

COMMENTARY

SPECIFIC SEQUENTIAL MYOFASCIAL TRIGGER POINT THERAPY IN THE TREATMENT OF A PATIENT WITH MYOFASCIAL PAIN SYNDROME ASSOCIATED WITH REFLEX SYMPATHETIC DYSTROPHY

Chang-Zern Hong M.D.*

Abstract: A patient with traumatic rotator cuff tear of the left shoulder developed severe myofascial pain syndrome with reflex sympathetic dystrophy (RSD) involving the left upper extremity. He was unable to tolerate any type of manual therapy or needle treatment due to severe allodynia in the whole left upper limb. This patient presented for treatment approximately 6 months after the onset of trauma. Treatment consisting of specific myofascial trigger point (MTrP) therapy, beginning with desensitization and gentle massage on the MTrP of the first dorsal interosseous muscle, followed by treatment of MTrPs of the wrist-finger extensors and anterior deltoid muscles was commenced. Allodynia was remarkably reduced and further physical therapy with modalities was administered. After 2 weeks of daily MTrP therapy, he received local steroid injection to the left shoulder and continued MTrP therapy 2-3 times per week. Approximately 2 months after the injection the patient was almost pain free with nearly full range of motion in his left shoulder. The mechanism of MTrPs and their association with RSD is discussed in this paper.

Keywords: Manual therapy, muscle pain, Myofascial Trigger Points, Reflex Sympathetic Dystrophy.

INTRODUCTION

Myofascial trigger point (MTrP) has been defined as a highly localized painful or sensitive spot in a palpable taut band of skeletal muscle fibers and is characteristically found in myofascial pain syndrome (MPS)(1-15). An

* Department of Physical Medicine and Rehabilitation
National Cheng Kung University. Tainan, Taiwan
and
Department of Physical Medicine and Rehabilitation
University of California Irvine. Orange, California

Reprint requests to:

Department of Physical Medicine and Rehabilitation
National Cheng Kung University. Tainan, Taiwan

active MTrP is one with spontaneous pain or pain in response to movement, while a latent MTrP is a sensitive spot with pain or discomfort in response to compression only (9, 10, 16). MPS is usually caused by or associated with acute injury or chronic repetitive trauma to soft tissues, lesions involving various structures, or emotional stress. Many medical conditions (perpetuating factors), including mechanical stress, metabolic or endocrine inadequacies, chronic infections, or psychological factors, may perpetuate MPS or may aggravate its severity (4, 14, 17).

The characteristics of a MTrP (4-6, 8-10, 13-16) include:

- 1). Specific tender spot (MTrP) in a palpable taut band (also referred to as nodule).
- 2). Consistent and characteristic referred pain pattern upon compression of an MTrP.
- 3). Local twitch response elicited by snapping palpation on MTrPs in some muscles, or by needling of MTrPs in almost all cases.
- 4). Restricted range of stretch due to shortening of muscle fibres in a taut band.
- 5). Muscle weakness with no remarkable atrophy due to waxing and waning phenomena of MTrPs.
- 6). Spread of pain to other parts of body in severe cases of myofascial pain syndrome.
- 7). Associated referred autonomic phenomena including vasomotor and pilomotor responses, and hypersecretion.

In a study by Gerwin et al, it was found that “spot tenderness”, “pain recognition”, and “taut band” are the most reliable signs and the minimal criteria needed to identify an MTrP, while “referred pain” and “local twitch response” are most useful as confirmatory signs of the MTrP (18, 19). It has been concluded that it is essential to have hands-on training in order to achieve a reliable examination of MTrP (18, 19). Speculation regarding objective confirmatory findings have included confirmatory electromyographic (EMG) recording and ultrasound imaging of Latent Trigger Points (LTPs), the spontaneous electrical activity of multiple active loci, and

SPECIFIC SEQUENTIAL MYOFASCIAL TRIGGER POINT THERAPY

HONG

histological findings of contraction knots in the MTrP site (20).

