15 treatments in five weeks and returned to work pain-free.

The 64-year-old hemiplegic man seen seven weeks post-stroke had a painful left shoulder with loss of muscle tone and sensory loss on that side, a subluxed humeral head downward and outward, and TrPs in the subscapular, supraspinatus, trapezius and serratus muscles. His VAS 8/10 pain became 2/10 after three treatments a week for five weeks without steroid and with physical therapy. With continued physical therapy for four months the patient reported 80 percent reduction in pain and improved mobility.

The 67-year-old lady with a nine-year history of rheumatoid arthritis and episodic severe bilateral shoulder joint pain and restriction responded to two or three weekly injections for up

to six weeks with resolution of her distressing acute pain attacks.

Comments

Although there have been reports of treating frozen shoulder by manipulation to full range of motion under surgical anesthesia, that method has not gained widespread acceptance. This concept of using anesthetic injections of the subscapularis muscle to permit pain free range of motion is a new approach. It emphasizes the paramount importance of performing the stretching exercises in a pain-free manner, which can be effectively achieved with other techniques that are not invasive.

Although the authors did not include examination for taut bands or specifically examine for attachment TrP tenderness the diagnostic criteria used are valuable diagnostic indicators of TrPs and the response to this total treatment regime is consistent with TrPs being the cause of the patient's symptoms. These case reports remind us that TrPs are often a major cause of musculoskeletal pain, especially in commonly seen acute muscle overload injuries.

The story of the hemiplegic patient is an example of the clinical impression that the shoulder pain that develops in many of these patients after about six weeks is largely due to TrPs and that the subluxations of the humeral head are frequently greatly aggravated by the taut band tension of the subscapularis muscle, which is the one that they treated. This relation between TrPs and shoulder pain in hemiplegia needs critical clinical research investigation. The fifth case illustrates a basic fact reported by a rheumatologist 25 years ago, that the most distressing pain symptom to a patient with rheumatoid arthritis is often caused by very treatable associated TrPs, but not with the therapy used to treat RA (26). The same is also true of osteoarthritis (27).

Although this treatment regime was clinically effective, the authors rightfully questioned the necessity for them to include corticosteroid in the initial injections and they did not include it at all for one patient. The fact that patients responded well to subsequent injections without corticosteroid enhances the doubts as to the necessity to include it initially.

Injection of steroids is not recommended for treatment of TrPs for several reasons (2).

In describing their technique, the authors seem to be more focused on nerve block effects that treating TrPs per se, and apparently depend on the anesthetic solution to inactivate any efferent nerve activity originating from TrPs. They make no mention of identifying the local twitch response, which often survives xylocaine injections, or the subject's pain response on encountering the TrP with a needle. No substance needs to be injected if the needle hits the endplates responsible for the TrP symptoms. Some clinicians find TrPs in this muscle quite response to injection and, more important, to much less invasive manual therapy techniques specific for TrPs such as, post-isometric relaxation (2,28). The approach described here is one way to achieve pain-free physical therapy for a few hours, but there is another non-invasive pain-free promising modality, specific microcurrent treatment that is coming into view [DGS].

Shinozaki T, Sakamoto E, Shiba S, Ichikawa F, Arakawa Y, Makihara I, Abe S, Ogawa A, Tsubat E, Imamura Y: Cervical plexus block helps in diagnosis of orofacial pain originating from cervical structures. *Tohoku J Exp Med* 210(1): 41-47, 2006.

Summary

This remarkable multiple study by dentists in Japan focuses on myofascial trigger points [TrPs] and reports on eight patients suffering from orofacial pain without relief following dental treatments. Three patients had ossification of the posterior longitudinal ligament for which one had received surgery, one a cervical disc hernia, one a whiplash injury with bulging of C4/5, one rheumatoid arthritis, and two had no history of head or neck injury. All patients described their pain as dull aching with periodic fluctuations. Routine dental x-ray evaluations were negative. Palpation of the cervical spine disclosed moderate to severe tender points at the transverse processes. Palpation of cervical muscles disclosed moderate to severe tender points in the trapezius, levator scapulae, sternocleidomastoid and the greater and smaller posterior rectus and splenius muscles. Pre-

sumably the greater and smaller posterior rectus muscles refer to the two portions of the semispinalis cervicis, one of which attaches to the first rib and the other portion to the second to fifth ribs, or to the entire semispinalis cervicis and the longissimus capitis, which, however, is much more of a lateral head flexor. The greater and smaller splenius muscles presumably refers to the splenius capitis and splenius cervicis. The TrPs were identified by palpation.

