Interaction between Trigger Points and Joint Hypomobility: A Clinical Perspective

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Objective of the Clinical Perspective

The relationship between muscle and joint dysfunctions is well recognized by clinicians, but few studies in the scientific literature analyze this relationship. The objective of this clinical perspective is to provide the neurophysiological basis of this relationship and its clinical implication for manual/physical therapists.

What Is a Muscle Trigger Point?

Simons et al[1] defined a muscle trigger point (TrP) as a tender point within a taut band of a skeletal muscle that is painful upon compression, contraction, or stretch and usually responds with a referred pain pattern distant from the point. From a clinical viewpoint, active TrPs cause concordant patient-related pain symptoms that are local and referred, whereas latent TrPs are those that also evoke both local and referred pain but without reproducing any symptom. This clinical distinction has been supported by the fact that concentrations of protons, bradykinin, substance P, calcitonin gene-related peptide, tumor necrosis factor-α, interleukin-1β, and serotonin are significantly higher in active TrPs than in latent TrPs and when compared to no-TrPs[2]. Further, both active and latent TrPs can provoke muscle imbalance, weakness, or altered motor recruitment[3] in either the affected muscle or in functionally related muscles.

The activation of muscle TrPs may result from a variety of factors, such as muscle overuse, mechanical overload, psychological stress, or other muscle TrP. Gerwin et al[4] hypothesized that the pathogenesis of TrPs could lead from injury or overload to muscle fibers, which leads to involuntary shortening and loss of oxygen and nutrient supply resulting in increased metabolic demand on the local tissues. The seemingly most credible etiological explanation of muscle TrPs is the so-called *integrated hypothesis,* which proposes that abnormal depolarization of muscle motor endplates and sustained muscular contraction give rise to a localized "ATP energy crisis." This is associated with sensory and autonomic reflex arcs that are sustained by central sensitization[5]. Endplate noise and spikes (EMG signals from dysfunctional motor endplate regions) have been identified and associated with TrPs[6,7] supporting the theory that muscle TrPs may be dysfunctional motor endplates[8].

Ge et al[9] have provided recent evidence of sympathetic facilitation of mechanical sensitization and facilitation of

ABSTRACT: The relationship between muscle trigger points (TrPs) and joint hypomobility is frequently recognized by clinicians. Among different manual therapies aimed at inactivating muscle TrPs, ischemic compression and spinal manipulation have shown moderately strong evidence for immediate pain relief. Reduction of joint mobility appears related to local muscles innervated from the segment, which suggests that muscle and joint impairments may be indivisible and related disorders in pain patients. Two clinical studies have investigated the relationship between the presence of muscle TrPs and joint hypomobility in patients with neck pain. Both studies reported that all patients exhibited segmental hypomobility at C3-C4 zygapophyseal joint and TrPs in the upper trapezius, sternocleidomastoid, or levator scapulae muscles. There are several theories that have discussed the relationship between TrP and joint hypomobility. First, increased tension of the taut muscular bands associated with a TrP and facilitation of motor activity can maintain displacement stress on the joint. Alternatively, it may be that the abnormal sensory input from the joint hypomobility may reflexively activate TrPs. It is also conceivable that TrPs provide a nociceptive barrage to the dorsal horn neurons and facilitate joint hypomobility. There is scientific evidence showing change in muscle sensitivity in muscle TrP after spinal manipulation, which suggests that clinicians should include treatment of joint hypomobility in the management of TrPs. Nevertheless, the order in which these muscle and joint impairments should be treated is not known and requires further investigation.

KEYWORDS: Joint Hypomobility, Manual Therapy, Muscle Trigger Points

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the local and referred pain reactions in muscle TrPs. This study found that pressure pain and referred pain thresholds over infraspinatus muscle TrPs decreased during a maneuver that increased sympathetic outflow to the skeletal muscle. Burnstock[10] suggested that under pathological conditions a sympathetic-sensory interaction with muscle TrP can be established; thus, the increased sympathetic efferent discharge can facilitate TrP activity. The vasoconstrictor activity evoked by the sympathetic maneuver can reduce the blood flow[11] and lead to delayed clearance of inflammatory substances and change the local chemical milieu at the TrP.

