

**THE EFFECTS OF LATENT MYOFASCIAL TRIGGER
POINTS ON MUSCLE ACTIVATION PATTERNS
DURING SCAPULAR PLANE ELEVATION**

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THE EFFECTS OF LATENT MYOFASCIAL TRIGGER POINTS ON
MUSCLE ACTIVATION PATTERNS DURING SCAPULAR PLANE
ELEVATION

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DECLARATION

This thesis contains no other material which has been accepted for the award of any other degree or diploma at any institution. Except where reference is made in the text, this thesis contains no material previously published or written by any other person, nor does it contain experimental data from another person's work. The content of this thesis is the results of work which was carried out during the period of candidature.

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LIST OF ABBREVIATIONS

5-HT	5-hydroxytryptamine or serotonin	ICC	Intra-class correlation coefficient
α -MN	Alpha motoneuron	K ⁺	Potassium
γ -MN	Gamma motoneuron	LT	Lower trapezius muscle
AC joint	Acromioclavicular joint	LTrP	Latent myofascial trigger point
Ach	Acetylcholine	MAP	Muscle activation pattern
AChE	Acetylcholinesterase	MAPs	Muscle activation patterns
AChRs	acetylcholine receptors	MD	Middle deltoid muscle
AHP	Afterhyperpolarisation	MEPP/s	Miniature endplate potential/s
ATP	Adenosine triphosphate	MPS	Myofascial pain due to trigger points
ATrP	Active myofascial trigger point	MTrP	Myofascial trigger point
BK	Bradykinin	NMDA	N-methyl-D-aspartate receptor
Ca ²⁺	Calcium ions	PIR	Post-isometric relaxation stretching
CGRP	Calcitonin gene-related peptide	PPT/s	Pressure-pain threshold/s
DDN	Deep dry needling	SA	Serratus anterior muscle
DOMS	Delayed onset muscle soreness	SDN	Superficial dry needling
DRG	dorsal root ganglion	SEA	Spontaneous electrical activity
EMG	Electromyography	sEMG	Surface electromyography
EPN	Endplate noise	SP	Substance P
H ⁺	Proton (hydrogen ion)	SR	Sarcoplasmic reticulum
IL-1 β	Interleukin one beta	SC	Sternoclavicular joint
ICR	Instantaneous centre of rotation	TNF- α	Tumour necrosis factor-alpha
Inf	Infraspinatus muscle	UT	Upper trapezius muscle

SUMMARY

Despite a paucity of experimental evidence, clinical opinion remains that though LTrPs allow pain-free movement, they are primarily associated with motor effects and occur commonly in 'healthy' muscles. In contrast, evidence exists to support the fact that ATrPs are prevalent and a common cause of pain in patients with musculoskeletal pain and have significant effects, including augmentation or inhibition of sensation and because of pain, movement adaptations. The primary aim of this study was to investigate the effects of LTrPs on the muscle activation patterns (MAPs) of key shoulder girdle muscles during scapular plane elevation of the arm, the results of which were presented in Chapters four and five. In connection with the main aim, a preliminary study was carried out to examine the frequency with which LTrPs occur in the scapular positioning muscles in a group of normal subjects.

To investigate the occurrence of LTrPs in the scapular positioning muscles of healthy subjects, a LTrP examination process was tested for intra-examiner reliability (see Appendix C). Subsequently, 154 healthy subjects volunteered to be screened for normal shoulder girdle function and then undergo a physical examination for the presence of LTrPs in the trapezius, rhomboids, levator scapulae, serratus anterior and the pectoralis minor muscles bilaterally. Of these subjects, 89.8% had at least one LTrP in the scapular positioning muscles (mean= 10.65 ± 6.8 , range=1-27), with serratus anterior and upper trapezius harbouring the most LTrPs on average (2.46 ± 1.8 and

2.36 ± 1.3 respectively). Consistent with clinical opinion, this study found that LTrPs occur commonly in the scapular positioning muscles. Having established this, the clinical significance of their presence and the question of whether they have motor effects was investigated, forming the remainder of the thesis.

To establish whether LTrPs in the scapular rotator muscles affected the timing of muscle activation of this muscle group, surface electromyography (sEMG) was employed during elevation of the arm in the plane of the scapula during conditions that occur commonly (unloaded, loaded and fatigued movement). Furthermore, sEMG was also used to measure the muscle activation of functionally related shoulder girdle muscles (infraspinatus as a representative of the rotator cuff group that acts on the humeral head to optimally position it during arm movements and the middle deltoid, an abductor of the arm) during the test movement. These studies found that LTrPs housed in the scapular upward rotator muscles affected the timing of activation and increased the variability of those activation times of this muscle group and were also associated with altered timing of activation in the functionally related infraspinatus and middle deltoid. Compared with the control group (LTrP-free), the MAPs of the LTrP group appeared to be sub-optimal, particularly in relation to preserving the subacromial space and the loading of the rotator cuff muscles. After the initial sEMG evaluations, the LTrP subjects were randomly assigned to one of two interventions: superficial dry needling (SDN) followed by post-isometric relaxation (PIR) stretching to remove the clinical signs of LTrPs or sham ultrasound, to act as a placebo

treatment where LTrPs remained. A subsequent sEMG evaluation found MAPs to be similar to the control group in most of the experimental conditions investigated. Of particular note, when LTrPs had been treated and the subjects repeated the fatiguing protocol, the resultant MAP showed no significant difference with that of the control group in the rested state, suggesting treating LTrPs was associated with an improved response to fatigue induced by repetitive overhead movements.

In conclusion, the findings of the current work were that LTrPs commonly occur in scapular positioning muscles and have deleterious effects of MAPs employed to perform elevation of the arm in the scapular plane during a number of experimental conditions (unloaded, loaded, and fatigued movement) and thus affect motor control mechanisms. Treating LTrPs with SDN and PIR stretching increases PPTs and removes associated taut bands and at least transiently (no follow up was performed) optimises the MAP during scapular plane elevation in commonly occurring conditions. Discussion includes possible neuromuscular pathophysiology that might explain these results.

CHAPTER 1

INTRODUCTION

1.1 Thesis Overview

The primary aim of this study was to investigate the effects of latent myofascial trigger points (LTrPs) on the muscle activation patterns (MAPs) of key shoulder girdle muscles during scapular plane elevation of the arm. In connection with the main aim, a preliminary study was carried out to examine the frequency with which LTrPs occur in the scapular rotator muscles in a group of normal subjects. The thesis is set out in the following manner:

Chapter 1 provides a brief introduction and rationale for the investigations, followed by the main aims. Chapter 2 provides a review of the relevant literature, which in the interests of clarity, has been divided into three sections. The first part (Sections 2.1, page 14) focuses on myofascial trigger points (TrPs) and reviews what is known of the problem that they represent, their clinical characteristics, the underpinning pathophysiology, how they are identified and the reliability of these techniques, and the treatment modalities available. It also presents the most recent description of the evolving “Integrated TrP Hypothesis” and discusses the nature of the active (ATrPs) and latent (LTrPs) forms. Second (Section 2.2, page 81) provides a discussion of elevation of the arm in the plane of the scapula, including kinematics, muscle activation and the upper extremity kinetic chain during optimal function. This is followed by a discussion of how these aspects may be altered by dysfunction, with particular emphasis on the relationships between scapular dyskinesis, rotator cuff overuse or dysfunction and subacromial impingement syndrome. Finally (Sections 2.3, page 105) muscle

activation patterns (MAPs) as one aspect of motor control are introduced and the effects of pain on MAPs outlined. The Chapter concludes with the presentation of the research questions that the experimental work was designed to answer.

Chapter three describes the investigations carried out to establish a protocol to identify the presence or absence of LTrPs in the scapular rotator muscles and includes a discussion on the reliability of this protocol. In addition, the results of a study to measure how commonly LTrPs occur in the scapular rotator muscles in a healthy sample are reported and discussed.

Chapter four reports on experimental work into the effects of LTrPs present in the scapular rotator muscles on MAPs during scapular plane elevation. The chapter includes the methodology employed and discusses the resultant MAPs under a variety of experimental conditions including unloaded, loaded and fatigued states.

Chapter 5 describes the outcomes of an experiment in which subjects with LTrPs performed scapular plane elevation, (under the same conditions as discussed in Chapter four) after undergoing either a clinical LTrP treatment or placebo intervention in order to investigate the effects of removing LTrPs on MAPs.

Chapter 6 summarises the findings and conclusions drawn as well as discussing the limitations of the work and provides suggestions for further study in the area.

1.2 Introduction to the Research Problem

Myofascial TrPs are a common source of pain and disability considered clinically important by various health professionals including general practitioners (McClaflin 1994), dentists (Jaeger 1994), chronic pain specialists (Roth, Horowitz & Backman 1998), gynaecologists (Reiter & Gambone 1991), neurologists (Gerwin, RD 1991), paediatricians (Fine 1987), rheumatologists (Fricton 1994), physiotherapists (Hanten, W. P. et al. 2000), chiropractors (Cohen & Gibbons 1998), osteopaths (McPartland 2004), acupuncturists (Itoh, Katsumi & Kitakoji 2004) and myotherapists or massage therapists (Delaney et al. 2002). Myofascial TrPs produce a well described set of signs and symptoms (Simons, D. G. 2004a) and produce varying degrees of neuromuscular dysfunction (Huguenin, LK 2004) and have been categorised as either 'Active' (ATrPs) or 'Latent' (LTrPs). Active TrPs give rise to pain at rest or upon movement or compression of the affected muscle while, LTrPs have been described as neuromuscular lesions eliciting a pain signal only upon direct compression. From a clinical point of view, ATrPs are thought to be a common source of pain in patient populations and accordingly have been the subject of increased study over the past ten years (Simons, D., Travell & Simons 1999). Conversely, LTrPs have been considered by clinicians to be sub-clinical and perhaps most significant as potential precursors to ATrPs if the affected tissue continues to be subjected to some noxious stimulus (Hong, C. Z. & Simons 1998). Whether they are "forerunners" of ATrPs or produce adverse affects of their own appears to be unknown, though clinical experience would suggest that they are a common

phenomenon in both the pain-free population and patients presenting for treatment of musculoskeletal disorders (Simons, D., Travell & Simons 1999). In addition to causing pain, given that ATrPs are clinically associated with significant motor dysfunctions including muscle weakness, loss of coordination, decreased work tolerance and autonomic phenomena such as abnormal sweating, persistent lacrimation, excessive salivation and pilomotor activity (Simons, D., Travell & Simons 1999), it is important to determine whether LTrPs can also produce deleterious effects since there may be no pain signal to alert the sufferer.

One way of gauging the effects of ATrPs on neuromuscular performance is by recording muscle activation patterns (MAPs) in sufferers performing normal movement patterns. The mechanism(s) by which ATrPs bring about changes in MAPs may relate to a number of factors, including the direct effects of pain (Lund et al. 1991; Sterling, Jull & Wright 2001) but also indirectly due to fatigue which may occur earlier during ongoing activity in painful muscles and muscle groups not recruited in an efficient pattern (Sterling, Jull & Wright 2001). Muscle activation patterns have commonly been investigated through the application of electromyography (EMG), using both surface and indwelling electrodes (Bogey, Cerny & Mohammed 2003) and altered MAPs so revealed, have been associated with painful musculoskeletal conditions such as low back pain (Hodges, P. W. & Richardson, C. A. 1999), ankle sprain (Bullock-Saxton 1994) and shoulder impingement syndrome (Wadsworth & Bullock-Saxton 1997) but have not been previously investigated in relation to myofascial TrPs.

