Myofascial Pain Syndromes–Trigger Points
David G. Simons
Jan Dommerholt

INTRODUCTION

This review of the myofascial pain literature features a remarkable collection of outstanding papers that will expand the current knowledge base of the etiology and clinical applications of myofascial trigger points [TrPs]. The paper by Shah et al. is a major milestone along the long tortuous path of understanding and acceptance of TrPs that began well over 100 years ago. These National Institutes of Health authors describe a remarkable new way to measure the histochemical milieu within TrPs and published the results in the authoritative Journal of Applied Physiology. They unequivocally identified in that milieu nine components that sensitize nociceptors, which readily accounts for a source of the local and referred pain features of TrPs. All of these components were significantly different in active compared to latent or no TrP muscles. This substantiates the validity of the purely clinical distinction between active and latent TrPs. Together, these two new findings strongly reinforce the concepts of the integrated hypothesis for the etiology of TrPs. Trigger points are no longer a syndrome of unknown etiology and of questionable validity.

Three papers described and substantiated new referred pain patterns from TrPs and were published in reputable journals, such as Pain and Headache. Each article review indicates whether it is prepared by Simons [DGS] or Dommerholt [JD].

ETIOLOGY


Summary

The novel and ingenious microanalytical system developed and employed by this National Institutes of Health group of clinicians and scientists sampled and measured the in-vitro biochemical milieu within normal muscle and at myofascial trigger points [TrPs] in near-real time at the sub-nanogram level of concentration. The system employed a microdialysis needle capable of continuously collecting microscopic samples [-0.5 µl] of physiological saline exposed to the extracellular milieu across a 105-µm thick semi-permeable membrane that permitted diffusion of solutes up to 75 kDa. Nine samples were taken from upper trapezius muscles of nine subjects in three groups: Normal [no neck pain, no TrPs]; Latent [no neck pain, latent TrP present]; Active [neck pain, active TrP present]. The needle was inserted near but not into a TrP [if present] that was identified by a tender spot in a taut band. After one minute four samples were taken every minute, then one sample every 10 seconds,
and then the needle was advanced approximately 1.5 cm until a local twitch was elicited by the needle. Then a sample was taken every 10 seconds for four minutes followed by five samples at one minute intervals. Local twitches did not occur in normal muscle. Selected analytes were analyzed by immunofluorescence capillary electrophoresis and capillary electrophrolography.

Concentrations of protons [H+], bradykinin, calcitonin gene-related peptide, substance P, tumor necrosis factor-α, Interleukin-1β, serotonin, and norepinephrine were found to be significantly higher in the Active group than either of the other two groups [P < 0.01]. The pH was significantly lower in the Active group than in the other two groups [P < 0.03]. In addition, following the twitch response, peak values of substance P and calcitonin gene-related peptide in active TrPs were significantly higher [active>latent>normal, [P < 0.02]. The two substances with the greatest increase in concentration related to the immune system, tumor necrosis factor-α and Interleukin-1β [P < 0.001].

Comment

The outstanding, authoritative, and controlled pioneering paper that explores the etiology of TrPs opens a new chapter in the history of TrPs. For the first time, this demonstration of high concentrations of effective nociceptive substances specifically and solidly identifies the source of the local and referred pain from TrPs, validating their clinical existence beyond doubt. In addition, it demonstrated clearly that the purely clinical distinction between latent and active TrPs that was based on the absence or presence of recognized pain when the TrP is compressed is associated with a highly significant objective difference in the nociceptive milieu of the two conditions. These two objective findings firmly substantiate the integrated hypothesis. It is no longer appropriate to begin an article on TrPs with the commonly used statement, “The etiology of TrPs is unknown.” At least, authors should note that there is a credible hypothesis explaining their nature that has substantial experimental verification and deserves serious consideration.

It is noteworthy that latent TrPs commonly cause motor dysfunctions in the same and associated muscles, but only active TrPs cause clinically symptomatic referred pain. Different parts of the central nervous system mediate these different kinds of effects produced by TrPs. This emphasizes the importance of identifying both active and latent TrPs when examining patients with musculoskeletal pain. The motor effects of TrPs are an investigational frontier that is almost untouched and promises a wealth of valuable clinical insights [DSG].