Manual Treatment of Muscle TrPs

A first systematic review analyzing the effectiveness of manual therapies in the management of TrPs found that few studies had analyzed manual interventions for TrPs[12]. Follow-up studies have found that an ischemic compression technique is effective in reducing pain sensitivity on latent[13] and active muscle TrPs[14] as well as pain elicited by active TrPs in patients with neck pain[15]. We have recently shown that neuromuscular approaches are also effective for reducing pain sensitivity in latent TrPs[16]. Other studies reported changes in range of motion after treatment with ischemic compression[17] or a post-isometric relaxation technique[18] of latent TrPs into the masseter muscle. From this, a recent review summarized moderately strong evidence supporting the use of ischemic pressure for immediate pain relief of muscle TrPs but only limited evidence for long-term pain relief[19]. Other manual therapies are usually applied in clinical practice (e.g., TrP pressure release, spray and stretch, passive stretching, acupressure, motor training exercises, or tapping) with satisfactory results although no evidence supporting its use currently exists.

Joint Hypomobility and Spinal Manipulative Therapy

Spinal manipulation is one of the most frequently employed therapies in the treatment of neck disorders[20] and low back pain[21]. Joint hypomobility is usually defined as a temporary reduction of mobility of a zygapophyseal joint[22] and is often the focus of treatment for mobilization or manipulation. We investigated patients with sub-acute to chronic hypomobility of the mid-cervical spine, detected by the lateral gliding test, and found that joint hypomobility was associated with reduced segmental joint mobility as measured by dynamic x-ray[23]. Further, we found that the application of a rotationally directed cervical manipulation may increase segmental motion at a localized region[24]. Clinical effectiveness of spinal manipulation in both neck[25,26] and low back pain[27-29] patients is further supported by several clinical studies.

An interesting finding is that the reduction of joint motion seems to be related to local muscles innervated from the segment[30]. Further, several guidelines and reviews have indicated that manipulation will produce greater gains when performed within a treatment package that includes exercise[31-34]. Therefore, muscle and joint impairments seem to be indivisible and related disorders in chronic pain patients.

Clinical Relationship between Muscle TrPs and Joint Hypomobility

A clinical relationship between TrPs and joint impairments has been suggested by several authors[35]. Lewit[36] emphasized the importance of the treatment of TrPs and joint dysfunctions when both were present. In clinical practice, therapists commonly use a treatment approach that includes different techniques directed at both muscle and joint dysfunction. It is clinically suggested that manual treatment of an inter-vertebral joint dysfunction may provoke a therapeutic effect in TrPs located in those muscles innervated by the manipulated segment.

Two clinical studies have investigated the relationship between the presence of muscle TrPs and joint hypomobility in patients with neck pain. In the first study, a significant relationship was found between TrPs in the upper fibers of the trapezius muscle and the presence of joint hypomobility (considered when an abnormal end-feel, an increased resistance, and a decreased joint gliding were found) at the C3-C4 segment[37]. In a second study with 35 participants, all patients exhibited posterior-anterior (PA) joint hypomobility at C3-C4 zygapophyseal joint and TrPs in the upper trapezius, sternocleidomastoid, and levator scapulae muscles, although a statistically significant correlation did not exist between the identified muscle TrP and PA joint hypomobility in the mid-cervical spine[38]. Both studies confirm clinical findings related to the relationship between muscle TrPs and joint hypomobility; nevertheless, greater sample sizes and future studies are needed to confirm these results.