Treatment techniques for MTrP (3-6, 8-10, 12-15, 17, 21-24) may include one or more of the following:

- 1). Manual therapy such as intermittent cold (with Fluorimethane or Ethyl Chloride spray, or ice massage) and stretch, which may be combined with other techniques such as post-isometric relaxation (25, 26), deep pressure soft tissue massage (by manual compression on a MTrP, combined with gentle stretch of muscle fibers in a MTrP region by moving the finger following the direction of muscle fibres), mobilisation, or manipulation.
- 2). Thermotherapy (usually combined with other therapy).
- 3). Electrotherapy by stimulation of muscle fibres around an MTrP may facilitate relaxation of a taut band and may improve local circulation.
- 4). Trigger point injections with local anaesthetic solution or dry needling into an MTrP (27-33).

In addition to the above items, the following 3 issues are essential for long-term inactivation of the MTrP:

- 1). Treatment of underlying causes.
- 2). Elimination of perpetuating factors.
- 3). Instruction in and consistent performance of a home program including self-stretching techniques, maintaining proper postures and therapeutic exercises, and other physical medicine modalities.

The clinical observation of autonomic phenomena associated with active MTrPs has been documented (12). MTrPs have been found to be a complication (or a manifestation) of reflex sympathetic dystrophy (RSD) (34). Trigger point injection could be an effective way to treat muscle pain in some RSD patients (35). In this case report, a patient with severe MTrPs and RSD was successfully treated with manual therapy for MTrP control.

CASE REPORT

History

A 25 year-old man was involved in an automobile accident and developed pain and mild swelling in the left shoulder. He was unable to move his left shoulder due to severe shoulder pain (especially at the anterior shoulder). Initial x-ray examination of the left shoulder was unremarkable. He was treated with non-steroid anti-inflammatory medicine and physical therapy (including cold, heat, and mobilisation). He had improved gradually.

Approximately 3 weeks after the initial injury, he twisted his left shoulder again when he was lifting a heavy object. At that time, the pain was much worse than the initial presentation. An MRI examination of the left shoulder

revealed evidence of rotator cuff tear at the supraspinatus tendon portion. No surgical intervention was recommended at that time. He continued to receive physical therapy. However, the pain in the left shoulder was getting worse gradually, especially during mobilisation therapy. About 1 month later, the pain spread to the left arm, forearm, wrist, and hand, progressively from the proximal to the distal portions. Swelling in the left hand was also noticed. Gradually, he also experienced episodic cold sensation with skin discoloration (pale or cyanosis) in his left hand. He became hypersensitive to touch in the whole left upper extremity and was unable to move his left upper limb due to severe pain. He was given oral steroid with little help. He was unable to take any local injection therapy due to severe pain and allodynia. He had tried acupuncture therapy but he was unable to tolerate the needle insertion. The primary physician diagnosed reflex sympathetic dystrophy and referred this patient for further management.

Physical Examination

The patient was first examined by the author approximately 6 months after the initial injury. On examination, this young man fixed his left upper limb in a position tightly closed to his body with half flexion of his elbow and wrist. Mild swelling and trophic change in the hand was noticed. He was unable to move the whole left upper extremity in all joints except slight flexion in the fingers. Allodynia was remarkable in the left shoulder-scapular area and the whole upper extremity. The skin in the left hand was very cold.

Treatment – (Specific Myofascial Trigger Point Therapy)

Since he refused to have any needle intervention (including MTrP injection), he was first treated with gentle massage on the MTrP of the left first dorsal interosseous muscle. This procedure was first approached with gradual desensitisation over the skin at the dorsum of the left hand. About 5 minutes later, he was able to tolerate gentle pressure to the MTrP of the left first dorsal interosseous muscle. Gentle massage over that MTrP was performed for about 20 minutes with gradual increase of pressure to the MTrP. Then he was able to tolerate touch (with reduced allodynia) on the left forearm and elbow. Gentle massage then was performed on the MTrP of the extensor carpi radialis muscle and the lateral epicondyle area (the common wrist-finger extensor tendons). The pressure to the MTrP was also increased gradually until he was able to tolerate the pressure up to 4-5 Kg/cm² approximately. About 20 minutes later, allodynia in the left arm and shoulder was also reduced. A similar massage procedure was then performed over the MTrP of the left anterior deltoid muscle for another 20 minutes, since this patient had mentioned that the original pain site was in the anterior shoulder.

Immediately after this treatment, he was able to move his

ACO

Volume 9 • Number 1 • March 2000

left shoulder, elbow and wrist to more than half range with no pain. The swelling of the hand was reduced remarkably and the hand was also getting warmer than before and he was then able to tolerate further treatment with thermotherapy (including hydrocollator heat packs and ultrasound), electrotherapy (muscle stimulation), and mobilisation.

