

NEURAL AND JOINT AFFERENCES AS ETIOLOGIC OR PERPETUATING FACTORS OF MYOFASCIAL TRIGGER POINTS

César Fernández de las Peñas, PT, PhD, Universidad Rey Juan Carlos, cesar.fernandez@urjc.es

Introduction

Simons et al. (1999) defined a muscle Trigger Point (TrP) as a tenderness spot within a taut band of a skeletal muscle that is painful with its stimulation (compression, contraction, stretch, overload) and usually responds with a referred pain pattern distant from the spot. From a clinical viewpoint, active TrPs cause pain symptoms, and their local and referred pain evoke a usual/familiar pain for the patient (Simons et al. 1999). In patients presenting with low back pain, the referred pain elicited by active muscle TrPs reproduces at least part of their clinical pain pattern. Latent TrPs also evoke referred pain with mechanical stimulation, but this pain is not a familiar/usual for the patient. Both active and latent TrPs can provoke motor dysfunctions, e.g. muscle imbalance, muscle weakness, or altered motor recruitment (Lucas et al. 2004), in either the affected muscle or in functionally related muscles (Simons et al. 1999).

The activation of muscle TrPs may result from a variety of factors, e.g. muscle overuse, mechanical overload, psychological stress, or other active TrP. Gerwin et al. (2004) have hypothesized that the pathogenesis of TrPs could result from injured or overloaded muscle fibers, which could lead to endogenous (involuntary) shortening, loss of oxygen supply, loss of nutrient supply and increased metabolic demand on local tissues. The most credible etiological suggestion for TrP pathogenesis is the integrated hypothesis, which proposes that abnormal depolarization of motor endplates and sustained muscular contraction give rise to a localized "ATP energy crisis" associated with sensory and autonomic reflex arcs that are sustained by central sensitization (McPartland and Simons 2006).

Several studies have considered the neurophysiologic mechanisms of TrPs, especially in musculoskeletal conditions, such as neck pain. In the present paper we will discuss neural and joint influences which may be involved in the activation or perpetuation of muscle TrPs. We will also integrate these concepts with the evidence in low back pain.

Sympathetic afferences and muscle trigger points

Ge et al. (2006) provided evidence of sympathetic facilitation of mechanical sensitization and facilitation of the local and referred pain reactions in muscle TrPs. In this study, we investigated changes in both pressure pain (PPT) and referred pain (RPT) thresholds over TrPs located in the infraspinatus muscle in patients with unilateral shoulder-arm pain. A maneuver involving increased intra-thoracic pressure, which increases sympathetic outflow to the skeletal muscle, was used. The results showed that PPT and RPT levels decreased significantly during the maneuver compared with normal respiration. Further, local and referred pain intensities increased during the maneuver.

Some authors have suggested that under pathological conditions a sympathetic-sensory interaction can be established (Burnstock 2002), thus the increased sympathetic efferent discharge could facilitate both mechanical hyperalgesia and allodynia. The vasoconstrictor activity evoked by the sympathetic maneuver can reduce the blood flow (Macefield and Wallin 1995) and lead to delayed clearance of inflammatory substances and change the local chemical milieu at the muscle TrP. Further, McNulty et al. (1994) found a pain intensity reduction at TrP following administration of sympathetic blocker.

Therefore, an impairment in the sympathetic nerve system could be an etiologic (cause) or perpetuating (consequence) factor for active TrPs.

Joint afferences and muscle trigger points

A relationship between muscle TrP and joint dysfunctions is suggested by some clinicians, but only a few studies have analysed this relationship. We found a statistically significant relationship ($P=0.03$) between the presence of TrPs in the upper trapezius muscle and the presence of cervical dysfunction at C3 and C4 vertebra (Fernández-de-las-Peñas et al 2005). Although it seems obvious that TrPs and joint dysfunction could be interconnected, several theories have been proposed. Perhaps the increased tension of the taut bands and facilitation of motor activity can maintain displacement stress on the joint, such that a TrP provokes the joint dysfunction. On the other hand, an abnormal sensory input from the joint dysfunction may reflexively activate active TrPs (Gerwin 1991). We have suggested that the muscle shortening and the increased tension caused by muscle TrPs may aggravate joints and maintain abnormal joint tension in the vertebra levels crossed by these muscles. It is also conceivable that TrPs in a muscle can provide a nociceptive barrage to the dorsal horn neurons and thereby facilitate restricted segmental motion (Fernández-de-las-Peñas et al. 2006a). Lowe (1993) found that joint dysfunctions can increase the responsiveness of motor neurons of adjacent muscles to nociceptive input from distant TrPs. Further, preliminary evidence suggested that a spinal manipulation can induce changes in pressure pain sensitivity over muscle TrPs (Kuan et al. 1997). These findings provide evidence that joint dysfunctions could induce TrP activity and that TrP activity could aggravate corresponding joint dysfunction.