Several theories have discussed the relationship between TrP and joint hypomobility. Perhaps the increased tension of the taut muscular bands and facilitation of motor activity can maintain displacement stress on the joint, such that a TrP provokes the joint dysfunction. In this way, it may be that muscle shortening and increased tension caused by muscle TrPs aggravate and/or maintain abnormal joint tension in the vertebra levels crossed by these muscles. An alternative explanation would be that an abnormal sensory input from the joint hypomobility[39] reflexively activates TrPs[40]. This hypothesis has been preliminarily confirmed by Lowe[41], who found that joint dysfunctions can increase the responsiveness of motor neurons of adjacent muscles to nociceptive input from TrPs. It is also conceivable that muscle TrPs provide a nociceptive barrage to the dorsal horn neurons[2,42] and thereby facilitate segmental hypomobility. The dorsal horn neural afferences connection may explain the neurophysiological mechanism of manipulative therapy by which spinal manipulation provokes an afferent bombardment from the articular and myofascial receptors, which produces pre-synaptic inhibition of segmental pain pathways and possibly activation of the endogenous systems[43].

There is preliminary evidence investigating changes in muscle sensitivity after the spinal manipulation. Vernon et al[44] reported that cervical manipulation produced significant increases in pressure pain threshold levels over tender points surrounding a cervical dysfunc-

tion. Kuan et al[45] found that spinal manipulation at C3-C4 and C4-C5 levels was effective in reducing pain and tightness from trapezius muscle TrPs. Ruiz-Sáez et al[46] have recently discovered that a manipulation directed at the C3-C4 segment evoked changes in pressure pain sensitivity in latent TrPs in the upper trapezius muscle. These results suggest that patients may benefit from manual treatment of joint hypomobility in the management of TrPs. For instance, in patients with allodynic responses, clinicians can start muscle TrP treatment with joint interventions without increasing the muscle pain associated with direct techniques. Nevertheless, the order in which these two musculoskeletal disorders should be treated is not known, and randomized controlled trials assessing the effectiveness of the different manual therapy procedures are needed.

Conclusion

The relationship between muscle TrP and joint dysfunctions is well recognized by clinicians, and there is emerging scientific evidence to support this relationship. Thus, clinicians should include assessment and treatment of both muscle TrPs and joint hypomobility in the management of pain patients. Future studies should investigate the order in which these musculoskeletal disorders are used to achieve the best clinical result.