Progress Examination

Examination of this patient, following specific myofascial trigger point therapy, revealed diffuse active MTrPs in the left shoulder girdle muscles and the whole left upper extremity. The most active MTrPs included upper trapezius, supraspinatus, anterior deltoid, triceps, extensor carpi radialis, extensor digitorum communis, and extensor carpi ulnaris. The range of motion of the left shoulder was restricted in all directions especially in flexion, abduction and external rotation. He had severe pain the left shoulder during active contraction of the shoulder abductors and external rotators. The muscle strength was remarkably reduced in these 2 muscle groups due to shoulder pain. There was hyperesthesia with mild allodynia in the anterior-lateral shoulder, lateral forearm, and the radial aspect of wrist and hand. Deep tendon reflexes were within normal limits. The diagnosis was myofascial pain syndrome with reflex sympathetic dystrophy involving the left upper extremity due to left rotator cuff tear.

Outcome

The patient continued to have this specific MTrP therapy once per day, except for a 2 week period where he was unable to attend, and during that time received a local injection to his left shoulder with Decadron LA 8 mg plus 1% xylocaine 5 cc. He had some increase in symptoms each time before the subsequent MTrP therapy, since the therapeutic effects only lasted for several hours after each treatment. He was also encouraged to move his left upper limb as much as possible to supplement the treatment program. He continued to receive MTrP therapy 2-3 times per week after injection. Two months after injection the patient complained of almost no pain and had regained nearly full range of motion in his left shoulder. A follow-up evaluation 6 months later revealed no evidence of recurrence.

DISCUSSION

The appropriate treatment of traumatic tendon-ligament injury includes physical therapy and anti-inflammatory therapy (either systemic or local). However, this patient did not respond to the systemic anti-inflammatory therapy and local physical therapy, and was not able to tolerate local injection. This case report demonstrated that specific MTrP therapy (especially manual therapy) could help for pain control so that the patient could tolerate a local steroid injection.

MTrP phenomena are the common results and primary pain problems of many different soft tissue lesions. The pathogenesis of MTrPs is probably related to integrative mechanisms in the spinal cord in response to sensitised nerve fibres associated with abnormal endplates (36, 37). The evidence of spinal cord mechanism in the pathogenesis of referred pain (38-42) and local twitch responses (43-45) have been well documented. As such RSD could be secondary to severe MTrP problems.

When a MTrP is very active (or very severe), satellite MTrPs may developed following the distribution of the referred pain via spinal cord mechanism (37, 46-48). Travell and Simons have illustrated the referred pain pattern for each individual muscle in the Trigger Point manual (12, 14, 15). Referred pain has been well recognized clinically, and its neurological mechanism has been documented (38-41, 49-51). There are silent (ineffective) synaptic connections in the dorsal horn (3, 40, 41, 50, 51). A sensitised or hyperirritable receptive field of a sensory neuron may cause increased sensitivity and enlargement of preexisting but "sleeping" nociceptive receptive fields in other sites of the muscle or even in other muscles corresponding to other sensory neurons by "unmasking" the silent synaptic connections (38-41). The afferent pain fibres from muscle nociceptors in the original receptive field have silent synaptic connections in the spinal cord to the sensory neurons of other receptive fields. When the original receptive field is sensitised, substance P and calcitonin gene-related peptide may be released in the site of dorsal horn neurons of the original receptive field and may diffuse into the neurons of the other receptive fields to activate the ineffective synaptic connection to become an effective connection (41). This would explain the referred pain developed in the other receptive fields when the original receptive field is stimulated. For a severe active MTrP, the irritation in its receptive field is very strong and the threshold of other spinal neurons corresponding to other receptive fields may be reduced remarkably. Finally spontaneous pain may occur in the sites of receptive fields of other sensory neurons. The expansion of receptive fields is caused by this "central sensitisation" mechanism (52). Through this mechanism, new MTrPs, or "satellite MTrPs", may develop in the referred zone of the original MTrP.

The concept of a key trigger point (29, 30, 46, 53) or a primary trigger point (14, 15) has been described in the literature. The key MTrP is the original MTrP produced immediately after an injury. However, if the original pathological lesion is not appropriately treated, MTrPs may spread out to the other sites of the body. It is also likely that these connections among the dorsal neurons may also influence the autonomic systems in the spinal cord so that RSD may develop in a severe case of MTrPs.