CLINICAL STUDIES


Summary

To determine the referred pain pattern of the pronator quadratus [PQ] muscle, these authors injected 35 arms of 35 healthy adults who had no history of neck or arm pain. They used a Teflon coated needle to verify the location of the needle in the PQ by electromyography and then injected 0.2 ml of six percent hypertonic saline. The pain patterns drawn by the patient were processed and combined by computer. Two main patterns appeared: 1. proximal and distal extension of the pain from the injection site broadly along the medial edge of the forearm in the majority of subjects [57 percent]; 2. a focus of pain over the muscle location on the volar side of the forearm that extended through two phalanges of the second and third fingers, with a less intense but similar pattern on the dorsal side of the forearm and hand. The other six subjects had various individual variations including one circumferential pattern of the wrist region. All subjects described the pain as severe [9-10/10], deep, aching, or throbbing and three complained of temporary referred numbness, but none experienced electric shock-like pain that would indicate nerve contact with the needle. These patterns include dermatomes C7-C8 and also ulnar and median nerve distributions. Other muscles referring pain from TrPs to this region are the deep and superficial flexor digitorum, flexor carpi ulnaris, and abductor
digiti minimi muscles. All of these TrPs should be considered as part of the differential diagnosis when the pain is characteristic of these dermatomes and nerves.

**Comment**

This remarkably well designed and documented study fills an important void in the myofascial trigger point [TrP] literature. The authors were understandably unaware that a recent German textbook illustrates the pain pattern of the PQ muscle observed in three patients which corresponds closely to the most intense pain reported in this study in the volar wrist region (1). This study warns of a much more extensive pain pattern. The authors were perplexed by the differences in referred pain patterns apparently because they were thinking in terms of peripheral innervation correlations. The referred pain is generated in the spinal cord and relates to the wiring and programming of spinal neurons, not peripheral nerves. However, peripheral nerve considerations are essential for making informed differential diagnoses, and the possibility of TrPs in the brachiialis, supinator, brachioradialis, subclavius, extensor digitorum communis, and scalenus minimus muscles should also be considered. Pain in this region is also commonly attributed to a carpal tunnel syndrome, the cause of which is often enigmatic when TrPs are not considered [DGS].


**Summary**

Fifteen healthy adults received 0.3 ml of six percent hypertonic saline in the abductor pollicis longus [APL] that was confirmed electromyographically with special care to avoid neurovascular trauma. The referred pain pattern from this muscle has not been published previously. Among the 15 subjects, 21 arms were injected in the central belly deep to the extensor digitorum communis muscle, close to the endplate region and location of central myofascial trigger points [TrPs] of the APL. The shaded pain drawing by each patient was transferred to a computerized pain chart system that analyzed the drawings. Nineteen of the 21 arms tested had a basic pattern of local pain at the site of injection and the most intense referred pain to the region overlying the radial styloid process and the anatomical snuffbox. Of those 19 subjects, seven experienced pain connecting these two regions. In five arms, pain was experienced in the dorsal aspect of the proximal segment of the third and fourth fingers in addition to the two isolated regions of pain. Only three cases reported only the local injection site pain and the finger pain without wrist pain.

The authors described the anatomy of this muscle in detail and noted that the most common pain patterns corresponded to patterns recognized as characteristic of the C6 dermatome, superficial radial nerve territory, and symptoms of de Quervain’s tenosynovitis. The less common finger pain overlaps with the C7 and C8 dermatomes. For this reason, patients with suspected involvement of these nerves or de Quervain’s tenosynovitis need an examination for APL TrPs.

**Comment**

This remarkably well designed and documented study fills an important void in the TrP literature. The authors included a thorough discussion of when TrP examinations are required as an important differential diagnosis. Other muscles that need to be examined for TrPs in these patients are the brachiialis, supinator, brachioradialis, subclavius, extensor digitorum communis, and scalenus minimus muscles. Pain in this region is also commonly associated with carpal tunnel syndrome, the cause of which is often enigmatic when TrPs are not considered [DSG].


**Summary**

Fernández de las Peñas et al. investigated whether myofascial trigger points [TrPs] in the
superior oblique muscle are involved in primary headaches such as chronic tension type headaches [CTTH] and episodic tension type headache [ETTH]. Fifteen subjects with CTTH, fifteen subjects with ETTH, and 15 matched controls were included in this study. The diagnosis of CTTH or ETTH was made according to the criteria of the International Headache Society. Subjects with CTTH had to have headache on at least 15 days per month; ETTH subjects less than 15 days per month. The immediate history was verified with a headache diary for four weeks prior to the trochlear examination. Subjects with ETTH were examined on headache-free days; subjects with CTTH were examined on days when their pain intensity was less than four out of 10 on a visual analog scale.