To identify the source of each patient's pain, the region surrounding each painful area was infiltrated with 0.5 ml of two percent lidocaine with epinephrine. Then, because of limitations imposed by mepivacaine toxicity, only TrPs in the superficial temporal, masseter, trapezius and sternocleidomastoid muscles were injected with one percent mepivacaine, this time without epinephrine. Each TrP was explored to elicit a twitch response and a radiating sensation. This technique needed much precision because of the minute size of the target and requires much skill. The radiating response was elicited in every TrP injected. On a later day, all patients received a blinded deep cervical nerve block that began with 1 ml of normal saline around each painful cervical vertebral transverse process followed by 1 ml of one percent mepivacaine at intervals of at least 15 min.

The orofacial-region blocks caused patient discomfort with unsatisfactory pain relief. After TrP injections, improvement in pain scores varied from 3/10 to 7/10, which was significantly better than the first set of injections [$P = 0.017$]. This improvement was acceptable but not satisfactory for four of the eight patients. Deep cervical injection of normal saline gave no pain relief, but the mepivacaine injection significantly relieved pain [$P = 0.011$] immediately after the procedure and lasted for several days. This relief was significantly better than after TrP injections [$P = 0.018$] and was considered satisfactory by most of the patients.

The deep cervical block affects a wide range of cervical muscles, but does not affect any masticatory muscles. The authors discuss fully why the symptoms and findings in these patients were not compatible with the diagnosis of cervicogenic headache and that this was not a treatment for that diagnosis. They concluded that the cause of the pain of these patients was

myogenic in origin and that the permanent resolution of the problem is quite difficult.

Comments

The authors' description of their criteria for identifying TrPs could be improved. Apparently the diagnostic criteria were muscle tenderness of unidentified degree and the identification of unspecified findings by palpation. The treatment section is very reassuring that they were dealing with TrPs because they always elicited a twitch response and/or a typical referred pain response with the needle.

The fact that the deep cervical plexus injections ended up providing more relief than just the TrP injections may be a result of experimental design. The authors did not inject posterior cervical muscles which they apparently found on examination were contributing to the patient's symptoms, as would be expected. The authors do not state how long the effects of the TrP injections lasted but, when done properly, they usually last for days if not weeks or longer, depending on perpetuating factors. Since cervical injections followed, they may have reaped the benefit of the previous TrP injections. The two papers by Fernandez et al. reviewed in this column, and especially the paper by Ceneviz et al., demonstrate how important cervical muscle TrPs are to head and neck pain. These three are only a small sample of recently published papers on this subject.

The significant orofacial pain relief from cervical spine region injections is most interesting and intriguing. We do not have a satisfactory identification of what structure[s] were the cause of the tenderness, and therefore, no satisfactory explanation of why the treatment was so effective. The enigmatic source of local spinal tenderness probably is not of central origin as hypothesized by the authors because peripheral localized analgesia was so effective. A similar, but also different, observation by Fischer and Imamura (29) may be relevant. They describe a sensitized spinal segment that is characterized by tenderness of the interspinous ligament at specific spinal levels. Examination shows dermatomal signs of radiculopathy. This finding responds to injection of the interspinous ligament with local anesthetic, which also results in

resolution of active TrPs in the muscles innervated by that segment. Their explanation of why the hypersensitivity of this structure develops and why treating it clears active TrPs is equally unsatisfactory to me. We need some thoughtful investigation of what structures are sensitized, why, and why are TrPs activated and then inactivated in sclerotomes supplied by those segments. This study is on the right track [DSG].

BRIEF REVIEWS

Rommel O, Kley RA, Dekomien G, Epplen JT, Vorgerd J, Hasenbring M: Muscle pain in myophosphorylase deficiency (McArdle's disease): the role of gender, genotype, and pain-related coping. Pain 124(3): 295-304, 2006.

In this study of pain characteristics in 24 patients with myophosphorylase deficiency [McArdle's disease], 23 subjects complained of pain. For 15 subjects the pain was intermittent and exercise-induced, while for eight subjects, the pain was permanent. A lack of the myophosphorylase results in a block of glycogen breakdown in muscle, which is expressed as exercise intolerance, myalgia, fatigue, and weakness. Among others, the authors suggested that the reported pain has similarities to myofascial pain syndrome. Even though they did not expand on this notion, it is important to consider myofascial trigger points as a contributing factor even in well-established diagnoses. It would be worthwhile to study the effects of treatment of trigger points in this patient population [JD].

Borg-Stein J, Wilkins A: Soft tissue determinants of low back pain. Curr Pain Headache Rep 10(5): 339-344, 2006.

This review article discusses several soft tissue causes of low back pain, including myofascial pain syndrome, and pain from tendinitis, bursitis, and fascial nerve entrapments. The author emphasized that non-ligamentous soft tissue disorders should be considered either isolated or in conjunction with discogenic

or facet-mediated causes of pain. While this paper does not offer any new insights, bringing soft tissue aspects of low back pain back into the foreground is always a positive endeavor [JD].

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