SPECIFIC SEQUENTIAL MYOFASCIAL TRIGGER POINT THERAPY

HONG

Treatment of a key MTrP may suppress satellite MTrPs (29, 30). When the threshold of sensory neurons is increased by treatment to the original MTrP, the threshold of other sensory neurons corresponding to the satellite MTrPs can also be increased. Similarly, effective treatment of a satellite MTrP in the referred zone of the key MTrP may also increase the pain threshold of other satellite MTrPs, or even the key MTrP, through the same spinal cord mechanism, since all these satellite MTrPs have certain connections with the key MTrP.

The pathogenesis and the therapeutic responses of severe MTrPs in our patient can be explained with the above mechanism. An original trauma to the left rotator cuff in this patient might activate latent MTrPs to become active MTrPs (37, 46) in the shoulder girdle muscles to cause the initial shoulder pain. When the original traumatic lesion was re-injured before it was completely healed, or a new rotator cuff lesion developed from a re-injury, MTrPs in the shoulder might be exacerbated, and might spread out gradually. Inappropriate or excessive stretching during mobilization therapy might also cause persistent (unhealed) lesion in the rotator cuff. Gradually, shoulder MTrPs (key MTrPs) might spread out to the whole limb. The referred zone of the key MTrP of anterior deltoid muscle covers the areas of biceps and triceps muscles. According to this specific referred pain patterns, the satellite MTrPs induced by the original anterior deltoid MTrP might have developed in the biceps or triceps muscles. Subsequently, MTrPs in the triceps might cause satellite MTrPs in the extensor carpi radialis muscles or other wrist-finger extensors (under the referred zone of triceps MTrP). Eventually, satellite MTrPs in the first dorsal interosseous muscle might develop since this muscle was under the referred zone of MTrPs of extensor indicis muscle. It was not unlikely that MTrPs in the whole upper limb might develop following the same mechanism. Finally, it might affect the autonomic system to develop RSD.

The MTrPs of the most distal muscle are usually the last to develop if the original key MTrPs were in the proximal portion of the limb, and thus might be the least sensitive (or least irritable) ones. This was the reason why the MTrP of the first dorsal interosseous muscle was selected for initial MTrP therapy. Inactivation of the distal satellite MTrPs might reduce the activity of other satellite MTrPs. Similarly, the proximal MTrPs would be inactivated gradually following subsequent MTrP therapy on other relatively more distal MTrPs.

Before the original aetiological lesion (traumatic rotator cuff inflammation) was completely eliminated, the therapeutic effects of MTrP therapy might be only temporary. After local steroid injection to the left shoulder, the inflammation of the injured rotator cuff might be healing gradually, and thus all MTrPs became less and

less active, and finally completely subside.