Pain and an earlier onset of fatigue coupled with repetitive muscular activity can exacerbate or lead to overuse injuries which are a common source of disability in sports (Gosheger et al. 2003), work (Fry 1987) or activities of daily living (Barthel et al. 1998). Furthermore, shoulder/neck and upper extremity overuse injuries are second only to lower back injuries in their prevalence among workers and in their cost to industry (Mehlum et al. 2006), making the upper limb a prime target for investigation and the region chosen for the current investigation. More specifically, the shoulder girdle was described as comprising the scapula, clavicle and humerus, their articulations with each other and the muscles that position and stabilise them. The shoulder girdle represents the most proximal of a series of well delineated functional units which must act in concert to ensure the normal positioning, range of motion and strength of the upper extremity and acts in what might be called "intrinsic motions" (where the limb is used relatively independently), and as a link through which forces summed in the lower extremities and torso are transferred to the upper extremity and ultimately to the hand, the most distal segment of the upper extremity kinetic chain (Kibler, W. B. 1998b). Kibler (1998) suggested that where there was dysfunction (caused by such factors as muscle weakness or altered activation patterns) in a proximal segment of a kinetic chain, more distal segments may alter function in order to preserve the movement outcome at the most distal segment. In theory, this process might expose more distal muscles and other tissues to increased loads, predisposing them to overuse injury.

The paucity of information regarding the occurrence and effects of LTrPs, the likelihood that they occur commonly in pain-free individuals and may develop into ATrPs, which have known deleterious effects, brands them worth investigating. Furthermore, overuse injuries in the upper extremity muscles are common and their amenability to the application of sEMG makes them a logical target for the investigation of the effects of LTrPs on muscle function both locally and “downstream” in more distal functional related segments.

The results of this work will contribute to improved understanding of the clinical relevance of LTrPs, the desirability of treating them in the absence of pain and may provide a foundation for future investigations into interventions that may reduce the prevalence of common upper extremity overuse conditions, including myofascial pain (Rashiq & Galer 1999; Simons, D. G., Hong & Simons 2002; Skootsky, Jaeger & Oye 1989), rotator cuff dysfunction (Blevins 1997) and shoulder impingement syndrome (Kibler, W.B. 2006{Michener, 2003 #1821), in those patients with a predisposition to developing them.

1.3 Aims of the Research Project

The general aim of this research was to investigate the effects of LTrPs on the timing of muscle activation in scapular rotator muscles and selected muscles located more distally in the kinetic chain. More specifically the work was designed to:

1. Investigate the frequency with which LTrPs occur within the scapula rotator muscles in a group of normal males and females.

2. Use sEMG to establish the MAPs of selected shoulder girdle muscles during elevation of the shoulder in the scapular plane in a healthy, LTrP-free sample under three conditions:
 - a. Unloaded
 - b. Holding external load
 - c. After fatiguing arm elevations

3. To determine the effects of LTrPs on MAPs by performing the same tests (aim 2) under the same conditions in a sample of individuals having one or more LTrPs in the scapular rotator muscles of the dominant arm.

4. To further test the effects of LTrPs on MAPs by comparing performance of the same tests (aim 2) after either sham (LTrPs remain) or clinically verified LTrP therapy (LTrPs removed) in the LTrP group.

CHAPTER 2

LITERATURE REVIEW

Chapter Overview

This chapter is set out in the following manner: first, the scope of the problem of Myofascial Pain Syndromes (MPS) is briefly presented followed by a discussion of the types of TrPs (the clinical hallmarks of MPS) their identification and differential diagnosis. An overview of pathophysiology, including the underpinning biochemical and electrophysiological features, is then presented to provide insight into the mechanisms by which TrPs exert their actions, which, given the major aim of the current work, is not meant to be exhaustive. The final sections deal with both the optimal and dysfunctional biomechanics of elevation of the arm in the scapular plane (the motion studied) and the underpinning motor control strategies.

Section 1: Myofascial pain syndromes (MPS)

2.1.1 The scope of the problem

In 2002, it was estimated that ten percent of the population of the USA or 23 million people had one or more chronic musculoskeletal problems (Alvarez & Rockwell 2002), while a study performed on the Dutch population suggested that the impact of unexplained musculoskeletal pain syndromes on perceived general wellbeing in the Netherlands was a significant problem for patients and physicians producing considerable economic consequences (Boonen et al. 2005). The same authors suggested that the complex nature of pain

syndromes including myofascial pain syndromes (MPS) and fibromyalgia created uncertainty and feelings of decreased control when compared to better understood inflammatory conditions such as ankylosing spondylitis or osteoarthritis. Given the apparent common occurrence of MPS and the significant adverse effects they appear to have on the physical and psychological wellbeing of humans, further investigation of the factors that underpin these conditions is warranted.

Myofascial trigger points (TrPs) are the characteristic clinical sign of MPS that cause regional muscular pain (Simons, D., Travell & Simons 1999). Though no large epidemiological studies reporting the prevalence of TrPs have been published, anecdotal evidence from experienced examiners implies that pain caused by TrPs is a very common phenomenon (Huguenin, LK 2004; McCain 1994; Simons, D., Travell & Simons 1999), particularly after trauma or sustained muscular fatigue. Supporting this view, Rashiq and Galer (1999) found that 70 percent of 41 patients diagnosed with Complex Regional Pain Syndrome had TrPs in the proximal musculature of the upper limb (Rashiq & Galer 1999). Other studies have reported TrPs as a source of pain in 50 percent of patients with temporomandibular disorders (Schiffman et al. 1990), 54 percent of patients presenting with head and neck pain (Fricton et al. 1985) and 30 percent of patients presenting with pain (unspecified) to a university medical centre (Skootsky, Jaeger & Oye 1989). Although the examination procedures used to identify TrPs in these studies were not uniform, making comparisons difficult, these findings lend support to

the notion that pain due to TrPs is common in patients with a variety of pain complaints. The following section will define these clinical entities.

2.1.2 Myofascial Trigger Points (TrPs): Definitions

The breadth and impact of MPS and the current lack of understanding of underlying mechanisms, provides a strong case for their investigation. Myofascial Pain Syndromes, of which the TrP is the defining clinical sign, is a common pain condition treated by many types of health practitioners (Simons, D. G. 2003). According to the most commonly accepted theory, a TrP is a hypersensitive nodule, or contraction knot contained in a taut band of skeletal muscle (Simons, D., Travell & Simons 1999), as opposed to healthy muscle, which does not contain taut bands or TrPs (Shah, J. P. et al. 2005). The TrP becomes painful, or the pain is exacerbated upon compression and may give rise to a characteristic referred pain pattern, referred tenderness, motor dysfunction and autonomic phenomena (Hong, C. 2006; Simons, D., Travell & Simons 1999; Simons, D. G. 2004a). Trigger points are classified as either Active (ATrP) or Latent (LTrP) with ATrPs causing spontaneous pain, whereas LTrPs are pain-free except when directly compressed, though they may give rise to more mild degrees of the other characteristics associated with ATrPs (Simons, D. G. 2004a).

In addition to being classified as active or latent, TrPs can be defined by location as “Central”, or “Attachment” and also according to precedence as

“Key” or “Satellite”. In these schemes, Central TrPs occur in the muscle belly within the motor endplate zone, while Attachment TrPs are found at the musculotendinous or tenoperiosteal junctions and are thought to be caused by the unresolved tension of the taut band of skeletal muscle produced by a Central TrP, indicating that Attachment TrPs occur secondary to Central TrPs. A Key TrP is responsible for inducing the formation of one or more Satellite TrPs and may be thought of as the ‘primary’ TrP and is usually a Central TrP. Satellite TrPs are considered Central TrPs that have been induced neurogenically or mechanically by the activity of a Key TrP but unlike the Attachment variety, Satellite TrPs have a wider distribution potentially developing in other muscles associated with the referred pain zone of the Key TrP, including synergists of the muscle harbouring the key TrP, or in its antagonists. Illustrating the interdependence of Satellite and Key TrPs, Simons and colleagues (1999) noted that when Key TrPs are treated effectively, symptoms associated with their Satellite TrPs also resolve without requiring direct treatment. The next section provides an overview of the principles which underlie the identification of TrPs in the clinical setting, obviously crucial to any investigation of their effects.

2.1.3 Diagnosis of Myofascial Pain Syndrome: Identifying myofascial TrPs

A diagnosis of MPS relies upon the identification of TrPs in specific muscles where their presence is known to account for a patient’s particular symptoms.

Because there are no readily available, reliable and appropriate objective tests for identifying TrPs, the diagnosis of MPS currently involves the recognition of a number of distinguishing features in the patient history, physical examination and the identification of specific clinical signs that characterise TrPs, as outlined in the following sections.

2.1.3.1 Patient history

Trigger point pain is typically described as a fairly constant, regional, usually deep, dull ache that is exacerbated by the performance of certain movements or adoption of particular postures in contrast to neuropathic pain, which is more commonly associated with burning, electricity-like sensations (Baldry, PE. 2005). Sufferers usually describe one of the following activities as preceding the onset of TrP-related pain:

1. Sudden muscle overload (e.g. a sudden and forceful contraction of the gastrocnemius when pushing off to begin sprinting).
2. Sustained muscular contraction with the muscles in a shortened position (e.g. sustaining head rotation to watch television or read in bed).
3. Repetitive activity, with pain increasing with increased exposure to the repetitive activity (e.g. using a screwdriver). (Simons, D. G. 2004a)

At times, patients may be aware of specific movements that are restricted due to the pain elicited by activating the TrP-affected muscle (Simons, D. G. 2004a) but can often move through a large proportion of the full range of movement at the joints crossed by the affected muscles, with pain or stiffness

appearing only at the end of the movement. For this reason, Simons (2004) suggested that it would be more correct to refer to such movement-related findings as 'increased sensitivity to stretch', rather than as an absolute decrease in the range of movement. In addition to increased sensitivity to stretch, patients may also report a loss of strength in affected muscles, in the absence of obvious atrophy. However, upon questioning the patient or resisted movement testing, it is often apparent that though the patient can perform tasks requiring strength, the effort needed is perceived as greater than before the onset of TrP symptoms. Furthermore the quality or coordination of movement may look or feel "wrong" (Simons, D., Travell & Simons 1999; Simons, D. G. 2003). Finally, Baldry (2005) pointed out, that because of the presence of sympathetic nerve fibres at TrP sites, TrP activity is frequently associated with the development of sympathetically-mediated symptoms including pilomotor changes (localised "goosebumps"), sweating, persistent lacrimation or sensations of intense coldness in the distal part of a limb, all of which can occur spontaneously or when pressure is applied to the tissues overlying a TrP. Where the patient history suggests TrP-mediated pain, a physical examination of specific muscles is initiated to attempt to identify the clinical signs of TrPs as discussed in the following paragraphs.

2.1.3.2 Physical examination findings

Baldry (2005) considered that, locating Active TrPs (ATrPs) through palpation was the most important part of the clinical examination, though he also advocated the use of physical tests to identify, or confirm a patient's reported

limited, painful, or uncoordinated movement. According to Simons and co-workers (1999), the best guide to the precise location of myofascial TrPs is the identification of the “taut band”, a task facilitated by positioning the patient to lengthen the muscle being examined to the point of a perceptible increase in resistance to movement. In this position, normal muscle fibers are still slack but the fibers of any taut bands are placed under additional tension, rendering them more easily distinguishable. When the muscle being examined has been positioned, “snapping palpation” (a cross-fiber plucking motion similar to plucking a guitar string) has been suggested to differentiate any taut bands from adjacent normal muscle fibers. Importantly, the presence of a taut band of skeletal muscle is not considered in itself, diagnostic of the presence of an ATrP and therefore a MPS, because taut bands and LTrPs have been identified in subjects with no pain complaint (Gerwin, R. D. et al. 1997; Njoo & Van der Does 1994; Wolfe et al. 1992). Once a palpable taut band of skeletal muscle has been located, the next critical sign is the identification of a tender nodule within it, by palpating along the taut band searching for a slightly enlarged nodule or the ‘focus’ of the contraction. According to Baldry (2005), these nodules are usually only a few millimetres in diameter, exquisitely painful to external manual compression and constitute the entity clinically referred to as a TrP. In patients who are pain-free prior to external compression, such a TrP is said to be ‘latent’ (LTrP). On the other hand, when pain is present, it is important that the application of external pressure elicits the patient’s complaint, which can be local or referred (Gerwin, R. D. et al. 1997). The presence of referred pain and the extent of the referred pain pattern, whether it be the partial or complete

referred pain pattern associated with a particular TrP, has been taken by Simons and colleagues (1999) as an indication of the irritability or sensitivity of the ATrP. An ATrP that exhibits local and all aspects of the referred pain pattern prior to the application of external compression is thus considered the most sensitive or irritable.