The examination of TrPs in the superior oblique muscle was based on modified criteria from Gerwin et al. and Simons et al. (2,3), and divided into four stages. A diagnosis of TrP was made when there was tenderness in the trochlear area, referred pain with prolonged pressure, and increased referred pain with either contraction or stretching of the superior oblique muscle. A TrP was classified as active if the subject recognized the referred pain as familiar.

All subjects with CTTH had pain with palpation. Eighty-six percent reported familiar referred pain with prolonged pressure outside the trochlear area, which increased with active contractions or stretching, and were classified as having active TrPs in the superior oblique muscle. The subjects with ETTH also reported pain with palpation. However, only 60 percent of subjects [nine subjects] reported referred pain that increased with active contractions but not with stretching. Only two subjects had active TrPs and seven had latent TrPs. Of the healthy controls, 67 percent had pain in the trochlear region with palpation. Only 27 percent reported referred pain with prolonged pressure, that increased slightly with contractions, but not with stretching.

The authors established that referred pain patterns from the superior oblique muscle are perceived as internal and deep pain located at the retro-orbital region, supra-orbital region, occasionally to the ipsilateral forehead, or deep within the eye. The authors acknowledged that referred pain could also originate in the supraorbital or supratrochlear nerves and suggested that both mechanisms may occur simultaneously.

Comment

A recent report by Yangüela et al., suggested that the pathogenic mechanisms of primary trochlear headaches may include injury or mechanical stress of the supraorbital and supratrochlear nerves (4). This paper is the first to identify a pain pattern produced by an intraocular muscle. It is an important contribution to the headache literature, and is published in Headache, the premier journal devoted to research and clinical applications of headaches. Trigger points are rarely considered in the headache literature. Yet, the researchers established specific referred pain patterns that are likely to be generated by TrPs in the superior oblique muscle. Clinicians are encouraged to include the superior oblique muscle in the assessment of patients with chronic and episodic tension type headaches [JD].


Summary

The objective of this study was to determine an association between cervical radiculopathy and tender spots in the neck and upper extremity. Sixteen subjects with clinical evidence of unilateral cervical [C5-C8] radiculopathy, including parascapular pain, arm pain, paresthesias, numbness, myotomal weakness, and diminished reflexes were examined with pain pressure algometry. Fourteen pairs of predetermined muscles points, mostly in the center of the muscle belly, were marked and evaluated. Each point was assessed once with the points on the non-affected side functioning as control points. The authors found statistically significant side-to-side differences for the deltoid and flexor carpi radialis muscles. Significantly greater numbers of tender spots were found on the side with radiculopathy for all subjects [75
Prior to the study, the authors examined 10 asymptomatic volunteers, who did not present with significant side-to-side differences. The authors concluded that the combination of unilateral tender spots and confirmatory signs of cervical radiculopathy should direct treatment to the cervical spine.

**Comment**

The authors decided not to differentiate between myofascial trigger points [TrPs], fibromyalgia tender points and other tender points, because of a presumed “lack of agreement in distinguishing trigger points from tender points . . .” The authors supported their choice by quoting several studies with a bias towards studies that failed to identify local pathological changes in fibromyalgia tender points. They did not consider studies by Gerwin et al. and Sciotti et al (2,5) which established interrater reliability for TrP identification, or Okifuji et al. and Russell, among others which have confirmed the reliability of the fibromyalgia tender point assessment (6,7). By limiting the algometry assessment to the center of the muscle belly, both TrPs and fibromyalgia tender points may have been missed. Furthermore, the authors did not include any recent studies that have demonstrated specific pathophysiological, histological, and electromyographic changes observed in TrPs (8-11).

The authors acknowledged several additional limitations to the study. The examiner was not blinded to the study and the study was not randomized. They mentioned that pressure algometry may not distinguish between cutaneous and muscular hypersensitivity (12). The authors did not consider that taking only one algometry measurement may also introduce significant bias. Nevertheless, the conclusion that muscle tenderness on the side of radiculopathy may reflect a “referral phenomenon” is reasonable and should be considered clinically, since radiculopathy-innervated muscles have been shown to have an increased number of TrPs [JD].


