Journal of Bodywork and Movement Therapies (2004) **8**, 160–166

ELSEVIER

Journal of
Bodywork and Movement Therapies

www.intl.elsevierhealth.com/journals/jbmt

LATENT TRIGGER POINT RESEARCH

Latent myofascial trigger points: their effects on muscle activation and movement efficiency

Karen R. Lucas[a,*], Barbara I. Polus[a], Peter A. Rich[b]

[a]*Department of Complementary Medicine, RMIT University, P.O. Box 71, Bundoora, Victoria 3083, Australia*
[b]*School of Medical Sciences, RMIT University, Victoria 3083, Australia*

Received 10 September 2003; received in revised form 1 December 2003; accepted 2 December 2003

KEYWORDS
Muscle activation pattern;
Latent trigger point;
Kinetic chain

Abstract Kibler (Medicine and Science in Sports and Exercise 30 (1998) 79) suggests that when there is dysfunction in a proximal body segment, distal segments have to change workloads in order to preserve movement outcomes at the most distal body segment. One aspect of function is the timing of muscle activation. As the presence of pain could affect the muscle activation pattern (MAP), the effects of pain-free latent myofascial trigger points (LTrPs) in the scapular rotator muscle group were investigated. Surface electromyography was used to identify the MAP of the upper and lower trapezius, serratus anterior, infraspinatus and middle deltoid during scapular plane elevation. Repeated measures ANOVA was used to compare the control group ($n = 14$) and the LTrP group ($n = 28$). The LTrP group was then randomly assigned to either placebo intervention or true treatment to investigate the effect of removing the LTrPs. The data established that LTrPs in the scapular rotator muscles changes the MAP of this muscle group and of muscles further distal in the shoulder girdle kinetic chain. Treatment to remove LTrPs normalised the MAP.

Introduction

Many clinicians claim that the body can be viewed as a series of segments that link together to form a kinetic chain. This chain then acts to transfer force and energy in a coordinated manner in order to produce a movement outcome at the most distal segment of the chain. Kibler (1998) suggested that where there is a deficiency in a proximal segment of this kinetic chain, changed workloads may be required in more distal segments in order to preserve the same movement outcome at the most distal segment. If this is the case, patients presenting with overuse or overload injuries of the limbs may also be experiencing dysfunction in more proximal segments.

Kibler (1998) proceeds to speak specifically about the upper limb. He suggests that the scapula and the muscles that attach it to the vertebrae and ribs, comprising the trapezius, serratus anterior (SA), rhomboids, levator scapulae and pectoralis minor, serve as the segment that links the trunk to the upper limb. In order for the scapula to be positioned effectively to perform its role in transfer of force from the trunk to the upper limb, the scapular positioning muscles must be recruited in the optimal muscle activation pattern (MAP). Therefore, motor control of this muscle group

*Corresponding author. Tel.: +61-3-9925-7596; fax: +61-3-9467-2794.
E-mail address: karen.lucas@rmit.edu.au (K.R. Lucas).

1360-8592/$ - see front matter © 2003 Published by Elsevier Ltd.
doi:10.1016/j.jbmt.2003.12.002

Table 1 Demographics of experimental groups.

Group	No.	LTrPs present	Mean age (yrs)	No. of females	No. of males
Control	14	No	35.6±8.6	7	7
LTrP placebo	14	Yes	31.7±9.9	6	8
LTrP treatment	14	Yes	36.0±13.1	4	10

becomes important for successful upper limb function.

Wadsworth and Bullock-Saxton (1997) performed experiments with young elite male swimmers with unilateral chronic shoulder impingement syndrome. They found that chronic shoulder pain was associated with altered timing of muscle activation in the upward scapular rotator muscles. They did not, however, have the opportunity to establish a cause and effect relationship between these two variables. That is, did the presence of the painful joint condition cause the scapular rotator muscles to be activated differently from swimmers with no shoulder pain or does the activation sequence of the scapular rotator muscles alter first, causing a biomechanical change that leads to shoulder joint overload and eventually pain, in alignment with Kibler's theory?

The presence of a painful condition could account for any changes in MAPs during a movement test. In order to investigate Kibler's theory, it was necessary to find pain-free subjects, with some sort of deficiency of function in a proximal segment of the kinetic chain.