Neurodynamics and muscle trigger points

It also is possible that nerve tissues can contribute to the origin or perpetuation of TrPs. Edgar et al. (1994) found that decreased extensibility of the upper quadrant neural structures, as assessed by the median nerve tension test, was associated with decreased length of the upper trapezius muscle. Since muscle tissues usually respond with increased tone during nerve tension tests, an increased mechanical pain sensitivity of nerve trunks could be involved in the sensitisation of TrPs. Therefore, TrPs in the upper trapezius muscle might indirectly have a relationship with neural impairment in the upper quadrant tissues.

Deep muscle impairment and trigger points

Several studies have investigated the presence of deep muscle and motor control impairments in low back pain; however, only few studies conducted on neck pain or headache have related these disorders to TrPs. For example, a deficit in the contraction of the deep cervical flexors has been found in patients with cervicogenic headache (Jull et al. 1999), neck pain (Jull et al. 2004), whiplash (Jull et al. 2000), and chronic tension type headache (Fernández-de-las-Peñas et al. 2007). Deep flexor impairment is usually associated with higher EMG amplitudes in the superficial neck flexors, e.g. sternocleidomastoid or anterior scalene muscles (Falla et al. 2004). An increase in EMG amplitude represents higher muscle activity during functional tasks of the spine, which would provoke muscle overload of these muscles. In that way, altered recruitment of the deep flexors could contribute to the development of TrPs in those muscles overloaded by the motor control impairment, e.g. the sternocleidomastoid (Fernández-de-las-Peñas et al. 2006b, 2007).

Further, we have recently found, in a pilot study, that reduced cross sectional area of the rectus capitis posterior minor muscle was associated with active TrPs in the suboccipital muscles in patients with chronic tension type headache (Fernández-de-las-Peñas et al. 2007, in press)

Muscle trigger points and low back pain

There are no scientific studies investigating the role of active muscle TrPs in patients with low back pain. Nevertheless, mobility disorders of the sacro-iliac joint and motor control impairments have been found in persons with low back pain, which allows us to apply the same hypothesis to low back patients.

Simons et al. (1999) described several trunk muscles with referred pain patterns that are likely to contribute to low back pain, including the quadratus lumborum, multifidus muscle gluteus minimus, gluteus maximus, superficial trunk muscles, psoas, etc. Further, clinical experiences suggest that muscle involvement is relevant for the management of patients with low back pain. Recently, O'Sullivan (2007) suggested a new classification of pelvic girdle disorders in which a peripherally mediated pelvic girdle pain, including a situation with either reduced or excessive force closure, was included. Since TrPs may contribute to peripheral sensitisation (Shah et al. 2005) or altered motor recruitment (Lucas et al. 2004), it is possible that muscle TrPs could be involved in the peripherally mediated pelvic girdle pain classification suggested by O'Sullivan (2007).

Several studies have demonstrated an impaired activity of the local abdominal muscles (Hodges & Richardson 1996; 1997). In contrast, activity of global muscles is often augmented (Hodges et al. 2003). This excessive activity in the superficial muscles, which is usually considered as a compensation for poor passive or active segmental support, could lead to the origin or perpetuation of muscle TrPs.

We could include more examples integrating neural impairments from the sciatic nerve, or impairments of the lumbar or sacro-iliac joints as possible perpetuating or etiologic factors for the development of muscle TrPs. Finally, we can not forget that these concepts about muscle TrPs should be integrated into a bio-psychosocial model incorporating cognitive and behavioural processes and interventions.

Conclusions

Muscle TrPs can be incorporated into the clinical examination of patients with low back pain, although future studies should investigate their relevance in these patients. A clinical reasoning for both diagnosis and treatment of muscle TrPs should include the analysis of joint and neural afferences, including motor control impairments. A global and multi-modal approach would be the best management for these patients.