A further diagnostic indicator of the presence of a TrP is the local twitch response (LTR), a transient twitch contraction that occurs either in the fibers of the taut band containing the putative TrP, a different taut band in the same muscle, or in a taut band in another muscle (Simons, D. G. 2004a). The LTR can be elicited by either strong compression of, or needle insertion into, the suspected TrP (Chen, J. T. et al. 2001) and is considered the most objective sign that a TrP has been identified or effectively treated (Gerwin, R. D. et al. 1997; Hong, C. Z. 1994b). Local twitch responses (LTRs) are spinal cord reflexes and have been recorded using electromyography (EMG), and palpated or observed by many authors (Audette, Wang & Smith 2004; Baldry, P. 2002a; Cummings & White 2001; Gerwin, R. D. et al. 1997; Hong, C. Z. 1994a).

In summary, according to current thinking, a myofascial TrP is said to be present when compression of a tender nodule located within a taut band of skeletal muscle reproduces the patient's pain complaint (ATrP) or elicits local or referred pain in otherwise pain-free individuals (LTrP) with confirmation provided by observation, palpation or EMG demonstration of an LTR in

response to stimulation of the TrP with external compression or needle insertion.

In terms of appropriate treatment, Chaitow (2003) stressed the point that the presence of TrPs indicates what he described as “neuromuscular overload” and could be either the cause (primary) or the result (secondary) of a condition where the outcome is neuromuscular overload. In the case of the latter, differential diagnosis of the original condition is clearly essential for effective treatment. Though critical in the diagnosis and treatment of MPS and TrPs, the many conditions that form part of the differential diagnosis are beyond the scope of the current work, however individuals interested in the breadth of the conditions that should be considered are referred to Appendix E (page 287) which provides an overview of the topic.

2.1.4 Clinical characteristics of TrPs

Finally, to highlight the generally agreed clinical characteristics of TrPs, the following list is provided reflecting the opinions of a number of authors (Hong, C. Z. 2000; Hong, C. Z. & Torigoe 1994; Simons, D., Travell & Simons 1999; Yunus 1994):

1. Compression of a TrP may elicit local and/or referred pain that is recognisable to the patient as their clinical complaint (pain recognition), or may aggravate their existing pain (where the TrP is Active).

2. Snapping palpation or rapid needle insertion into the TrP may elicit a local twitch response (LTR).
3. Restricted range of stretch or increased sensitivity to stretch of muscle fibres in a taut band may cause perceived tightness of the involved muscle and some discomfort at the end of the range of motion.
4. A muscle with a TrP may be weak, but usually displays no noticeable atrophy.
5. Patients with TrPs may have localised autonomic phenomena.
6. An ATrP causes pain at rest or in response to movement, whereas a LTrP is asymptomatic except when compressed.

These clinical characteristics will be discussed later in the chapter with regard to the reliability and validity of the TrP examination process (Section 2.1.8, page 55)

2.1.5 Pathophysiology of myofascial TrPs

Based upon extensive work over many years, recently presented in an invited editorial, Simons (2005) suggested that ATrPs usually affect the sensory nervous system by either augmenting sensation, manifested as referred pain and tenderness, or inhibiting it as evidenced by a region of referred anaesthesia. In contrast, he considered that LTrPs more commonly augmented or inhibited the motor functions of the muscle(s) containing them and possibly referred these affects to functionally related muscles (Simons,

D. G. 2005). Both clinical (Simons, D., Travell & Simons 1999) and biochemical (Shah, J. P. et al. 2005) differences have been found when comparing ATrPs and LTrPs, suggesting that they are different entities, however most TrP authorities (Gerwin, R. D. 2005; Hong, C. Z. & Simons 1998; Huguenin, LK 2004; Simons, D. G. 2004b) currently appear to consider that LTrPs can be clinical forerunners of ATrPs. For example, Simons (2005) suggested that though the roles of LTrPs had not been reported in the peer-reviewed scientific literature, they had been the subject of clinical discussion and observation by clinicians, with a growing consensus that the motor effects of LTrPs profoundly influence the coordination of muscle activation and overall balance (Simons, D. G. 2005). In this vein, Simons (2005) posited a mechanism by which LTrPs could progress to the spontaneously active form through muscle overload secondary to altered MAPs. In this model, the abnormal pattern produces overload in inappropriately recruited muscles in order to implement a normal motor program. Hong (2004) suggested that LTrPs may exist in almost every pain-free skeletal muscle and, depending on the stimuli to which that muscle is exposed, could become ATrPs in the face of continued noxious stimuli. More controversially, he considered that though ATrPs could be inactivated (no longer spontaneously painful) through treatment, that they never fully “disappeared”, rather, they converted to the latent form, tender upon compression but not spontaneously painful (Hong, C. Z. 2004). These notions raise a number of questions:

1. Do LTrPs have deleterious effects on motor function if left untreated?
2. Is it possible to completely de-activate LTrPs?

3. Do any positive effects on motor function follow de-activation of LTrPs?

The current experimental program was designed primarily to answer these questions (Chapters 4 and 5).

To consider the suggested nature and effects of Active and Latent TrPs by Hong (2004) and Simons (2005) respectively, and to better understand the clinical presentation of myofascial TrPs it is useful to have some understanding of the underlying pathophysiology. The following section provides the underpinning knowledge for this understanding by presenting an overview of the relevant electrophysiological and biochemical TrP studies, beginning with the former.

2.1.5.1 Electrophysiological studies of myofascial TrPs

(a) Abnormal endplate noise/Spontaneous electrical activity

Many authors have reported electrical phenomena associated with TrPs. A marked increase in the frequency of continuous, low voltage (50-100 microvolts (μV)) electrical activity with occasional spike activity (200 to 700 μV), has been found in both animal (Hong, C. Z. & Yu 1998; Macgregor & Graf von Schweinitz 2006; Simons, D. G., Hong & Simons 1995) and human skeletal muscle (Hubbard, D. R. & Berkoff 1993; Simons, D. G., Hong & Simons 2002), centred on the point of maximal tenderness of a taut band of

skeletal muscle, within the motor endplate zone, that is, a TrP. Originally this TrP associated electrical activity was coined 'spontaneous electrical activity' (SEA), but more recently it has been referred to as abnormal 'endplate noise' (EPN) (Hong, C. Z. 2002; Kuan et al. 2002; Simons, D. G. 2004b; Simons, D. G., Hong & Simons 2002; Simons, D. G. & Mense 2003). Simons and co-workers (1995) found that the continuous low-amplitude electrical activity (10 to 50 μ V and occasionally up to 80 μ V) could be recorded from both active and latent TrP regions, however, the intermittent spike activity (>100 μ V, biphasic) could only be recorded from ATrP regions (Simons, D. G., Hong & Simons 1995). The minute loci from which TrP EPN can be recorded have been defined as the "active loci" of TrPs and are described as dysfunctional motor endplates (Hong, C. Z. & Simons 1998; Simons, D. G., Hong & Simons 1995; Simons, D. G., Hong & Simons 2002). It has been suggested that EPN results from excessive leakage of acetylcholine (ACh) from nerve terminals across the synaptic cleft, resulting in an endogenous shortening of the exposed contractile elements in the absence of a 'nerve-initiated' muscle contraction (Hong, C. Z. 2004; Simons, D.G. 2001). To support this notion, blocking or inhibiting acetylcholinesterase (AChE), an enzyme that breaks down the neurotransmitter acetylcholine at the synaptic cleft, effectively increasing ACh concentrations at the neuromuscular junction, produced intense focal sarcomere contraction in the exposed muscle fibers in rats (Duxson & Vrbova 1985).

The normal nerve excitation-induced quantal release of ACh from pre-synaptic terminal vesicles into the synaptic cleft to be taken up by

acetylcholine receptors (AChRs) in the post-synaptic membrane of the muscle cell is dependent upon the influx of calcium ions (Ca^{2+}) across the pre-synaptic terminal membrane. However, leakage of individual molecules of ACh (termed non-quantal release) from the motor nerve pre-synaptic terminal is neither excitation-induced nor dependent on the presence of Ca^{2+} (Gerwin, RD, Dommerholt & Shah 2004). Both mechanisms of ACh release (quantal and non-quantal) trigger miniature endplate potentials (MEPPs), which in turn can result in a propagated action potential, the usual trigger for muscle contraction. In the case where a TrP is developing, significantly increased non-quantal ACh release is thought to increase the number of MEPPs enough to depolarise an exposed post-junctional membrane to threshold initiating a single fiber action potential that can be recorded as an endplate spike (Simons, D.G. 2001; Simons, D. G., Hong & Simons 2002) As pointed out by Simons and colleagues (1999), this process may produce the taut band of the TrP through contracture of individual sarcomeres by causing a sustained partial depolarisation of the muscle cell membrane, rather than activation of the entire muscle fibre (Gerwin, RD, Dommerholt & Shah 2004), Activation of whole muscle fibres would be expected to result in EMG activity at rest, a phenomenon not associated with TrPs, the exceptions being endplate spikes associated with TrP endplate noise and where a LTR is elicited (Simons, D. G. & Dexter 1995).

As comprehensively discussed by Gerwin and colleagues (2004), AChE can inhibit or terminate ACh action at the post-synaptic neuromuscular junction by breaking it down in the synaptic cleft. This action can decrease the

miniature endplate potential activity associated with TrPs (TrP EPN) along with any motor endplate induced muscle cell depolarisation. However, AChE activity is inhibited by an acidic pH (Mense 2003), such as can result from muscle ischemia and certain exercise regimes (Stauber et al. 1990). In addition, low pH augments the release of Calcitonin Gene Related Peptide (CGRP), which also acts to down-regulate the activity of AChE. These processes result in increased concentrations of ACh available to act on the muscle cell membrane and can cause abnormal EPN. As will be discussed later in the chapter, TrPs have been shown to be associated with both increased CGRP secretion and decreased pH locally, thereby providing a potential mechanism for TrP-related abnormal EPN. Clinically, abnormal EPN may therefore provide a valuable tool to evaluate the effects of different interventions on TrPs. For example, both phentolamine (a sympathetic nervous system blocking agent) (Chen, J. T. et al. 1998) and verapamil (a calcium channel blocker) (Hou, C.R. et al. 2002), have been used to inhibit SEA/EPN at TrP sites in rabbits, confirming the involvement of the autonomic nervous system in the development and perpetuation of TrPs and giving insight into potential methods of treatment.