Latent myofascial trigger points (LTrPs) are pain-free neuromuscular lesions that are associated with muscle overload and decreased contractile efficiency (Simons et al., 1999, p. 12). There is also some evidence (Lucas et al., 2001; Simons et al., 1999, p. 12) that these lesions are prevalent in the community, rendering LTrPs as relevant lesions to investigate, as understanding their potential effects would be useful for many community members.

Accordingly, our research questions became:

1. Do LTrPs in the scapular rotator muscles alter the timing of muscle activation in this muscle group?
2. What is the effect on the timing of activation of muscles more distal in the kinetic chain of the upper limb?

Methods

Subjects

After gaining approval from the University Human Research Ethics Committee, 154 pain-free university staff and students volunteered to be examined for 'normal' shoulder girdles and subsequently, the presence of LTrPs in their scapular rotator muscles bilaterally. Subjects ($n = 112$) were excluded from the study if they did not have 160° of arm elevation, had a positive apprehension test (glenohumeral instability), upper limb tension test (neurological dysfunction), significantly increased thoracic kyphosis, reported any pain in the upper back, neck or either upper limb in the previous week or if they were under 18 or over 60 years of age. If the subject had a 'normal' shoulder girdle, they were examined for the presence of LTrPs in the scapular rotator muscles (all parts of the trapezius, rhomboids, SA, levator scapulae and pectoralis minor) on the dominant side, using the procedures explained in Lucas et al. (2001) and outlined below.

The main reason for the high number of excluded subjects was due to the fact that the two LTrP groups were filled first, meaning that any subject who had LTrPs after this point was excluded until a control group could be established. Ultimately, three groups were formed as described in Table 1.

Identification of LTrPs

The definition used to identify an LTrP in this study was 'a tender point within a palpable taut band of skeletal muscle that had a pressure-pain threshold (PPT) of less than that expected in normal muscle tissue (see, Fischer's values, 1987; and Table 2), with or without referred pain or a LTR' (local twitch response).

Pectoralis minor and SA were examined lying supine and all parts of the trapezius and rhomboids and the levator scapulae were examined lying prone.

A therapist who had been trained and was experienced in LTrP examination used the identification procedures outlined by Simons et al. (1999, pp. 116–117) briefly described as follows. The subject was lying on a table in a warm and relaxed state in an examination gown. The subject was then positioned to lengthen the muscle being examined to the point of a perceptible increase in resistance to movement. In this position, the

Table 2 Lowest PPT (kg/cm^2) at which a muscle can be considered 'normal' (Fischer 1987).

	Males (kg/cm^2)	Females (kg/cm^2)
Upper trapezius	2.9	2.0
Scapular muscles	3.6	2.7

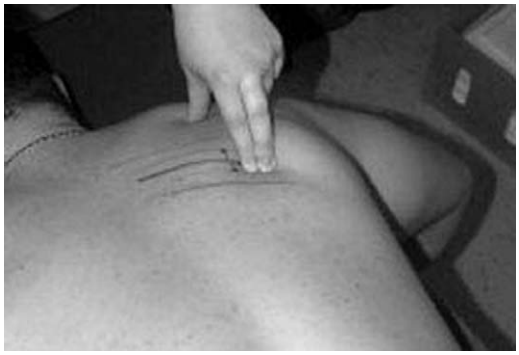

Figure 1 Palpation perpendicular to the direction of the muscle fibres to identify the taut band.

normal muscle fibres are still slack but the fibres of any taut bands are placed under additional tension, which renders them most easily distinguishable from the normal fibres. Next, cross-fibre palpation was used to identify any taut bands (Fig. 1). Fibre examination occurred via flat palpation for all muscles except the upper trapezius (UT), which was examined using pincer palpation. If a taut band was identified, the examiner then palpated along the taut band searching for a slightly enlarged point or the 'focus' of the contraction. When the examiner had identified this point, the subject was asked if the point was tender when compressed. If the subject subjectively indicated a tender point, the PPT of the tender point was measured with an algometer (Activator Methods Inc., Phoenix, Arizona, USA) (Fig. 2) using the procedure validated by Fischer (1987). If the PPT was less than that of 'normal' muscle tissue (Table 2), the tender point was defined as an LTrP and its position was marked on the skin. The subject was also asked if the pain referred elsewhere and the tender point was stimulated with snapping palpation to attempt to elicit an LTR. PPT measurements were repeated 3 times and a mean taken in order to ensure that the value was reliable. All three PPTs were taken in quick succession (within approximately 30 s) due to the fact that LTrPs can be inactivated by sustained pressure (Hou et al., 2002).