References

- Burnstock G. *Structural and chemical organization of the autonomic neuroeffector system*. In: *Handbook of the autonomic nervous system in health and disease*, edited by Bolis CL, Licinio J, Govoni S, New York: Marcel Dekker, Inc., 2002, p. 1-53.
- Edgar D, Jull G, Sutton S. *Relationship between upper trapezius muscle length and upper quadrant neural tissue extensibility*. *Aust J Physiother* 1994;40:99-103.
- Falla D, Jull G, Hodges PW. *Patients with neck pain demonstrate reduced electromyographic activity of the deep cervical flexor muscles during performance of the craniocervical flexion test*. *Spine* 2004; 29: 2108-2114
- 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 (1); 13: 27-35
- 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 Manual Manipulative Ther* 2006a; 14: 88-94
- Fernández de las Peñas C, Alonso Blanco C, Cuadrado ML, Gerwin RD, Pareja JA. *Myofascial trigger points and their relationship to headache clinical parameters in chronic tension type headache*. *Headache* 2006b; 46: 1264-1272
- Fernández de las Peñas C, Pérez de Heredia M, Molero Sánchez A, Miangolarra Page JC. *Performance of the craniocervical flexion test, forward head posture, and headache clinical parameters in patients with chronic tension type headache: A pilot study*. *J Orthop Sports Phys Ther* 2007; 37: 33-39
- Fernández-de-las-Peñas C, Cuadrado ML, Arendt-Nielsen L, Ge HY, Pareja JA. *Association of cross sectional area of the rectus capitis posterior minor muscle with active trigger points in chronic tension type headache: A pilot study*. *Am J Phys Med and Rehabil* 2007 (in press)
- 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
- Gerwin RD. *The management of myofascial pain syndrome*. *J Musculoskeletal Pain* 1993; 1:83-94.
- Gerwin RD, Dommerholt D, Shah JP. *An expansion of Simons' integrated hypothesis of trigger point formation*. *Curr Pain Head Reported* 2004; 8: 468-475
- Hodges P, Richardson C. *Inefficient muscular stabilization of the lumbar spine associated with low back pain: a motor control evaluation of transverses abdominis*. *Spine* 1996; 21: 2640-2650
- Hodges P, Richardson C. *Contraction of the abdominal muscles associated with movement of the lower limbs*. *Phys Ther* 1997; 77: 132-144
- Hodges P, Moseley G, Gabrielsson A, Gandevia S. *Experimental muscle pain changes feed-forward postural responses of the trunk muscles*. *Exp Brain Research* 2003; 151: 262-271
- Jull G, Barrett C, Magee R, Ho P. *Further clinical clarification of the muscle dysfunction in cervical headache Cephalalgia* 1999; 19: 179-185
- Jull G. *Deep cervical flexor muscle dysfunction in whiplash* *J Musculoskeletal Pain* 2000; 8:143-154
- Jull G, Kristjansson E, Dall'Alba P. *Impairment in the cervical flexors: a comparison of whiplash and insidious onset neck pain patients*. *Man Ther* 2004; 9: 89-94
- 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.
- Lowe JC. *The sub-luxation and the trigger point: measuring how they interact*. *Chiropractic J* 1993; 8: 32-35
- 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
- Macefield VG, Wallin BG. *Modulation of muscle sympathetic activity during spontaneous and artificial ventilation and apnoea in humans*. *J Auton Nerv Syst*. 1995;53:137-147.
- McNulty WH, Gevirtz RN, Hubbard DR, Berkoff GM. *Needle electromyographic evaluation of trigger point response to a psychological stressor*. *Psychophysiology*. 1994;31:313-316.
- McPartland JM, Simons DG. *Myofascial trigger points: translating molecular theory into manual therapy*. *J Man Manipul Therapy* 2006; 14: 232-239
- O'Sullivan P, Beales D. *Diagnosis and classification of pelvic girdle pain disorders Part 1: A mechanism based approach within a bio-psychosocial framework*. *Man Ther* 2007; 12: 86-97

Shah JP, Phillips TM, Danoff JV, Gerber LH An in vitro microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. *J Appl Physiol* 2005; 99: 1977-1984

Simons DG, Travell J, Simons LS. *Myofascial pain and dysfunction: The trigger point manual: Volume 1. 2nd edition*, Baltimore: Williams & Wilkins, 1999.