(b) Local Twitch Response (LTR)

Simons and Dexter (1995) recorded EMG activity from TrP-related taut bands of skeletal muscle in response to snapping palpation of the TrP. In every case, the taut band was electrically silent in the absence of TrP stimulation or when the subject was relaxed. However, intramuscular

electrodes detected obvious electrical (contractile-related) activity milliseconds after snapping palpation of the TrP, which the authors concluded was objective evidence that an LTR resulted from stimulating TrPs. The minute sites within clinically identified TrPs, that when stimulated, elicit LTRs from their associated taut bands, have been defined as the “sensitive loci” of the TrP and are thought to be sensitised nociceptors (Hong, C. Z. & Simons 1998; Hong, C.-Z. et al. 1996). Interestingly, LTRs have also been recorded from muscle tissue outside of the TrP region, suggesting that stimulation of activated nociceptors in the TrP region generates a spinal reflex that has widespread inputs (Hong, C. Z. 1994a) including nearby muscle nociceptors (other than those situated within the TrP region), which may also be also sensitised (Hong, C. Z. & Simons 1998; Hong, C. Z. & Torigoe 1994; Hong, C. Z., Torigoe & Yu 1995). Hence, the transient, contraction that is the LTR can appear at sites other than the taut band containing the TrP, even in a taut band in another muscle (Borg-Stein & Simons 2002; Simons, D., Travell & Simons 1999). Other than the observable twitch, elicitation of an LTR is sometimes associated with quite intense discomfort, paresthesia or sharp pain (Hong, C. Z. 2004), as opposed to the dull, aching pain that digital compression of the TrP usually elicits or exacerbates. However, eliciting an LTR is currently considered the most reliable sign that a TrP has been stimulated and is therefore present (Gerwin, R. D. et al. 1997). Unfortunately, eliciting LTRs requires specific and effective stimulation of the TrP, which in turn requires both adequate access to the TrP and an appreciable level of skill (Hong, C. Z. & Torigoe 1994; Huguenin, LK 2004). In addition, LTRs can vary in size and the number that can be elicited

from multiple stimulations of the same TrP, depends upon the irritability of the TrP (that is, the more sensitive ATrPs more readily produce larger and more numerous LTRs than the pain-free LTrPs) and how effectively the TrP is stimulated (Shah, J P 2003). Shah (2003) suggested that the appearance of a large, visually observable (as opposed to palpable or recordable) LTR during treatment was indicative of a more complete resolution of TrP-mediated symptoms. Indeed, Hong (1994) had previously established the importance of eliciting an LTR as an indication of effective treatment of TrPs (Hong, C. Z. 1994b) and more recent investigations have used the LTR for this purpose. For example, in an animal study investigating the inhibitory effect of dry needling on TrP-related EPN, Chen and co-workers (2001) found that eliciting an LTR was associated with a significant reduction in abnormal EPN at the TrP site (Chen, J. T. et al. 2001). As discussed more fully in later sections of this chapter, Shah and co-workers (2005) collected analytes from the local environment of TrPs pre and post LTR and found that interstitial concentrations of pain mediators were significantly different, particularly at ATrP sites, post LTR, hinting at a mechanism by which LTRs can alter TrP status.

A number of authors (Hong, C. Z. 1994b, 1994a, 2002; Hong, C. Z. et al. 1997; Hong, C. Z. & Simons 1998; Hong, C. Z. & Torigoe 1994; Hong, C. Z., Torigoe & Yu 1995) have provided evidence that LTRs are primarily spinal cord reflexes. Hong (1994b) found that the electrical activity associated with LTRs was diminished in denervated human muscle (Hong, C. Z. 1994a) and nearly completely lost in rabbits following lidocaine block or transection of the

innervating nerve (Hong, C. Z. & Torigoe 1994). In addition, another group lead by Hong found that LTRs were lost in rabbits following spinal shock (induced by spinal cord transection above the level of the nerves supplying the TrP-affected muscle), but returned over a 2.5 hour recovery period, while cutting the motor nerve from the spinal cord to the affected muscle resulted in total loss of LTRs (Hong, C. Z., Torigoe & Yu 1995), indicating that higher levels of the central nervous system are not required for an LTR, but the motor nerve is essential. The fact that the response was diminished rather than abolished in human studies suggests that local transmission (for example: axon reflexes) may also play a role (Hong, C. Z. 1994a).

In summary, eliciting LTRs is extremely useful in first confirming the presence of a TrP and the size and number LTRs elicited during treatment appears to give the clinician an indication of the sensitivity of the TrP and the effectiveness of the TrP treatment.

(c) *TrP referred pain*

In addition to the LTR, sensitive loci are also the sites from which pain and referred pain can be elicited by mechanical stimulation, particularly through needling techniques (Hong, C. Z. 1994b; Hong, C. Z. et al. 1997; Hong, C. Z. & Simons 1998). In an investigation of the referred pain associated with TrPs, Hong and co-workers (1997) found a pressure-dependent ability to elicit referred pain from ATrPs and to a lesser extent LTrPs, but also from what was considered to be normal muscle tissue within the same muscle (Hong,

C. Z. et al. 1997). They hypothesised that the ability to elicit referred pain at the less irritable LTrP and from normal muscle was at least in part due to a chemical sensitisation of nociceptors in and around a TrP. However they did not exclude other recognised mechanisms of referred pain such as localised inflammatory responses, scar or skin TrPs or myofascial TrPs too small to be clinically identified in the 'normal' muscle tissue, any of which could account for referred pain in response to pressure. As an alternative explanation, a number of workers have suggested that referred pain following noxious stimulation of sensitive loci in a TrP is due to central sensitisation in the spinal cord (Hoheisel, Mense & Simons 1993; Hong, C. Z. 2000; Mense & Simons 2001; Woolf & Salter 2000). Central sensitisation in the current context refers to a process in which there is an expansion of the neuron population in the spinal cord responding to ongoing nociceptive input from muscle, resulting in additional sensory neurons being activated (particularly wide dynamic range neurons) and pain perception in the structures associated with the expanded receptive field. In a review of the pathogenesis of muscle pain, Mense (2003) described the mechanism underlying this process and speculated that a sequence of events, described in Figure 2.1, was likely to occur in the dorsal horn neurons.

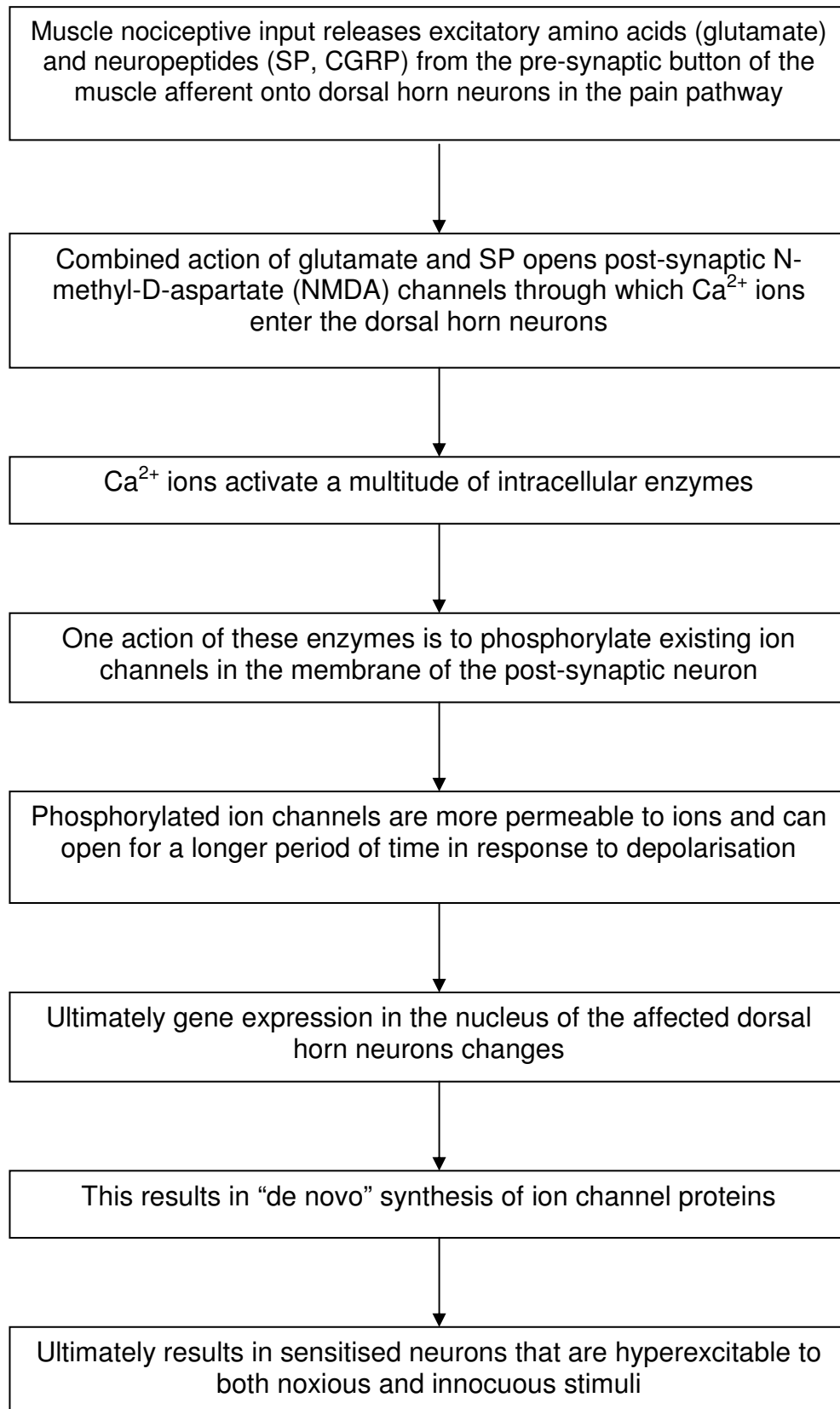


Figure 2.1: Proposed process that underpins the development of a hyperexcitable dorsal horn neuron.

To provide an example of this process, in experiments on anaesthetised rats, the most prominent effect of an acute experimental inflammation of the gastrocnemius muscle was an expansion of the neurons responding to the noxious muscle's afferent input. Dorsal horn neurons responding to stimulation in control animals were restricted to the L4 and L5 spinal levels whereas populations of neurons responding to the inflammatory stimulus in experimental group animals included the L3 in addition to the L4 and L5 levels. This response occurred within a relatively short time period (a few hours), indicating that the population of dorsal horn neurons responding to nociceptive input from the gastrocnemius muscle had grown (Hoheisel, Koch & Mense 1994). This higher "synaptic efficacy" was said to be caused by hyperexcitability of the spinal neurons, involved the actions of numerous neurotransmitters, is called central sensitisation and is well recognised in pain medicine. One example of the complex nature of this process is provided by Mense (2003), who reported that hyperalgesia was mediated by SP acting on neurokinin receptors and glutamate acting on NMDA receptors on post-synaptic dorsal horn neurons, but thought that spontaneous pain resulted from other processes including reduced spinal release of nitric oxide, a substance that is usually released continuously in the dorsal horn and acts to inhibit the background discharge of nociceptive neurons (Hoheisel, Sander & Mense 1995).

To summarise, TrP referred pain arises in a situation where nociceptive input from the muscle is strong or long-lasting, (e.g. where ATrPs are present), leading to the induction of central sensitisation in dorsal horn neurons

associated with pain transmission. Synapses in adjacent spinal cord segments that are usually 'silent' then become responsive to input from the affected muscle leading to an 'expansion' of the 'target area' of the muscle in the spinal cord or brain stem. Should the expansion reach sensory neurons that service peripheral areas other than the affected muscle, the patient perceives pain in those areas as well, even though no injury or damage has occurred in the structures of the new receptive field (Mense 2003).