The order of muscles assessment was randomised for each subject. This examination process was

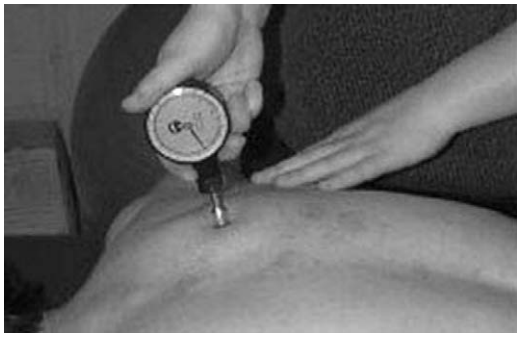

Figure 2 Using the pressure algometer to measure the PPT of a tender point.

found to have intra-examiner reliability (kappa statistics = 0.71–1 muscle dependent; intraclass correlation coefficient (ICC) for PPTs = 0.92) using a test/retest protocol with 30 min between examinations for the examiner who performed the LTrP examination.

All LTrP subjects had at least one LTrP in the scapular rotator muscle group of the dominant arm.

Time at the onset of muscle activation

Surface electromyography (SEMG) was used to measure the time of onset of muscle activity of five muscles on the dominant arm. The upper and lower trapezius (LT) and SA represented upward scapular rotators, the infraspinatus (inf) was selected to represent the rotator cuff muscle group and the middle deltoid (MD) as a prime mover during elevation of the arm in the scapular plane. The latter two muscles were selected from muscle groups that are functionally different from the scapular rotators and found further distal from them in the kinetic chain of the upper limb.

Bipolar Ag/AgCl electrodes (3M Red Dot) were used and were positioned according to Cram and Kasman (1998). The raw EMG signal from each muscle was collected using an eight-channel data recording system (Powerlab, ADInstruments, Castle Hill, NSW). The EMG signal was amplified, filtered (low pass = 500 Hz, high pass 10 Hz) and then rectified and smoothed using a root mean squared (RMS) calculation. The sampling speed was 2000 samples/s. A custom-built microswitch was placed on the subject's thigh to align with the subject's ventral forearm, immediately proximal to the wrist creases. When the forearm moved away from the body a voltage change was recorded, signifying the start of the movement. This enabled time at the onset of muscle activity to be normalised to the start of the movement.

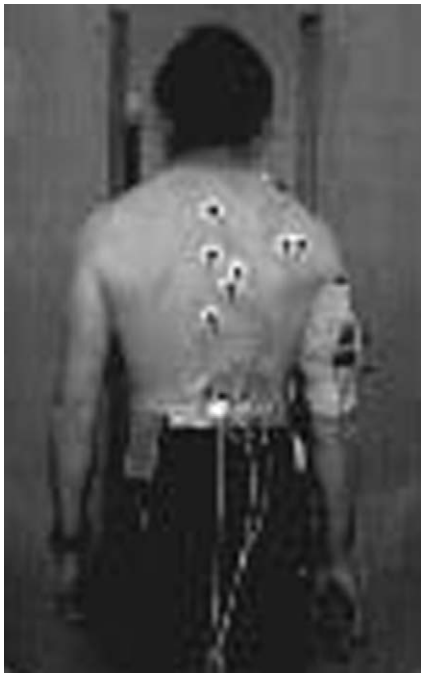

Figure 3 Starting position of the test movement.

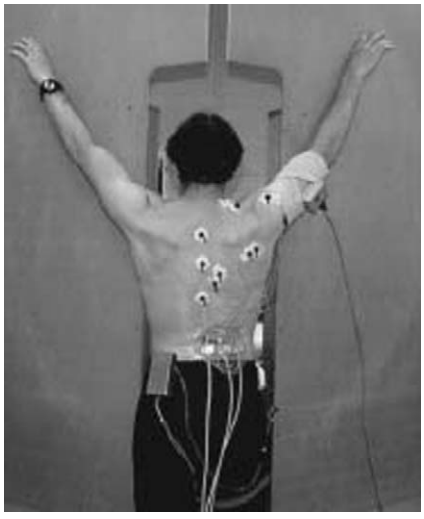

Figure 4 Performing elevation of the arm in the scapular plane. Note that the lateral aspect of the index finger remains in contact with the movement guide to prevent any external rotation of the shoulder.

Recordings were carried out according to the procedures reported by Wadsworth and Bullock-Saxton (1997). Plane of motion, standing posture, postural sway and the velocity of movement (40°/s) were controlled (see Fig 3).