REFERENCES

1. Simons DG, Travell J, Simons LS. *Myofascial Pain and Dysfunction: The Trigger Point Manual*: Volume 1. 2nd ed. Baltimore, MD: Williams & Wilkins, 1999.
2. Shah JP, Phillips TM, Danoff JV, Gerber LH. An *in vitro* micro-analytical technique for measuring the local biochemical milieu of human skeletal muscle. *J Appl Physiol* 2005; 99:1977–1984.
3. Lucas KR, Polus BI, Rich PA. Latent myofascial trigger points: Their effects on muscle activation and movement efficiency. *J Bodywork Mov Ther* 2004;8:160–166.
4. Gerwin RD, Dommerholt D, Shah JP. An expansion of Simons' integrated hypothesis of trigger point formation. *Curr Pain Head Reported* 2004;8:468–475.
5. McPartland JM, Simons DG. Myofascial trigger points: Translating molecular theory into manual therapy. *J Man Manipulative Ther* 2006;14:232–239.
6. Simons DG. Do endplate noise and spikes arise from normal motor endplates? *Am J Phys Med Rehabil* 2001;80:134–140.
7. Couppé C, Midttun A, Hilden J, Jorgensen U, Oxholm P, Fuglsang-Frederiksen A. Spontaneous needle electromyographic activity in myofascial trigger points in the infraspinatus muscle: A blinded assessment. *J Musculosk Pain* 2001;9:7–16.
8. Simons DG, Hong CZ, Simons L. Endplate potentials are common to midfiber myofascial trigger points. *Am J Phys Med Rehabil* 2002;81:212–222.
9. Ge HY, Fernández-de-las-Peñas C, Arendt-Nielsen L. Sympathetic facilitation of hyperalgesia evoked from myofascial tender and trigger points in patients with unilateral shoulder pain. *Clin Neurophysiol* 2006;117: 1545–1550.
10. Burnstock G. Structural and chemical organization of the autonomic neuroeffector system. In: Bolis CL, Licinio J, & Govoni S, eds. *Handbook of the Autonomic Nervous System in Health and Disease*. New York: Marcel Dekker, 2002.
11. Macefield VG, Wallin BG. Modulation of muscle sympathetic activity during spontaneous and artificial ventilation and apnœa in humans. *J Auton Nerv Syst* 1995;53:137–147.
12. Fernández-de-las-Peñas C, Sohrbeck-Campo M, Fernández-Carnero J, Miangolarra-Page JC. Manual therapies in the myofascial trigger point treatment: A systematic review. *J Bodywork Mov Ther* 2005;9:27–34.
13. Fryer G, Hodgson L. The effect of manual pressure release on myofascial trigger points in the upper trapezius muscle. *J Bodywork Mov Ther* 2005;9:248–255.
14. Fernández-de-las-Peñas C, Alonso-Blanco C, Fernández-Carnero J, Miangolarra-Page JC. The immediate effect of ischemic compression technique and transverse friction massage on tenderness of active and latent myofascial trigger points: A pilot study. *J Bodywork Mov Ther* 2006;10:3–9.
15. Gemmell H, Miller P, Nordstrom H. Immediate effect of ischæmic compression and trigger point pressure release on neck pain and upper trapezius trigger points: A randomized controlled trial. *Clin Chiropr* 2008;11:30–36.
16. Ibáñez-García J, Alburquerque-Sendín F, Rodríguez-Blanco C, et al. Changes in masseter muscle trigger points following strain-counter/strain or neuro-muscular technique. *J Bodywork Mov Ther* 2009;13:2–10.
17. Fernández-de-las-Peñas C, Fernández-Carnero J, Galán-del-Río F, Miangolarra-Page JC. Are myofascial trigger points responsible of restricted range of motion? A clinical study (abstract). *J Musculosk Pain* 2004;12 (Suppl 9):19.
18. Rodríguez-Blanco C, Fernández-de-las-Peñas C, Hernández-Xumet JE, Peña-Algaba G, Fernández-Rabadán M, Lillo MC. Changes in active mouth opening following a single treatment of latent myofascial trigger points in the masseter muscle involving post-isometric relaxation or strain/counterstrain. *J Bodywork Mov Ther* 2006;10:197–205.
19. Vernon H, Schneider M. Chiropractic management of myofascial trigger points and myofascial pain syndrome: A systematic review of the literature. *J Manipulative Physiol Ther* 2009;32:14–24.
20. Howing JL, Gross A, Gasner D, et al. A critical appraisal of review articles on the effectiveness of conservative treatment for neck pain. *Spine* 2001;26:196–205.
21. Jette DU, Jette AM. Professional uncertainty and treatment choices by physical therapists. *Arch Phys Med Rehabil* 1997;78:1346–1351.
22. Triano JJ. Biomechanics of spinal manipulative therapy. *Spine J* 2001;1:121–130.
23. Fernández-de-las-Penas C, Downey C, Miangolarra-Page JC. Validity of the lateral gliding test as a tool for the diagnosis of inter-vertebral dysfunctions in the lower cervical spine. *J Manipulative Physiol Ther* 2005;28:610–616.
24. Fernández-de-las-Peñas C, Downey C, Miangolarra-Page JC. Immediate changes in radiographically determined lateral flexion range of motion following a single cervical HVLA manipulation in patients presenting with mechanical neck pain: A case series. *Int J Osteopath Med* 2005;8:139–145.
25. Martínez-Segura R, Fernández-de-las-Peñas C, Ruiz-Saez M, et al. Immediate effects on neck pain and active range of motion after a single cervical high-velocity low-amplitude manipulation in subjects presenting with mechanical neck pain: A randomized controlled trial. *J Manipulative Physiol Ther* 2006;29:511–517.
26. Vernon H, Humphreys K, Hagino C. Chronic mechanical neck pain in adults

treated by manual therapy: A systematic review of change scores in randomized clinical trials. *J Manipulative Physiol Ther* 2007;30: 215–227.