(d) TrP associated autonomic nervous system involvement

Autonomic phenomena have been observed to develop as a result of TrP activity (Hong, C. Z. 2000; Simons, D., Travell & Simons 1999). and the alpha-adrenergic antagonists phentolamine and phenoxybenzamine have been shown to eliminate (Hubbard, D. 1996) or significantly decrease (Chen, J. T. et al. 1998) EMG spike activity recorded at active loci of TrPs as well as significantly reduce subjective reports of TrP-related pain, observations that confirm autonomic nervous system involvement. In other human studies (Chung, Ohrbach & McCall 2004; Hubbard, D. 1996; McNulty et al. 1994) sympathetic activity has been shown to modulate motor activity at TrPs (EMG activity). In addition, a recent study (Ge, Fernandez-de-Las-Penas & Arendt-Nielsen 2006) provided evidence of a sympathetic-sensory interaction at TrPs manifested as sympathetic hyperactivity to mechanical sensitisation and related sympathetic facilitation of the mechanisms underlying local and referred muscle pain. The authors found that elevating intrathoracic pressure,

(a manoeuvre known to increase sympathetic outflow to muscles) resulted in decreased pain thresholds and increased the perceived intensity of both local and referred pain at TrPs sites, phenomena which did not occur at control sites. These findings infer either local (rather than generalised sympathetic hyperactivity) or some form of differentiated sympathetic activation in the painful and non-painful muscles. Given their findings and the fact that referred pain is a central sensitisation process initiated by peripheral sensitisation (Mense 2004), Ge and colleagues (2006) suggested that sympathetic facilitation of referred pain may involve specific peripheral, spinal and supraspinal sensory and sympathetic structures and their interactions (Ge, Fernandez-de-Las-Penas & Arendt-Nielsen 2006). The mechanisms by which sympathetic activity might facilitate sensory sensitisation are unknown and the data that do exist have been described as controversial (Maekawa, Clark & Kuboki 2002). In any case, Ge and colleagues (2006) speculated that because elevated intrathoracic pressure induces sustained and pronounced sympathetic efferent activity to muscles, resultant increased vasoconstrictor activity might reduce blood flow to TrP-affected muscle fibres and lead to delayed clearance of inflammatory substances. The result is a changed biochemical milieu at the TrP site, one amenable to inducing referred pain and the subject of the following discussion.

2.1.5.2 The Biochemical Milieu of TrPs

Local myofascial pain occurs because of the release of substances from damaged muscle such as adenosine triphosphate (ATP) (Reinohl et al. 2003), bradykinin (BK), 5-hydroxytryptamine (5-HT or serotonin), prostaglandins (PGs) and potassium (K^+) and because of an increase in protons (H^+), causing local acidity, such as occurs with ischemia and exercise (Gerwin, RD, Dommerholt & Shah 2004) In groundbreaking work on TrPs, Shah and co-workers (2005) developed and tested a device to measure the biochemical milieu of muscle tissue in vivo. The measuring device consisted of an acupuncture needle with a hollow bore converting it into a microdialysis needle which could be used simultaneously as an acupuncture needle during routine treatment of TrPs. The investigators recruited three groups of subjects: control group (no pain and no TrPs, N=3); a LTrP group (no pain, but upper trapezius LTrPs, N=3) and an ATrP group (pain and upper trapezius ATrPs, N=3). Analytes were measured at three time points. A 'pre' level was measured two minutes after needle insertion but before needle advancement (used in the control group to simulate needle movement required to obtain a LTR) or eliciting an LTR for both TrP groups. The 'peak' values were measured at five minutes after needle insertion which was immediately after the needle advancement/LTR. The 'post' values were measured at 11 minutes after needle insertion, six minutes after the respective needle movements. The ATrP group had a lower pressure-pain threshold (PPT) than both LTrP and control groups, though this difference did not reach statistical significance ($p < 0.08$). For all three time points combined,

the amounts of the pro-inflammatory cytokines: tumour necrosis factor- α (TNF- α), interleukin-1beta (IL-1 β) and the pain-associated neuropeptides: bradykinin, calcitonin gene-related peptide (CGRP), substance P (SP), 5-hydroxytryptamine (5-HT or serotonin) and norepinephrine were significantly higher in the ATrP group than the other two groups ($p < 0.01$). At peak time, (5 minutes after the start of data collection when the needle was advanced and both TrP groups demonstrated LTRs), peak values of CGRP and SP were significantly different in all three groups (active > latent > normal, $p < 0.02$). In the ATrP group, the 'post' or recovery values (six minutes after LTR had been elicited) of CGRP and SP were significantly lower than the pre and peak values ($p < 0.02$). While accepting potential limitations in the data collection procedure (such as using optimal flow rates for the collection of the analytes), the authors considered their most important finding to be the higher analyte levels and lower pH values for the ATrP group. In addition, the drop in analyte levels that followed the 'peak' values was greatest in the ATrP group, suggesting a greater treatment response due to chemical changes which they associated with the LTR. However, since there was also a decrease of analyte levels in the other two groups, they conceded that needle movement, whether it elicits an LTR or not, may cause similar chemical changes in the immediate vicinity. Importantly, the concentrations were higher in the ATrP group compared with both the LTrP and control groups from the first moment of data collection, which the authors suggested could be due to either an altered biochemical milieu associated with ATrPs, or an increased sensitivity of the tissue surrounding an ATrP to a mechanical stimulation like needle insertion. In addition, local tissue chemical concentrations are known to

fluctuate with variations in blood flow (Langberg et al. 2002). An area of increased oxygen saturation, (presumably associated with increased blood flow), surrounding a central area of hypoxia, (presumably related to ischemia), in the vicinity of TrPs has been demonstrated (Bruckle et al. 1990). This finding resulted in the 'working hypothesis' that TrPs are associated with increased metabolic demand and a decreased ability to support those demands, both concepts implying that ATrPs may induce different blood flow patterns which in turn may alter membrane recovery properties or interstitial chemical concentrations.

Since a number of pain and inflammatory mediators have been found at TrP sites, it is important to consider what each may contribute to the pathophysiology of the TrP. However, it is also important to note that the nature of the relationships between these mediators and MTrPs though likely, is speculative, since much of the discussion is based upon the findings of Shah and colleagues (2005), which though a well designed study, was based upon a small subject pool (N=9).

(a) *Local tissue pH*

Hyperalgesia secondary to mechanical stimulation of muscle arises when the dorsal horn has been bombarded with persistent nociceptive input from peripheral afferents, (such as is presumed to result when ATrPs are present) and is maintained by neuroplastic changes in the CNS, even after the

cessation of nociceptor activity (Sluka, Kalra & Moore 2001). Sluka and colleagues (2001) used an animal model of persistent mechanical hyperalgesia induced by repeated intramuscular injections of low pH saline at levels similar to those seen with tissue inflammation, muscle pain, fibromyalgia and eccentric and maximal concentric exercise, and found that an acidic milieu, in the absence of muscle damage, appeared sufficient to cause significant changes in the properties of nociceptors, associated afferent fibers and dorsal horn neurons. An acidic pH is well known to stimulate the production of bradykinin (also found at ATrP sites) during local ischemia and inflammation (Gerwin, RD, Dommerholt & Shah 2004) and may contribute to an explanation for the occurrence of secondary mechanical hyperalgesia in patients with ATrPs, since lower pH values and secondary mechanical hyperalgesia both appear to characterise ATrPs (Gerwin, RD, Dommerholt & Shah 2004; Shah, J. P. et al. 2005).

(b) Pain-associated neuropeptides:

(i) Calcitonin gene-related peptide (CGRP) and Substance P (SP)

Calcitonin gene-related peptide (CGRP) co-exists with ACh at the alpha motoneuron (α -motoneuron) terminals and acts as a facilitator of ACh release into the synaptic cleft (Mense et al. 2003) It is released when the motor nerve is stimulated or when ACh accumulates, as may occur when acetylcholinesterase (AChE) secretion is inhibited (Mense et al. 2003).

Generally speaking, CGRP is known to be a vasodilator, an augmenter of autonomic and immunologic functions and a modulator of neurotransmission at central and peripheral synapses (Gerwin, RD, Dommerholt & Shah 2004). Importantly, in relationship to TrPs, by increasing the number of surface acetylcholine receptors (AChRs) on the muscle cell membrane near the motor endplate (Fernandez et al. 2003) and by inhibiting AChE activity at the neuromuscular junction (Hodges-Savola & Fernandez 1995), it has been hypothesised that CGRP increases the relative concentration of ACh at the motor endplate, ultimately resulting in an increased frequency of miniature endplate potentials or endplate noise (EPN), followed by sarcomere contraction and the formation of a taut band of skeletal muscle (Gerwin, RD, Dommerholt & Shah 2004) characteristic of all TrPs (see Gerwin et al. (2004) for comprehensive review).

According to Shah and colleagues (2005), SP and CGRP are produced in the dorsal root ganglion (DRG) cells and over 90 percent of these chemicals are transported antidromically to the sensory endings, allowing a constant basal release from the nociceptor to its local milieu (Yaksh 1995). This basal release of SP and CGRP is greatly increased in response to nociceptor activation caused by sensitising agents, such as occurs when H⁺ and BK bind to their receptors on the nociceptor. This results in “bursts” of SP and CGRP release into the muscle where they have a profound effect on the local biochemical milieu and microcirculation by stimulating a continuous cycle of increasing production of inflammatory mediators and neuropeptides, leading to an increasing barrage of nociceptive input to dorsal horn neurons

associated with pain transmission (Gerwin, RD, Dommerholt & Shah 2004). Some orthodromic transport of small amounts of SP are also conveyed from the dorsal root ganglion to the dorsal horn cells, a process that contributes to neuroplastic changes in the dorsal horn which are amplified with prolonged nociceptor activation, ultimately affecting neuronal activity and the perception of pain (Shah, J. P. et al. 2005). Recall that Shah and colleagues (2005) found significantly elevated levels of SP and CGRP in the vicinity of active and latent (ATrPs > LTrPs) TrPs and that SP and CGRP levels decreased significantly after the LTR was elicited in the ATrPs. They suggested this decrease in SP and CGRP concentrations might be due to an “interference with nociceptor membrane channels” or “transport mechanisms that are associated with an augmented inflammatory response”. In addition, the post LTR decrease in SP and CGRP concentrations may occur relative to a local increase in blood flow (Shah, J. P. et al. 2005). Both of these findings provide insight into the potential mechanisms underlying the symptomatic relief that follows TrP treatments that have the capacity to elicit LTRs or increase local blood supply.

(ii) *Bradykinin (BK)*

Muscle cell local ischemia, resulting in local hypoxia and associated with a local acidic pH, is known to stimulate the production of BK in muscle cells (Shah, J. P. et al. 2005; Sluka, Kalra & Moore 2001) which results in first activating, then sensitising muscle nociceptors (Baldry, PE 2001). Shah and

co-workers (2005) found significantly greater BK concentrations in the ATrP group compared with subjects with LTrPs or no TrPs ($p < 0.01$), which might be expected given that ATrPs are spontaneously painful suggesting that nociceptors have been sensitised and patients therefore are aware of pain. Given that LTrPs are painless except when directly compressed, this suggests that nociceptors associated with LTrPs have been activated by lower levels of BK and therefore will react to direct mechanical stimulation of those nociceptors. With continued or increased exposure to BK or other endogenous nociceptor stimulating compounds, nociceptors may become sensitised and cause spontaneous pain, presumably converting a LTrP to an ATrP.

(iii) Serotonin and norepinephrine

According to Shah and colleagues (2005), SP causes mast cell degranulation, with resultant release of serotonin and histamine and the up-regulation of pro-inflammatory cytokines, including TNF- α , which in turn, stimulates the production of norepinephrine (noradrenalin), a process which may explain the significantly elevated levels of serotonin and noradrenalin that were found in ATrP subjects (Shah, J. P. et al. 2005). Since increased levels of noradrenalin are likely associated with increased sympathetic activity in the motor endplate region and increased sympathetic activity has also been associated with ATrPs (Chen, J. T. et al. 1998; Ge, Fernandez-de-Las-Penas & Arendt-Nielsen 2006), Shah's group hypothesised that noradrenaline-mediated increased sympathetic activity may reduce the

mechanical threshold to elicit an LTR, a finding reported by Shah's group in subjects with ATrPs.