Elevation of the arms in the scapular plane was performed without allowing the subject to externally rotate at the end of the range (Fig 4). This restricted subjects to 160° of movement and allowed the inf to act as a glenohumeral stabiliser rather than a prime mover in external rotation. Subjects practised the velocity of movement with the metronome prior to the EMG evaluation until they could reliably reproduce the required movement velocity. After adequate rest, subjects performed three trials of the movement with a four-second rest between trials to re-establish a baseline signal.

To identify the onset of muscle activity, the algorithm suggested by Hodges and Bui (1996) for a low-noise signal analysis (10 ms windows, 1 standard deviation (SD) above the baseline and 500 Hz low pass filter) was employed. The time of onset of muscle activity was defined as the time at the start of the first 10 ms window whose mean was more than one SD above the mean of the baseline. The accuracy of this process was confirmed by a visual inspection to ensure that the time identified as the beginning of muscle activity was not associated with ECG or other artefacts.

All subjects (control and LTrP) underwent the same SEMG protocol. The 'LTrP Treatment' subjects then received myofascial dry needling followed by passive muscle stretch to remove LTrPs (true treatment). This treatment was chosen as it is the most efficient method of removing TrPs (Cummings and White, 2001) and potentially causes less trauma to soft tissue than does other manual interventions, such as ischaemic compression. The 'LTrP Placebo' subjects received sham ultrasound, which acted as a placebo intervention where LTrPs remained after the intervention. All LTrP subjects repeated the SEMG protocol after their respective interventions to investigate the effect of removing LTrPs. Treatment interventions were completed in approximately 20 min and the repeat SEMG occurred approximately 30 min after the initial SEMG evaluation finished.

Results

Group data are shown in Fig. 5. The group mean activation times and SD of activation times are shown for each muscle. The control group displays a relatively stable, sequential MAP where the UT is always activated first, on average 115 ms prior to movement start. Immediately after the arm leaves the side of the body, the Inf (mean = 75 ms) and the MD (mean = 201 ms) are activated. The SA and LT were activated 433 ms and 776 ms after movement start, respectively, and displayed more variability in activation times than did the preceding three muscles.

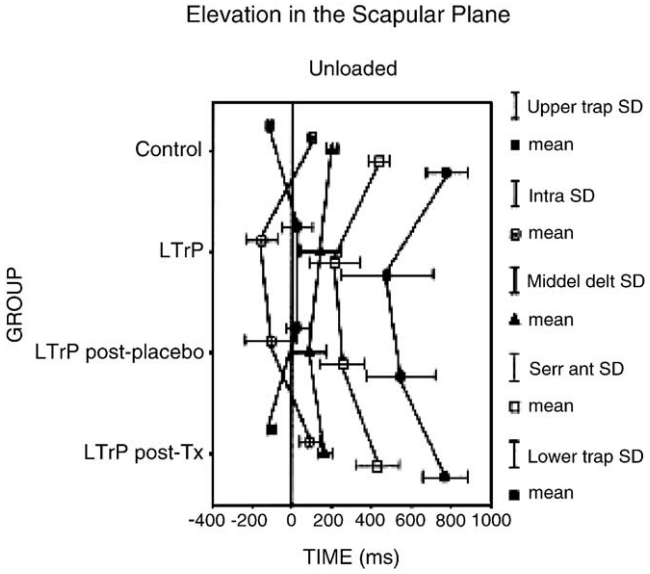

Figure 5 Comparison of the time of muscle activation between groups. P—placebo intervention (LTrPs remain); Tx—treatment intervention (LTrPs removed); UT—upper trapezius; Inf—infraspinatus; MD—middle deltoid; SA—serratus anterior; LT—lower trapezius. Time zero is the time at which the hand left the side of the body (movement start).

The only consistency in the LTrP group pre-interventions was the early activation of the Inf (−121 ms vs. 75 ms (control), significant at $p<0.05$). The remainder of the activation pattern was variable with significant differences ($p<0.05$) also found between the mean activation times for the UT (27 ms vs. −115 ms (control)). Although the MD was significantly more variable in activation time where LTrPs existed, the mean activation time was similar to the control group. While SA and LT appear to have different mean activation times from the control group, they were not statistically different. These two muscles had the highest variability of activations times in the control group, which may account for their p values (0.053 and 0.19, respectively) not reaching statistical significance. The LTrP group also displayed significantly ($p<0.05$) increased variability in activation times for all muscles.