REFERENCES

1. Fricton JR. Myofascial pain syndrome. *Neuro Clinics of North Amer* 1989; 7(2): 413-27.
2. Fricton JR. Myofascial pain syndrome, Characteristics and Epidemiology. In: Fricton JR, Awad EA editors. *Myofascial Pain and Fibromyalgia. Advances in Pain Research and Therapy*, New York: Raven Press, 1990; 17(5): 107-27.
3. Fricton JR. Myofascial pain: clinical characteristics and diagnostic criteria. *J Musculoskel Pain* 1993; 1(3/4): 37-47.
4. Fricton JR. Myofascial Pain. In: Masi AT, editor. *Fibromyalgia and Myofascial Pain Syndromes*, Bailliere's Clinical Rheumatology; International Practice and Research. Bailliere Tindall (Saunders), Philadelphia, 1994; 8(4): 857-80.
5. Gerwin RD. Myofascial aspects of low back pain. *Neurosurg Clinics North Amer* 1991; 2(4): 761-84.
6. Gerwin RD. The clinical assessment of myofascial pain. In: Turk DC, Melzack R, editors. *Handbook of Pain Assessment*. The Guilford Press, New York, 1992: 61-70.
7. Rachlin ES. Trigger point. In: Rachlin ES editor. *Myofascial Pain and Fibromyalgia: Trigger Point Management*. St. Louis: Mosby, 1994: 145-57.
8. Rosen NB. The myofascial pain syndrome. *Phys Med Rehab Clinics North Amer* 1993; 4: 41-63.
9. Simons DG. Myofascial pain syndrome due to trigger points. In: Goodgold J, ed. *Rehabilitation Medicine*, Mosby, St. Louis, 1988: 686-723.
10. Simons DG. Muscular Pain Syndromes. In: Fricton JR, Award EA editors. *Myofascial Pain and Fibromyalgia. Advances in Pain Research and Therapy*, Raven Press, New York, 1990; 17: 1-41.
11. Simons DG, Travell JG. Myofascial pain syndromes. In Wall PD, Melzack R eds. *Textbook of Pain*. Churchill Livingstone, New York, 1989: 368-85.
12. Simons DG, Travell JG, Simons LS. *Travell & Simons's Myofascial Pain and Dysfunction: The Trigger Point Manual, Vol. 1, 2nd ed.*, Williams & Wilkins, Baltimore, 1999.
13. Sola AE, Bonica JJ. Myofascial pain syndromes. In: Bonica JJ, Loeser JD, Chapman CR, Fordyce WD, editors. *The Management of Pain*, 2nd ed. Lea & Febiger, Philadelphia, 1990: 352-67.
14. Travell JG, Simons DG. *Myofascial Pain and Dysfunction: The trigger point manual, Vol. 1*, Williams & Wilkins, Baltimore, MD., 1983.
15. Travell JG, Simons DG. *Myofascial Pain and Dysfunction: The trigger point manual, Vol. 2*, Williams & Wilkins, Baltimore, MD., 1992.
16. Rachlin ES. History and physical examination for regional myofascial pain syndrome. In: Rachlin ES editor. *Myofascial Pain and Fibromyalgia: Trigger Point Management*. St. Louis: Mosby, 1994: 159-72.
17. Gerwin RD. The management of myofascial pain syndromes. *J Musculoskel Pain* 1993; 1(3/4): 83-94.
18. Gerwin RD, Shannon S, Hong C-Z, Hubbard D, Gevirtz R. Identification of myofascial trigger points:

ACO

- interrater agreement and effect of training. *Pain* 1997; 69: 65-73.
19. Simons DG. Clinical and etiological update of myofascial pain from trigger points. *J Musculoskel Pain* 1996; 4(1/2): 93-121.
 20. Simons DG. Diagnostic criteria of myofascial pain caused by trigger points. *J Musculoskel Pain* 1999; 7(1/2): 111-20.
 21. Friction JR. Management of myofascial pain syndrome. In: Friction JR, Awad EA editors. *Myofascial Pain and Fibromyalgia. Advances in Pain Research and Therapy*, New York: Raven Press, 1990; 22(5): 325-46.
 22. Hong CZ, Simons DG. Response to standard treatment for pectoralis minor myofascial pain syndrome after whiplash. *J Musculoskel Pain* 1993; 1: 89-131.
 23. Hong CZ, Chen YC, Pon CH, Yu J. Immediate effects of various physical medicine modalities on pain threshold of the active myofascial trigger points. *J Musculoskel Pain* 1993; 1(2): 37-53.
 24. Rachlin ES. Trigger point management. In: Rachlin ES ed. *Myofascial Pain and Fibromyalgia: Trigger Point Management*. St. Louis: Mosby, 1994: 173-95.
 25. Lewit K. Postisometric relaxation in combination with other methods of muscular facilitation and inhibition. *Man Med* 1986; 2: 101-4.
 26. Lewit K, Simons DG. Myofascial pain: relief by post-isometric relaxation. *Arch Phys Med Rehabil* 1984; 65: 452-6.
 27. Chu J. Dry needling (intramuscular stimulation) in myofascial pain related to lumbar radiculopathy. *Eur J Phys Med Rehabil* 1995; 5: 106-21.
 28. Gunn CC, Milbrandt WE, Little AS, Mason KE. Dry needling of motor points for chronic low-back pain: a randomized clinical trial with long-term follow-up. *Spine* 1980; 5: 279-91.
 29. Hong CZ. Myofascial trigger point injection. *Critical Review of Physical and Rehabilitation Medicine* 1993; 5(2): 203-17.
 30. Hong CZ. Consideration and Recommendation of Myofascial trigger point injection. *J Musculoskel Pain* 1994; 2(1): 29-59.
 31. Hong CZ. Trigger point injection: Dry needling VS Lidocaine injection. *Amer J Phys Med Rehabil* 1994; 73(4):256-63.
 32. Jaeger B, Skootsky SA. Double blind, controlled study of different myofascial trigger point injection techniques. *Pain* 1987;4 (Suppl):560.
 33. Lewit K. The needle effect in relief of myofascial pain. *Pain* 1979;6:83-90.
 34. Hooshmand H. Referred pain and trigger point. In Hooshmand H, editor. *Chronic pain: Reflex sympathetic dystrophy, prevention and management*. Boca Raton, Florida: CRC Press, 1993; 8: 83-90.
 35. Hooshmand H. Management of RSD. In Hooshmand H, editor. *Chronic pain: Reflex sympathetic dystrophy, prevention and management*. Boca Raton, Florida: CRC Press, 1993; 12: 123-68.
 36. Hong CZ. Current research on myofascial trigger points - pathophysiological studies. *J Musculoskel Pain* 1999; 7(1/2): 121-9.
 37. Hong CZ, Simons DG. Pathophysiologic and electrophysiologic mechanism of myofascial trigger points. *Arch Phys Med Rehabil* 1998; 79: 863-72.
 38. Hoheisel U, Mense S, Simons DG, Yu XM. Appearance of new receptive fields in rat dorsal horn neurons following noxious stimulation of skeletal muscle: a model for referral of muscle pain? *Neuroscience Letters* 1993; 153: 9-12.
 39. Mense S. Considerations concerning the neurological basis of muscle pain. *Canadian J Physiol Pharmacol* 1991; 69(5): 610-6.
 40. Mense S. Nociception from skeletal muscle in relation to clinical muscle pain. *Pain* 1993; 54: 241-89.
 41. Mense S. Referral of muscle pain: new aspects. *Amer Pain Soc J* 1994; 3: 1-9.
 42. Mense S. Biochemical pathogenesis of myofascial pain. *J Musculoskel Pain* 1996; 4(1/2):145-62.
 43. Hong CZ, Torigoe Y. Electrophysiologic characteristics of localized twitch responses in responsive bands of rabbit skeletal muscle fibers. *J Musculoskel Pain* 1994; 2(2): 17-43.
 44. Hong CZ, Torigoe Y, Yu J. The localized twitch responses in responsive bands of rabbit skeletal muscle fibers are related to the reflexes at spinal cord level. *J Musculoskel Pain* 1995; 3(1): 15-33.
 45. Hong CZ, Yu J. Spontaneous electrical activity of rabbit trigger spot after transection of spinal cord and peripheral nerve. *J Musculoskel Pain* 1998; 6(4): 45-58.
 46. Hong CZ. Pathophysiology of Myofascial trigger point. *J Formos Med Assoc* 1996; 95(2): 93-104.
 47. Hong CZ. Algometry in evaluation of trigger points and referred pain. *J Musculoskel Pain* 1998; 6(1): 47-59.
 48. Hong CZ, Chen YN, Twehous D, Hong D. Pressure threshold for referred pain by compression on the trigger point and adjacent areas. *J Musculoskel Pain* 1996; 4(3): 61-79.
 49. Gerwin RD. Neurobiology of the Myofascial Trigger Point. In: Masi AT, ed. *Fibromyalgia and Myofascial Pain Syndromes*, Bailliere's Clinical Rheumatology; International Practice and Research. Bailliere Tindall (Saunders), Philadelphia 1994; 8(4):747-62.
 50. Simons DG. Referred phenomena of myofascial trigger points. In: Vecchiet L, Albe-Fessard D, Lindblom U, Giamberardino MA, eds. *Pain Research and Clinical Management, New Trends in Referred Pain and Hyperalgesia*. New York: Elsevier, 1993; 7: 341-57.
 51. Simons DG. Neurophysiological basis of pain caused by trigger points. *Amer Pain Soc J* 1994; 3(1): 17-9.
 52. Coderre TJ, Katz J, Vaccarino AI, Melzak R. Contribution of central neuroplasticity to pathological pain: review of clinical and experimental evidence. *Pain* 1993; 52: 259-85.
 53. Hong CZ, Simons DG. Remote inactivation of myofascial trigger points by injection of trigger point in another muscle. *Scand J Rheumatol* 1992; 94(Suppl): 24.