(c) *Pro-Inflammatory cytokines:*

(i) *Tumour necrosis factor-alpha (TNF- α) and Interleukin-1beta (IL-1 β)*

Tumour necrosis factor-alpha and IL-1 β were significantly elevated in ATrP subjects in Shah's study (2005). In vivo and in vitro serological studies of peripheral blood and CNS assays have shown TNF- α to be critically involved in the pathogenesis of pain states (Myers, R., Wagner & Sorkin 1999). In animal models, local administration of TNF- α evoked spontaneous electrophysiological activity in afferent C and A-delta nerve fibers, resulting in low grade nociceptive input which contributed to central sensitisation in the dorsal horn. This electrophysiological activity was reduced when anti-TNF- α was administered to these animals (Myers, R., Wagner & Sorkin 1999). Tumour necrosis factor-alpha has also been shown to cause hyperalgesia several hours after injection in rat muscles that was reversed by systemic administration of a non-opioid analgesic, metamizol, indicating that TNF- α is associated with hyperalgesia in an animal model (Schafers, Sorkin & Sommer 2003). Additionally, TNF- α did not cause histopathological evidence of tissue damage nor motor dysfunction, however one day after injection, TNF- α did cause elevated levels of CGRP, nerve growth factor (NGF) and prostaglandin E₂ (PGE₂) in the muscle, potentially influencing the ability of

the muscle to develop a taut band (due to the effects of increased CGRP) or nociceptive pain.

In summary, TNF- α is increased in ATrPs and is capable of inducing ongoing nociceptive activity onto dorsal horn cells, acting as one of the drivers for central sensitisation and expansion of the receptive field, resulting in referred pain, spontaneous activity of dorsal horn transmission cells and an increased magnitude of response from these cells to nociceptive input. These findings suggest that TNF- α and pro-inflammatory cytokines such as IL-1 β may play a role in the development of muscle hyperalgesia and directing treatment at pro-inflammatory cytokines may therefore be beneficial for the treatment of ATrPs (Schafers, Sorkin & Sommer 2003).

A link between the presence of ATrPs and the development of a chronic musculoskeletal pain state has not been specifically studied in a controlled environment, however, local muscle pain is known to be associated with the activation of muscle nociceptors by a variety of endogenous substances including neuropeptides, prostaglandins and inflammatory mediators (Mense & Simons 2001) as previously discussed. Nociceptive receptors in muscle display a host of different receptor molecules in their membranes, including receptors for BK, 5-HT, H⁺ and prostaglandins that are released from damaged tissues. These biochemicals bind with their receptors on nociceptors bringing the membrane closer to threshold for an action potential. Once summation is sufficient, action potentials result, leading to local muscle pain and tenderness (Mense & Simons 2001). Because BK, 5-HT and H⁺

lower the usually high threshold for stimulation of the muscle nociceptors, the activated muscle nociceptors are then more easily sensitised and begin responding to otherwise innocuous, weak stimuli such as light pressure and muscle movement (Shah, J. P. et al. 2005). The continued presence of such biochemicals may therefore underpin the mechanisms that perpetuate TrP pain.

The following section provides an overview of the most current theory of the pathogenesis of TrPs, which brings together much of the biochemical and electrophysiological findings just discussed.

2.1.6 The Integrated Trigger Point Hypothesis

The following proposition is the work of Simons and colleagues (1999) and has been referred to as the “Integrated Trigger Point Hypothesis” and represents an amalgamation of information from electrophysiological and histopathological studies in an attempt to clarify the pathophysiology of TrPs. Simons (1999) first postulated that an ‘energy crisis’ occurred in local muscle tissue when energy requirements, due to persistent levels of increased muscle fiber tension, exceeded supply. In an effort to explain both the origin of the energy crisis and the absence of motor unit action potentials in the palpable taut band of a TrP in resting muscle, it was postulated that an increase of calcium ions (Ca^{2+}) occurred independently of depolarization

stimulated sarcoplasmic reticulum (SR) release. Though damage to either the SR itself or to the muscle cell membrane (sarcolemma) could expose local actin and myosin filaments to a sufficient increase in Ca^{2+} to initiate contractile activity, repair processes could be expected to rapidly respond to such a phenomenon. However, in the case of the taut bands of TrPs, which are electrically silent, it appeared likely that the sustained contractile activity was associated with abnormal depolarisation of the exposed part of the post-synaptic membrane due to continued non-quantal release of ACh from dysfunctional motor nerve terminals. This phenomenon could then account for the contracture of sarcomeres in the vicinity of the motor endplate persisting indefinitely without motor unit action potentials. It then follows that ongoing contractile activity of the sarcomeres markedly increases the metabolic demands of this tissue and the shortening of the filaments compresses the local network of capillaries compromising tissue metabolism which has been shown to fail when contractile activity reaches 30% to 50% of maximal effort, if the contraction is sustained. The result of this combination of events is a localised but critical energy crisis. Removing excessive Ca^{2+} from the muscles fibers should, under normal circumstances, reverse the effect in a short period of time, however, since the Ca^{2+} pump responsible for returning excess Ca^{2+} to the SR, is dependent upon an adequate supply of ATP and also appears to have increased sensitivity to low ATP levels relative to the contractile mechanism itself, impaired uptake of Ca^{2+} associated with the effects of the energy crisis further exposes the contractile elements to continued high concentrations of Ca^{2+} and continued contractile activity –

completing a vicious cycle (Simons, D., Travell & Simons 1999). The process is summarised in Figure 2.2.

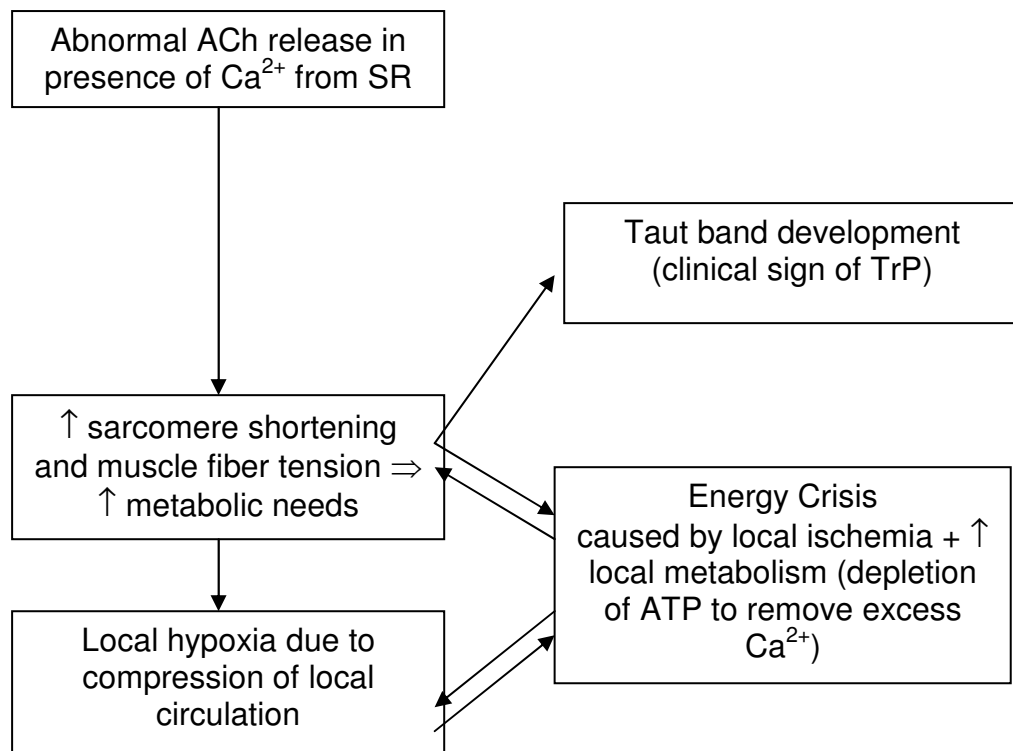


Figure 2.2: The development and perpetuation of the ‘energy crisis’ component of Simons’ Integrated TrP Hypothesis. Adapted from Simons et al. (1999, p. 71).

Taking the hypothesis further, Simons concluded that the tissue energy crisis (incorporating severe local hypoxia) might be associated with the release of vasoreactive and nociceptor sensitising substances such as H⁺, bradykinin, CGRP, SP, TNF- α , IL-1 β , serotonin and noradrenalin, thus resulting in nociceptive and autonomic sensory afferent input to the dorsal horn, as depicted in Figure 2.3 taken from Simons (2004a).

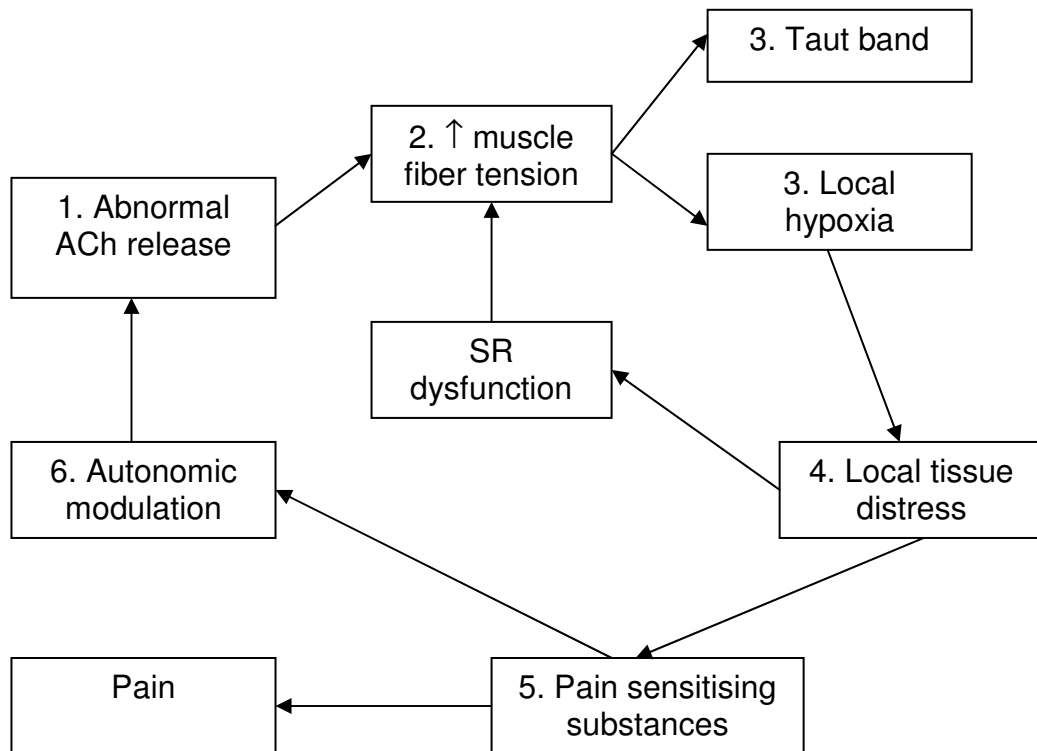


Figure 2.3: Flow diagram of Simons' integrated hypothesis. The numbers indicate a possible order, though the cyclic nature of the relationship between events has been noted. Reproduced from Simons (2004a).

Another hypothesis to explain the pathogenesis of TrPs that combined the experimental findings of the motor and sensory phenomena associated with TrPs was published by Hong and Simons (1998). Figure 2.4 provides their schematic of the TrP which they described as multiple active loci in a TrP region from which EPN could be recorded. This low amplitude, continuous electrical activity was said to be caused by increased concentrations of ACh at dysfunctional motor endplates which contributed to a focal contracture of local sarcomeres, the precursor to the formation of a taut band. They stated

that a TrP region and its surrounding muscle tissue also contained many sensitive loci, which were in fact sensitised nociceptors from which local pain, referred pain and LTRs could be elicited when adequately stimulated (Hong, C. Z. 2004).