**Summary**

A novel, digital algometer was used to determine if manual pressure-release treatment of latent trapezius myofascial trigger points [TrPs] in 35 subjects reduced the pain pressure threshold [PPT] of the TrPs during treatment. The TrPs were identified in student volunteers by the presence of a tender spot in a taut band on which pressure produced referred pain in the head and/or neck. The digital algometer used a circular 0.86 cm² capacitance pressure sensor taped to the examiners thumb with a computer display of the pressure applied. The examiner was blinded as to the value displayed. Subjects were randomly allocated to the treatment [manual pressure release] or the control [sham myofascial release] group. The PPTs were significantly higher [P < 0.001] following treatment in the treatment group vs. no change in the control group. The maintenance of a constant applied pressure during a 60 second treatment, starting at a moderate, easily tolerated pain level [7/10] resulted in a progressive reduction in perceived pain and a significant increase in the pressure required to reproduce the original pain level following pressure treatment [P < 0.001]. The three blinded repeated PPT measures before and after treatment were highly repeatable [intraclass correlation coefficient = 0.952] using the novel algometer. A Fischer-type spring algometer produced results of similar reliability for TrPs in superficial muscles, but the finger algometer was clearly superior for TrPs in deeply placed muscles.

**Comment**

This randomized, controlled, and blinded study that clearly identifies the TrP-identification criteria that were used reports two literature innovations: the use of a finger palpometer on TrPs and measurement of reduction in TrP sensitivity during application of manual pressure. This is one of the few well-controlled
studies that confirm the effectiveness of manual therapy of TrPs.

One common criterion for identifying a TrP is the presence or absence of a pain response to digital pressure. A convenient digital palpometer that permits measurement of pressure applied to the tenderest spot in a taut band, which can be identified only by palpation, would facilitate standardization of the amount of pressure applied. This method should help to improve the reliability of making the diagnosis of a TrP and deserves further investigation, as suggested by the authors.

This reviewer fully agrees that more research is needed to establish the therapeutic mechanism for this treatment. An alternate mechanism considered in the discussion section attributed the response to this type of therapy to reactive hyperemia in the local area due to a counter-irritant effect of a spinal mechanism that produced reflex relaxation. To this reviewer’s knowledge there is no spinal reflex mechanism to cause the increased tension of the taut band to be relaxed. Quite the contrary, motor nerve action potentials are conspicuous for their absence, and there is impressive experimental evidence that the core of the TrP, where it feels most tense, is ischemic, not hyperemic. It is important that proposed mechanism be consistent with established experimental evidence [DSG].


Summary

This interesting study aimed to investigate the intra-rater reliability of trigger point [TrP] identification. Fifty-eight subjects with rotator cuff tendinitis of at least six weeks duration but less than 18 months were included in this study. Subjects needed to have a painful resisted movement in at least one of the ranges of abduction, external or internal rotation with or without a painful arc. Subjects with arthritis, capsulitis, rotator cuff tears, bicipital tendinitis, cervical syndrome, shoulder pain due to neurological or vascular disorders, and subjects with a history of intra-articular or subacromial steroid injections were excluded. One examiner evaluated the rotator cuff muscles of all subjects for the presence of spot tenderness, a palpable taut band, a jump sign, a local twitch response, pain recognition, and referred pain. The same examiner repeated the process three days later without access to the previous assessment. The researchers used the kappa statistic to describe the degree of agreement between assessments.

The analysis revealed perfect agreement with a kappa value of 1.0 for the taut band, spot tenderness, jump sign, and pain recognition. Kappa values for local twitch response were 1.0 for the teres minor muscle, and 0.75 for the infraspinatus muscle. Kappa values for referred pain ranged from 0.79 for the subscapularis muscle to 0.88 for the teres minor muscle. The authors suggested that the excellent intra-reliability statistics were due to methodological rigor, evidenced by good palpation techniques, standardized positioning, and perhaps even expertise of the examiner. The authors concluded that it is possible to reliably determine the presence of a taut band, spot tenderness, jump sign, and pain recognition.

Comment

Although the authors suggested that the high agreement was due to their methodology, it is not clear from this paper, whether that conclusion indeed can be drawn. The authors already acknowledged that the relatively brief interval between assessment dates may have introduced recall bias from the examiner.