The LTrP group post placebo showed no significant differences from the activation pattern prior to placebo intervention. In contrast, the LTrP group, in whom LTrPs were removed using myofascial dry needling and passive muscle stretch, did display a significant difference in the mean activation times from the pre-intervention condition for the UT and Inf. The SA muscle was approaching significance ($p = 0.064$). Although the LT appears to be activated later post treatment, this difference did not reach significance, possibly due to the large range (529 ms) in activation times within this muscle pre-treatment. The variability in activation times significantly ($p<0.05$) decreased post treatment for all muscles except the SA.

There was no significant difference between the control group and the LTrP treatment group (LTrPs removed) in terms of the mean activation times for any muscle, and only the SA remained more variable in activation time in the post-treatment condition.

Discussion

The control group displayed a relatively stable and sequential MAP. In this group, the UT is consistently activated before movement start. As activity was measured in the UT, but the arm had not moved from the side of the body, perhaps the UT is tensioned prior to movement start in order to create subacromial space in which the humeral head can rotate.

In the control group, the Inf was activated 75 ms after movement start on average. This seems reasonable, considering that the primary role of the Inf in this situation should be to slightly depress the humeral head and to stabilise it by compressing it into the glenoid. Interestingly, the MD was not activated until approximately 200 ms after the arm left the side of the body. This adds fuel to the argument that a muscle other than MD is responsible for initiating the movement of the hand from the side of the body. Although kinematic data were

not collected, based on the movement speed of 40°/s, it could be estimated that at 200 ms post movement start, the arm would be in the vicinity of 8° into the abduction range. According to Liu et al. (1997), the supraspinatus has its most advantageous moment arm at approximately 30° of scapular plane elevation, which would suggest that the supraspinatus was also not most suited for the initial movement of the arm. In this study, the only muscle activated prior to movement start in controls was the UT. The only means by which this muscle could have moved the arm is by lifting the lateral clavicle and then via the acromioclavicular (AC) and glenohumeral joints, superiorly translating the entire upper limb. Without kinematic data, it was not possible to confirm where the first movement occurred that may be responsible for the arm lifting away from the side of the body.

SA and LT were activated 433 and 776 ms after movement start, respectively, in the control group. These data are supported by Bagg and Forrest's finding (1988) that these scapular rotator muscles have more advantageous moment arms once the scapular has rotated its glenoid superiorly and the instantaneous centre of rotation of the scapula has migrated laterally.

A significantly different temporal sequence of muscle activation was measured when LTrPs were present in the scapular rotator muscles suggesting that LTrPs do affect the timing of the MAP in this muscle group and of muscles more distal in the upper limb chain. Vasilyeva and Lewit (1996, p. 135) suggested that when UT is abnormally shortened, it contracts earlier than deltoid during arm elevation. This implies that when UT is of normal length, it will be activated after the deltoid. In the current study, control subjects activated the UT prior to the MD while the LTrPs group activated the UT significantly later than controls, although still prior to MD. Although it might be argued that a muscle containing an LTrP will have some shortened sarcomeres, the muscle as a whole may not be significantly hyper-excited to be shortened in length. Although the current data appear to contradict the clinical opinion of Vasilyeva and Lewit with regard to motor patterns during shoulder abduction, it may be a case of comparing apples to oranges, due to the fact that specific muscle lengths were not measured in the present study. What can be supported is that when UT contains an LTrP, it is activated at approximately the same time as the arm begins to move from the side of the body, whereas when this muscle is LTrP-free, it is clearly activated before the arm leaves the side of the body. If one of the intentions of an early activation of UT is to begin elevating the acromion via the AC joint to create increased subacromial space, then later or inefficient activation of this muscle during this movement may predispose an individual to impingement of structures between the humeral head and the inferior surface of the acromion.

Where LTrPs exist, the Inf is activated before the arm leaves the body instead of immediately after movement start, as was the case in the control group. This implies that the Inf may be active for longer when LTrPs are present in the scapular rotator muscles. The relationship between LTrPs in scapular rotator muscles and the increased time period for which the Inf is active becomes interesting when considering the prevalence of rotator cuff overload and tendinitis in both Dutch (Windt et al., 1995) and Australian (Green et al., 2003) communities and the challenges this condition provides for both patients and practitioners.