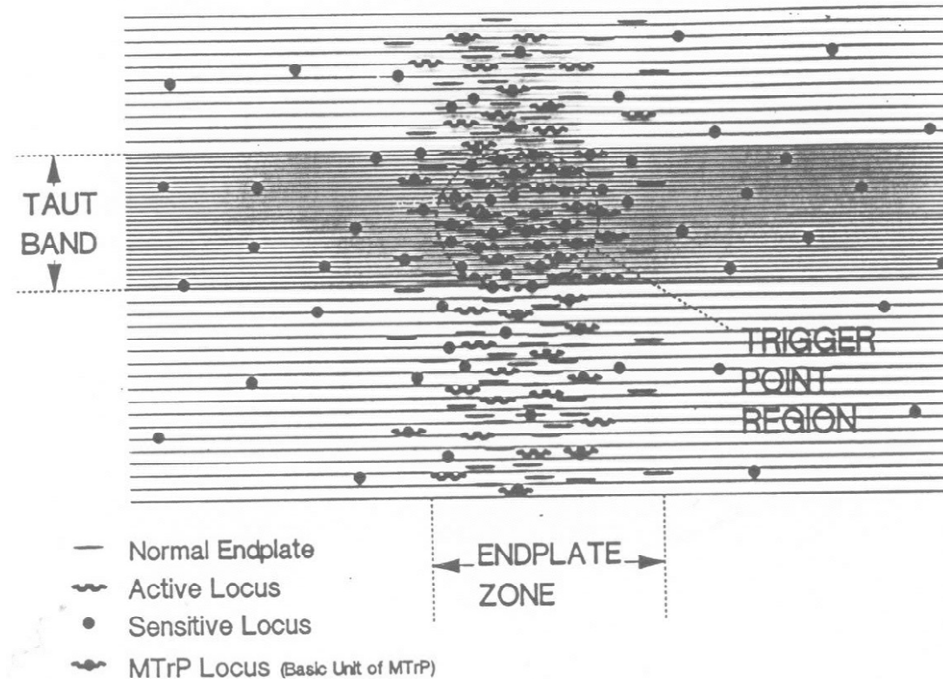


Figure 2.4: Schematic of the motor and sensory components of a myofascial TrP. Reproduced from Hong and Simons (1998).

2.1.7 An expansion of the Simons' Integrated Hypothesis

In an extensive discussion incorporating recent and related studies, with the intent of expanding on Simons' "Integrated Hypothesis", Gerwin and colleagues (2004) concluded that in healthy muscle there exists an equilibrium between the release, breakdown and removal of ACh from its receptors (AChRs) in the post-synaptic membrane by acetylcholinesterase (AChE) that is disturbed by muscle injury. In injured muscle, there is a

release of substances that activate muscle nociceptors causing pain but also facilitating ACh release, inhibiting ACh breakdown and removal from the AChRs and up-regulating AChRs. These changes could then result in an ongoing increased binding of ACh to the muscle cell membrane, leading to the development of persistent sarcomere contraction, as is characteristic of the myofascial TrP. The authors (Gerwin, RD, Dommerholt & Shah 2004) hypothesised that the activating event in the development of TrPs was the execution of either unaccustomed eccentric exercise or maximal concentric exercise all of which can lead to muscle fiber damage and to segmental hypercontraction within the muscle fiber. The resulting hypoperfusion (caused by capillary constriction), might then increase the damage caused by continuation of the exercise, exacerbated by increased capillary constriction through sympathetic nervous system adrenergic activity. The resultant ischemia and hypoxia then add to the development of tissue injury with a resultant local acidic pH which in turn inhibits AChE activity, increases release of CGRP and activates acid sensing ion channels (ASICs) on muscle nociceptors as previously discussed. An acidic pH, whether induced experimentally (Sluka, Kalra & Moore 2001), or resulting from inflammation, or muscle overload through exercise, is sufficient by itself to cause widespread changes in the properties of nociceptors, axons and dorsal horn neurons. In addition, any breakdown of muscle fibers (that might result from exercise), results in the release of pro-inflammatory mediators such as SP, CGRP, BK, 5-HT and cytokines (TNF- α and IL-1 β) that can profoundly alter the functioning of the motor endplate and the sensitivity and activity of muscle nociceptors as well as wide dynamic range neurons in the spinal

cord. Increased availability of ACh, caused by several factors (increased release of ACh mediated by CGRP; increased pre-synaptic motor terminal adrenergic receptor activity; inhibition of AChE caused by CGRP; up-regulation of AChRs through the action of CGRP, acidic pH), leads to increased motor endplate activity. In their scenario (Gerwin, RD, Dommerholt & Shah 2004), the taut band then results from the increase in ACh activity. With regard to sensory changes associated with TrPs the model implicates alterations in miniature endplate potential frequency, or EPN, which is increased as a result of greater ACh effect. Release of BK, K⁺, H⁺ and cytokines from injured muscle, activate nociceptors, thereby causing tenderness and pain. The presence of CGRP compels the system to become chronic, potentiating the motor endplate response and, along with SP, potentiates the activation of muscle nociceptors. The combination of acidic pH and pro-inflammatory mediators at the ATrP contributes to segmental spread of nociceptive input into the dorsal horn and leads to the expansion of multiple receptive fields. These neuroplastic changes in dorsal horn neurons occur in response to a continuous nociceptive barrage, with further activation of neighbouring and regional dorsal horn neurons that now have lower activation thresholds. This results in the observed phenomena of hypersensitivity, allodynia and referred pain that is characteristic of ATrPs (Gerwin, RD, Dommerholt & Shah 2004) (see Figure 2.5, reproduced from Gerwin, Dommerholt and Shah (2004)).

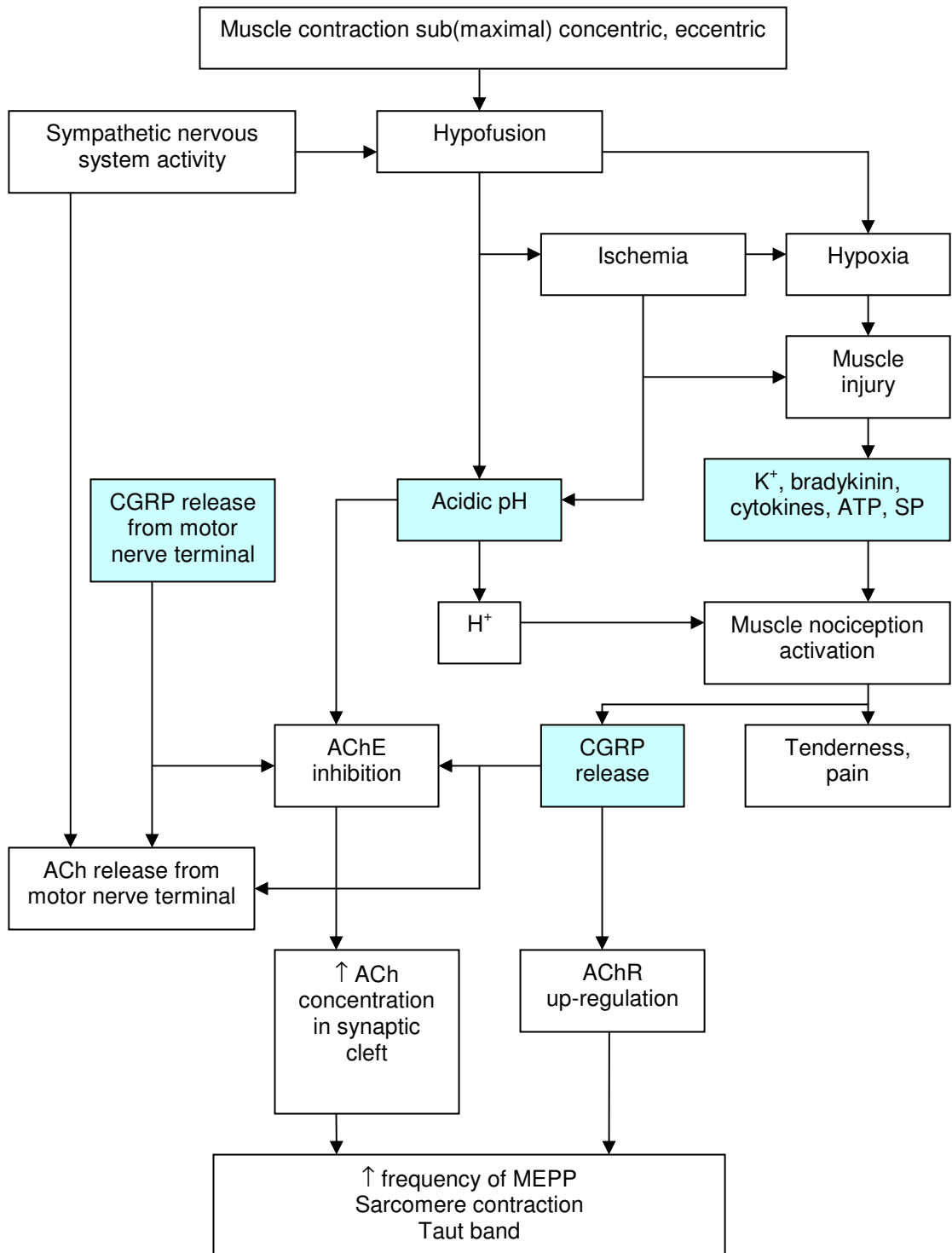


Figure 2.5: A schematic outline of the expanded TrP hypothesis. (ACh-acetylcholine, AChE-acetylcholinesterase, AChR-acetylcholine receptors, ATP-adenosine triphosphate, CGRP-calcitonin gene-related peptide, H⁺-protons, K⁺-potassium, MEPP-miniature endplate potential, SP-substance P) Reproduced from Gerwin et al. (2004).

To clarify, the figure illustrates the following steps in the model put forward by Gerwin and colleagues (2004): the activating event is muscle activity that stresses muscle beyond its tolerance and leads to muscle injury and capillary constriction. Muscle injury results in release of substances that activate muscle nociceptors and causes pain. Capillary constriction occurs as a result of both muscle contraction and sympathetic nervous system activation resulting in hypofusion and ischemia. The local pH becomes acidic, inhibiting AChE activity. CGRP is released from the motor nerve terminal and from injured muscle. CGRP inhibits AChE, facilitates ACh release and up-regulates AChRs. The end result is increased ACh activity with increased frequency of miniature endplate potentials (MEPPs), sarcomere hypercontraction and the formation of taut bands. The highlighted boxes indicate those events that have been identified or are supported by microdialysis studies of the TrP. (Gerwin, RD, Dommerholt & Shah 2004).

Now that the potential mechanisms of TrP pathology have been highlighted, the ability to accurately identify them in both clinical and research settings, (a critical factor in assessing, treating or researching TrPs effectively), will be discussed.

2.1.8 The identification of TrPs and the reliability of the examination process

Where the patient history suggests TrP-mediated symptoms, the ability to identify the appropriate clinical signs is obviously of great importance, since no imaging or laboratory tests exist to diagnose them (Borg-Stein & Simons 2002). In addition, there are no official clinical diagnostic criteria for their identification, although a number of authors have suggested that the minimal criteria are “spot tenderness”, “pain recognition” and a “taut band”. Confirmatory signs include eliciting referred pain and local twitch responses (LTRs) (Gerwin, R. D. et al. 1997; Simons, D. G. 2004a).