The use of the kappa statistic may in itself be problematic. The kappa statistic is a chance-corrected measure of agreement, which considers the proportion of observed agreements and the proportion of agreements expected by chance. In this study, the same examiner made only two observations for each variable. A TrP characteristic was either present or absent. If the examiner found for example a local twitch response on both occasions, the reliability estimate would automatically be 100 percent. However, if a local twitch response was found on only one of the assessment dates, the rating for that characteristic would immediately drop to 50 percent. In other words, the small sample size used in this study can provide misleading
results. For example, the authors reported that referred pain in the subscapularis muscle was less reliable. Analysis of their results reveals that referred pain was absent in 55 subjects during the first assessment and in 56 subjects during the second assessment. Referred pain was present in three and two subjects during the first and second assessment, respectively, resulting in a kappa statistic of 0.79. The kappa statistic does not differentiate whether the examiner was actually skilled in determining the presence of each of the TrP characteristics in each muscle. Subscapularis TrPs were found in only three subjects or 5.2 percent, which makes this a very small sample size.

Another potential problem with the study is that to measure agreement, it is crucial to have enough variance among subjects. In a group with homogeneous characteristics, the percentage of agreements will always be high. The subjects in this study were most likely a rather homogenous group. All subjects had rotator cuff tendinitis with a painful resisted movement and were confirmed by the same examiner who also executed the study. It is thus not clear that the intra-rater reliability in this study can be established with the kappa statistic. The authors did not discuss any of these potential problems [JD].

**BASIC RESEARCH**


**Summary**

Use of statin drugs for hypercholesterolemia is associated with muscle toxicity characterized by myalgia, cramp, exercise intolerance, and fatigability. Application of simvastatin on human skeletal muscle fibers triggers a Ca\(^{2+}\) wave of intra-cellular calcium that mostly originates from sarcoplasmic reticulum release of calcium and mitochondrial membrane depolarization.

**Comment**

Although this paper does not explicitly mention trigger points [TrPs], it helps greatly to explain the strong clinical experience of successful practitioners of TrPs that statins commonly severely aggravate TrP symptoms that are relieved only by discontinuing the statin. Since increased release of Ca\(^{2+}\) is an essential component of the integrated hypothesis, this effect of statins should be no surprise. Since the muscle is an energy engine, and mitochondria are the key source of aerobic metabolism, and TrPs are hypothesized to be energy deficient, these research findings would add additional fuel to this metabolic TrP fire. If your patient has a sudden unexplained flare of TrPs or is refractory to treatment, be sure to check their statin intake [DGS].

**REVIEW ARTICLES**


**Summary**

In this brief review article, Frobb approached the management of patients with myofascial pain syndrome from a medical acupuncture perspective combined with a neuropathic pain model. The neuropathic pain model maintains that myofascial pain is the result of injury to afferent sensory nerves with subsequent spontaneous ectopic firing and dysfunctional peripheral and central sensitization. According to Frobb, this will result in the creation of an aberrant axonal loop with a short-circuiting stimulation of efferent motor neurons back to muscles. To treat local hypertonicity, physical therapies used in the treatment of myofascial pain, must stimulate nerve receptors and trigger reflexive responses in the axonal reflex pathway. In the treatment of patients with low back pain, acupuncture points may give access to the various branches of the sciatic, glutal, and sacral nerves, and the internal and external branches of the posterior rami offering an entry into treatment of those muscles involved in myofascial pain. The author promoted “neural
acupuncture” which consists of injecting lidocaine directly in specific acupuncture points to block ectopic firing and promote restoration of injured axons. Although not effective for every patient, Frobb suggested using neural acupuncture as a first-line therapy. A positive response to treatment was described as “a clinically improved status sustained for five to seven days post treatment.”

Comment

While it is conceivable that this highly speculative neuropathic model is a possible factor in the etiology of myofascial pain, this article does not mention that other treatments directed at myofascial trigger points may be equally effective. To this reviewer's knowledge there is no scientific evidence of any spinal reflex mechanism that reduces local muscle hypertonicity. Unfortunately, the article does not include any references, which is probably due to the nature of the periodical in which it was published. The author published a similar well-referenced article a few years ago (13) [JD].


Summary

Persistent myalgia following whiplash is commonly attributed to psychosocial factors, illness behavior, or poor coping skills. Evidence indicates that peripheral and central sensitizations are largely responsible and that myofascial trigger points [TrPs] often play a critical role. These patients evidence central sensitization characteristic of “wind-up” that is caused by a persistent peripheral pain source that shows evidence of regional specificity. Trigger points are just one of such a source in these patients. One study found clinically relevant TrPs in every one of 54 consecutive patients. A recent study by Shah et al. that is also reviewed in this issue demonstrates the presence of multiple potent nociceptive histochemicals in an active TrP that explain the persistent pain input from them (14). Treatment of active TrPs in whiplash patients is an important part of effective therapy (15).