27. Erhard RE, Delitto A, Cibulka MT. Relative effectiveness of an extension program and a combined program of manipulation and flexion and extension exercises in patients with acute low back syndrome. *Phys Ther* 1994;74:1093–1100.
28. Koes BW, Bouter LM, van Mameren H, et al. The effectiveness of manual therapy, physiotherapy, and treatment by the general practitioner for nonspecific back and neck complaints: A randomized clinical trial. *Spine* 1992;17:28–35.
29. Triano JJ, McGregor M, Hondras MA, et al. Manipulative therapy versus education programs in chronic low back pain. *Spine* 1995;20:948–955.
30. Fryer G, Morrys T, Gibbons P. Paraspinal muscles and inter-vertebral dysfunction: Part 1. *J Manipulative Physiol Ther* 2004;27: 267–274.
31. Gross AR, Kay TM, Kennedy C, et al. Clinical practice guideline on the use of manipulation or mobilization in the treatment of adults with mechanical neck disorders. *Man Ther* 2002;7:193–205.
32. Gross AR, Hoving JL, Haines TA, et al. A Cochrane review of manipulation and mobilization for mechanical neck disorders. *Spine* 2004;29:1541–1548.
33. Gross AR, Goldsmith C, Hoving JL, et al. Conservative management of mechanical neck disorders: A systematic review. *J Rheumatol* 2007;34:1083–1102.
34. Chou R, Huffman LH, American Pain Society, American College of Physicians. Nonpharmacologic therapies for acute and chronic low back pain: A review of the evidence for American Pain Society/American College of Physicians clinical practice guideline. *Ann Intern Med* 2007;147:492–504.
35. Maitland G, Hengeveld E, Banks K, English K. *Maitland´s Vertebral Manipulation*. 6th ed. London, UK: Butterworth-Heinemann, 2000.
36. Lewit K. *Manipulative Therapy in Rehabilitation of the Locomotor System*. 2nd ed. Oxford, UK: Butterworth-Heinemann, 1991.
37. Fernández-de-las-Peñas C, Fernández J, Miangolarra JC. Musculoskeletal disorders in mechanical neck pain: Myofascial trigger points versus cervical joint dysfunctions: A clinical study. *J Musculoskeletal Pain* 2005; 13:27–35.
38. Fernández-de-las-Peñas C, Alonso-Blanco C, Alguacil-Diego IM, Miangolarra JC. Myofascial trigger points and posterior-anterior joint hypomobility in the mid-cervical spine in subjects presenting with mechanical neck pain: A pilot study. *J Man Manip Ther* 2006;14:88–94.
39. Bakkum BW, Henderson C, Hong S, Cramer G. Preliminary morphological evidence that vertebral hypomobility induces synaptic plasticity in the spinal cord. *J Manipulative Physiol Ther* 2007;30:336–342.
40. Gerwin RD. The management of myofascial pain syndrome. *J Musculoskeletal Pain* 1993;1:83–94.
41. Lowe JC. The subluxation and the trigger point: Measuring how they interact. *Chiropractic J* 1993;8:32–35.
42. Kuan TS, Hong CZ, Chen JT, Chen SM, Chien CH. The spinal cord connections of the myofascial trigger spots. *Eur J Pain* 2007;11:624–634.
43. Wall PD. The dorsal horn. In: Wall PD & Melzack R, eds. *Textbook of Pain*. 2nd ed. Edinburgh, UK: Churchill Livingstone, 2006.
44. Vernon HT, Aker P, Burns S, Viljakaanen S, Short L. Pressure pain threshold evaluation of the effect of spinal manipulation in the treatment of chronic neck pain: A pilot study. *J Manipulative Physiol Ther* 1990; 13:13–16.
45. Kuan TS, Wu CT, Chen S, Chen JT, Hong CZ. Manipulation of the cervical spine to release pain and tightness caused by myofascial trigger points [Abstract]. *Arch Phys Med Rehabil* 1997;78:1042.
46. Ruiz-Sáez M, Fernández-de-las-Peñas C, Rodríguez-Blanco C, Martínez-Segura R, García-León R. Changes in pressure pain sensitivity in latent myofascial trigger points in the upper trapezius muscle following a cervical spine manipulation in pain-free subjects. *J Manipulative Physiol Ther* 2007; 30:578–583.