There is a significant difference ($p<0.05$) in the variability of the MAP both within and between subjects when LTrPs are present in the scapular rotator muscles. This suggests that their presence decreases the consistency of the MAP in this muscle group and the Inf and MD, representing the next functional segments of the upper limb chain. The MAP of this group displayed co-contraction of muscles, a feature usually associated with muscle fatigue (Chabran et al., 2002). This indicates that where LTrPs exist, the strategy used to elevate the arm displays coping behaviours that are associated with decreased movement efficiency, mainly co-contraction and increased variability of the MAP.

As a final point of discussion, although it was found that removing LTrPs normalised the MAP, it is not known for how long this normalisation lasts or whether repeated or adjunctive interventions would be necessary for a long-term effect.

Clinical significance

MTrPs are not just contracted muscle fibres but neuromuscular lesions that form part of a neurological loop that affects and is affected by the CNS. This is evidenced by the fact that removing LTrPs normalises the MAP.

The presence of LTrPs in the scapular rotator muscles is associated with changes in motor control prior to the presence of pain. The changes described above may predispose individuals to increased risk of subacromial impingement, overuse

of the inf and decreased efficiency of movement during scapular plane elevation.

Conclusions

LTrPs in the scapular rotator muscles do alter the timing and decrease the consistency of the MAP of this muscle group and muscles more distal in the upper limb chain. These findings occurred in the absence of pain and may have implications in the areas of shoulder impingement syndrome, rotator cuff overuse and in training optimal movement efficiency of the upper limb.

References

Bagg, S.D., Forrest, W.J., 1988. A biomechanical analysis of scapular rotation during arm abduction in the scapular plane. American Journal of Physical Medicine and Rehabilitation 67, 238–245.

Chabran, E., Maton, B., Fourment, A., 2002. Effects of postural muscle fatigue on the relation between segmental posture and movement. Journal of Electromyography and Kinesiology 12, 67–79.

Cram, J.R., Kasman, G.S., 1998. Introduction to Surface Electromyography. Aspen Publishers, Maryland, USA.

Cummings, T.M., White, A.R., 2001. Needling therapies in the management if myofascial trigger point pain: a systematic review. Archives of Physical and Medicine and Rehabilitation 82, 986–992.

Fischer, A.A., 1987. Pressure algometry over normal muscles. Standard values, validity and reproducibility and pressure threshold. Pain 30, 115–126.

Green, S., Buchbinder, R., Hetrick, S., 2003. Physiotherapy interventions for shoulder pain. Cochrane Database System Reviews 2, CD004258. PMID: 12804509.

Hodges, P.W., Bui, B.H., 1996. A comparison of computer based methods for the determination of onset of muscle contraction using electromyography. Encephal Clinical Neurophysiology 101, 511–519.

Hou, C.R., Tsai, L.C., Cheng, K.F., Chung, K.C., Hong, C.Z., 2002. Immediate effects of various physical therapeutic modalities on cervical myofascial pain and trigger-point sensitivity. Archives of Physical Medicine and Rehabilitation 83, 1406–1414.

Kibler, W.B., 1998. Shoulder rehabilitation: principles and practice. Medicine and Science in Sports and Exercise 30, 79–85.

Lucas, K.R., Polus, B.I., Rich, P.A., 2001. Latent myofascial trigger points and temporal recruitment patterns of the shoulder girdle. Proceedings of the Third Conference of the International Shoulder Group, September 4–5, 2000, Newcastle-upon-Tyne, UK.

Liu, J., Hughes, R.E., Smutz, W.P., Niebur, G., An, K.N., 1997. Roles of deltoid and rotator cuff muscle in shoulder elevation. Clinical Biomechanics 12, 32–38.

Simons, D.G., Travell, J.G., Simons, L.S., 1999. The Trigger Point Manual, Vol 1, 2nd Edition. Williams and Wilkins, Baltimore, USA.

Van der Windt, D.A., Koes, B.W., de Jong, B.A., Bouter, L.M., 1995. Shoulder disorders in general practice: incidence, patient characteristics, and management. Annuals of the Rheumatic Diseases 12, 959–964.

Vasilyeva, L.F., Lewit, K., 1996. Diagnosis of muscular dysfunction by inspection. In: Liebenson, C.R. (Ed.), Rehabilitation of the Spine. Williams and Wilkins, USA.

Wadsworth, D.J.S., Bullock-Saxton, J.E., 1997. Recruitment patterns of the scapular rotator muscles in freestyle swimmers with subacromial impingement. International Journal Sports Medicine 18, 618–624.