Comment

One reason the cause of persistent musculoskeletal pain following whiplash is so enigmatic likely relates to the almost total absence of recognition in the published literature of the TrP contribution. This review paper eloquently emphasizes the importance of TrP in this condition. It is fervently hoped other authors concerned with whiplash patients will pick up on this lead and do prospective, controlled, blinded studies that substantiate the TrP contribution to whiplash symptoms. A novel and remarkably effective therapy, frequency specific microcurrent, has been demonstrated to be effective in similar TrP conditions such as head, neck, and face pain (16) [DGS].

CASE REPORTS


Summary

Edwards described five case studies where postural factors were likely to contribute to the persistence of myofascial trigger points [TrPs]. The first case was a chef with intermittent complaints of right-sided suprascapular and posterolateral arm pain. Active TrPs were found in the right medial scalene muscle, coracobrachialis, and triceps. Aggravating activities involved contractions of the triceps and included motorcycling, mashing potatoes, and bracing of the arms on the knees during sitting. After three sessions of superficial dry needling, muscle stretching exercises, and correction of the sitting posture, the pain resolved.

The second case involved a female with longstanding trochanteric pain. Previous interventions had failed. Pain increased with sitting and driving. Trigger points were identified in the right quadratus lumborum and gluteus minimus, which with palpation reproduced the patient’s pain. The patient was successfully treated with superficial dry needling and posture corrections. Edwards suggested that sitting with the legs crossed, tucked underneath, or sitting in a side-flexed position may have
maintained TrPs in the quadratus lumborum muscle with referred pain into the trochanteric region. The other cases included several faulty sitting and sleeping postures associated with TrPs. In all cases, superficial dry needling combined with muscle stretching exercises, and posture correction resolved the problems.

Edwards emphasized that identification of poor postural habits is important in patients with TrPs. By drawing attention to the poor postures, patients are able to break the habit.

Comments

Simons, Travell, and Simons have long recognized the importance of paying attention to mechanical perpetuating factors (3). These case studies illustrate nicely how relatively simple posture corrections can make significant differences in the outcome of physical therapy, although it is not clear from the case reports what the relative contribution was of each of the interventions [superficial needling procedures, muscle stretching exercises, and posture corrections]. In addition to evaluating common faulty sitting, standing, and sleeping postures, it is important to evaluate common and prolonged work postures (17,18). Keep in mind that posture should not just be viewed from a strict biomechanical perspective, as posture is also a reflection of the person’s personality and inner feelings (19) [JD].


Summary

Fifty-one patients with active scars and with altered resistance to palpation that elicits their pain, and several control examinations were treated by mobilizing all layers of soft tissue affected by the scar, regardless how deeply placed-down to bone. The palpation technique and findings were comparable to those used for trigger points [TrPs]. Two manual techniques for tissue stretching are illustrated and treatment included application of hot packs and stroking the scar area for relaxation. Restoring tissue motility between the scar and rib periosteum is particularly important at the angle of the rib and close to the sternocostal joints. Mobilization of scar tissue over the spinous processes and the posterior superior iliac spine is especially important following spinal surgery. Frequently the most active part of a scar is at its ends, where treatment becomes most important. In 31 of the 51 cases the scars were an essential cause of symptoms that responded well to this type of scar treatment. Symptoms were improved in 13 cases, and not in three cases.

Comments

Although this paper only refers to TrPs by analogy, it is included because scars often refer pain like TrPs, can be confusingly similar in the symptoms that they produce, and are equally important therapeutically. One must watch Prof. Lewit treating a patient to appreciate the consummate skill and depth of understanding that lies behind this paper [DGS].

BRIEF REVIEW


This paper provides a comprehensive review of an evidenced-based approach to the clinical management of persons with whiplash injuries. The authors reviewed in much detail the whiplash literature and concluded that the treatment of persons suffering from whiplash injuries should always include muscular, fascial, and spinal interventions. Spinal manipulations advocated included upper cervical, cervico-thoracic junction, thoracic, thoraco-lumbar junction, and pelvic girdle manipulations. The authors found that myofascial trigger points [TrPs] are commonly present after whiplash. They promoted TrP manual therapies, including neuromuscular technique, muscle energy techniques, myofascial release applied to the occipital region, and TrP deactivation approaches, particularly focused on the trapezius, suboccipital, scalene, and sterno-
cleidomastoid muscles. The approach advocated by these Spanish researchers have been shown to be effective and provide a solid starting point for clinical management and future studies of persons suffering from whiplash injuries (15)[JD].

REFERENCES