Attempts have been made to objectify the identification process. An early pioneer in this area, Fischer (Fischer, AA 1987b, 1987a; Fischer, A. 1988; Fischer, L. 1999; Kraus & Fischer 1991), validated the use of a pressure algometer as a reliable and useful tool for measuring the pressure-pain threshold (PPT) of TrPs and compared them to the PPT of normal muscle tissue and thus defined the PPT as the minimum pressure causing a pain response (Fischer, A. 1988; Hanten, W. et al. 2000; Reeves, Jaeger & Graff-Radford 1986). Reeves and co-workers (1986) conducted three small studies and concluded that pressure algometry measurements were reliable in measuring the PPT of TrPs reporting good to excellent intra- and inter-examiner reliability (($r=0.69-0.97$, $N=15$) and ($r=0.71-0.89$, $N=15$)) respectively. In their work, the validity of measuring the PPT of TrPs was demonstrated by discriminating between TrPs and adjacent, non-TrP muscle

tissue. In a related study, dealing with PPTs and associated referred pain, Hong and colleagues (1997) found that when no limit was placed on the amount of pressure used by examiners, referred pain could be elicited not only from sensitive loci within ATrPs, but also from the taut band and normal muscle tissue (Hong, C. Z. et al. 1997). Together, these results suggest that using PPTs to standardise the pressure under which a lesion responds with pain may decrease the likelihood of obtaining false positives when examining for the presence of TrPs. Many studies (Edwards & Knowles 2003; Fernandez-de-Las-Penas, C et al. 2003; Ge, Fernandez-de-Las-Penas & Arendt-Nielsen 2006; Hong, C-Z. 1998; Hou, C. R. et al. 2002; Kamanli et al. 2005; Sciotti et al. 2001; Simons, D. G. 1988, 2004b) have since used algometers to measure PPTs of TrPs to contribute to the identification process and also to measure any changes post-intervention.

Another approach to the problem of reliable TrP identification has been the use of thermography. While early results showed promise (Diakow 1988, 1992; Kruse & Christiansen 1992), Swerdlow and Dieter (1992), soon showed that thermographic 'hot spots' often observed in the upper back were not ATrPs, thereby casting doubt on thermographic techniques for TrP identification (Swerdlow & Dieter 1992). In still another approach, Sciotti and colleagues (2001) specifically focused on the precision with which four trained clinicians could locate LTrPs in the upper trapezius muscle of 10 subjects, as opposed to measuring the inter-examiner reliability of identifying the clinical signs of a TrP (for example taut band, tender point, LTR etc). Using a three-dimensional camera system they found this system a valid tool

for measuring the location of LTrPs in the upper trapezius to a similar level of precision as the most commonly used locating methods – the clinicians' fingers.

In terms of the clinical signs of TrPs: spot tenderness, jump sign (where the patient 'jumps' or moves because of pain caused by compressing the TrP), pain recognition, taut band, referred pain and LTR, a number of authors have carried out studies of inter-examiner reliability calculating and reporting the resultant Kappa statistics (Gerwin, R. D. et al. 1997; Hsieh et al. 2000; Nice et al. 1992; Njoo & Van der Does 1994; Wolfe et al. 1992)). Table 2.1 adapted from Simons and colleagues (1999, p. 32), summarises the available Kappa values for each clinical sign.

Table 2.1: Kappa values for inter-examiner reliability of examinations for TrP characteristics.

Examination for clinical sign	Wolf et al. 1992	Nice et al. 1992	Njoo et al. 1994	Gerwin et al. 1997	Hsieh et al. 2000 (best of 2 studies)	Mean
Spot tenderness	0.61		0.66	0.84		0.70
Jump sign			0.70			0.70
Pain recognition	0.30		0.58	0.88		0.59
Taut band	0.29		0.49	0.85	0.22	0.46
Referred pain	0.40	0.38	0.41	0.69	0.34	0.44
LTR	0.16		0.09	0.44	0.12	0.19
Mean	0.35	0.38	0.49	0.74	0.23	

As can be seen, inter-examiner reliability varied with the different clinical signs with the most consistent results found with spot tenderness, jump sign and pain recognition, then locating a taut band and eliciting referred pain, with the least agreement between examiners found for the LTR. In agreement, Simons and colleagues (1999) rated the difficulty of identifying each clinical sign from easiest to most difficult as follows: spot tenderness and jump sign < pain recognition < taut band and referred pain, with eliciting an LTR being the most difficult (Table 2.2) (Simons, D. 1997). The same author estimated the diagnostic value of each sign (regardless of examiner

agreement or ease of examination), from least to most valuable as: jump sign, referred pain and spot tenderness < taut band < pain recognition < LTR, though conceding that successful identification was dependent upon the degree of “palpatory access” to the muscle. Simons and colleagues (1999) also suggested that the combination of spot tenderness within a palpable taut band identified by a skilled examiner, was likely to be ‘highly diagnostic’ of a TrP.

Table 2.2: Comparative reliability of diagnostic examination for TrPs, including estimates of relative difficulty of performing the examinations and estimated relative diagnostic value of each examination by itself regardless of other findings. Reproduced from Simons et al. (1999), p. 33.

	No of Studies	Mean Kappa	Difficulty	Diagnostic value alone
Jump sign	1	0.70	+	+
Spot tenderness	3	0.70	+	+
Pain recognition	3	0.59	++	+++
Taut band	4	0.46	+++	++
Referred pain	5	0.44	+++	+
LTR	4	0.19	++++	++++

One ‘+’ = easiest to identify or least diagnostic value. Multiple “++++” = most difficult to identify or greatest diagnostic value.

In the light of multiple reliability studies (Lew, Lewis & Story 1997; Nice et al. 1992; Njoo & Van der Does 1994; Wolfe et al. 1992) (as referred to above) that have found poor inter-examiner reliability in identifying TrPs, a number of authors either stated (Gerwin, R. D. et al. 1997; Sciotti et al. 2001) or suggested (Lew, Lewis & Story 1997) that the accurate identification of TrPs relied heavily upon effective palpation skills and specific knowledge of musculoskeletal structure and function and was therefore an individual skill that might be trainable. However, in the most recent inter-examiner reliability study located, Hsieh and colleagues (2000) found that the reliability for identifying the clinical signs of TrPs (palpating a taut band, eliciting referred pain and LTRs) in muscles associated with low back pain in 52 subjects (26 asymptomatic and 26 subacute low back pain) was no different between trained and untrained clinicians (Hsieh et al. 2000), a finding contrasting with those of Gerwin's group (Gerwin, R. D. et al. 1997). It has been suggested that this discrepancy may in part be explained by the different anatomical regions and patient populations studied by the two groups (Sciotti et al. 2001).

Only one study was identified that investigated the intra-examiner reliability of identifying the clinical signs of TrPs in the rotator cuff muscles (one muscle group under investigation in the current work). In 51 patients diagnosed with rotator cuff tendinitis (Al-Shenqiti & Oldham 2005), Al-Shenqiti and Oldham (2005) used a test-retest protocol over three days, and found that the same examiner reliably identified the presence or absence of the taut band, spot tenderness and pain recognition, but had more variable success with referred

pain and LTRs (Table 2.3), suggesting the former clinical signs were more reliable than the latter in identifying TrPs.

Table 2.3: Kappa statistics for intra-examiner reliability of the identification of clinical signs of TrPs in the rotator cuff muscles of 51 patients with rotator cuff tendonitis, extracted from the results of Al-Shenqiti and Oldham (2005).

Examination for clinical sign	Al-Shenqiti and Oldham 2005
Spot tenderness	1
Jump sign	1
Pain recognition	1
Taut band	1
Referred pain	0.79-0.88
LTR	0.75-1
Mean	0.92-0.98

As previously stated, Gerwin and colleagues (1997) listed the minimal criteria for ATrP identification as ‘spot tenderness’, ‘pain recognition’ and a ‘taut band’, with confirmatory signs including ‘referred pain’ and ‘LTR’. However, the ability to elicit or recognize a LTR has consistently been found to have poor inter-examiner agreement suggesting its utility is dependent upon palpatory access to the muscle fibers being examined and the skill and experience of the examiner. Notwithstanding, the LTR has been identified as

the most reliable sign of TrP presence (Shah, J P 2003; Simons, D. 1997), and has also been linked with effective treatment (Hong, C. Z. 1994b; Shah, J P 2003) (see also pages.19-21 and 28-31), hence, the ability to effectively stimulate a TrP to elicit LTRs, either by palpation or needling, is a clinical skill of utmost importance for the successful management of myofascial TrPs.

Two studies have investigated aspects of TrP identification in asymptomatic subjects alone. In the first, Lew and colleagues (1997) used two experienced clinicians to identify the location of LTrPs in the upper trapezius muscles of 58 volunteers and mark their locations on an enlarged body diagram. These authors did not report Kappa statistics because of limitations in their data, but reported that both examiners showed complete agreement on the LTrP location in two subjects (3.85% of those subjects with LTrPs) and both identified the same six subjects who had no LTrPs in the upper trapezius (Lew, Lewis & Story 1997), implying that inter-examiner agreement for locating LTrPs in this muscle was poor. Sciotti and co-workers (2001) also published results pertaining to the inter-examiner reliability of precisely locating LTrPs in the upper trapezius of 20 healthy subjects using a highly sensitive and specific three-dimensional camera analysis system. They used a coordinate system to record the positions of LTrPs in the upper trapezius in three planes as they were identified by two trained examiners and reported that their ability to reliably localise them essentially approached a precision limited only by the physical dimensions of the clinician's own fingertips. The same authors also suggested that the clinical characteristics of a LTrP might be somewhat different to those of an ATrP, particularly with regard to the

better inter-examiner reliability of identifying the taut band of the LTrP, a suggestion they based upon earlier work that found lower kappa values for identification of the taut band in ATrPs (Gerwin, R. D. et al. 1997; Wolfe et al. 1992). In support of this contention, Hsieh and colleagues (2000) found that the 'expert examiner' in their study was able to identify a taut band in 70% of 10 muscles in 26 healthy subjects, a finding that also supported the idea that the taut band may have greater reliability as a clinical sign for LTrPs than for ATrPs. In addition, because LTrPs are pain-free unless stimulated by digital compression or needle insertion, the 'pain recognition' criterion for TrP recognition would seem to be more appropriately defined as the *presence of local and/or referred pain in response to stimulation*. Finally, because LTrPs are also likely to produce less marked examination findings than ATrPs (Simons, D. G. 2004a), the criteria of 'a tender nodule within a taut band that elicits pain upon compression' are all that are required to identify the less irritable LTrPs.

Considering that the focus of the present study, it is important to gain some insight into how commonly TrPs occur. The following section provides information on the prevalence of TrPs in general followed by an introduction to the prevalence of LTrPs in particular. The available information on LTrPs is fleshed out in Chapter three which presents an investigation of the topic.

2.1.9 The prevalence of TrPs

The information under this heading has also been presented at the beginning of Chapter Three which presents a study of the occurrence of LTrPs in the scapular rotator muscles (page 119). Its duplication here is in the interests of continuity through the literature review and for the reader's convenience.

There have been no large epidemiological studies specifically examining the prevalence of TrPs (Baldry, PE 2001), although anecdotal evidence from experienced examiners implies that pain caused by TrPs is a very common phenomenon (Huguenin, LK 2004; McCain 1994), particularly after trauma or sustained muscular fatigue. In support of this view, Rashiq and Galer (Rashiq & Galer 1999) found that 70 percent of 41 patients diagnosed with Complex Regional Pain Syndrome had TrPs in the proximal musculature of the upper limb. Other studies have reported TrPs as a source of pain in 50 percent of patients with temporomandibular disorders (Schiffman et al. 1990), 54 percent of patients presenting with head and neck pain (Friction et al. 1985) and 30 percent of patients presenting with pain (unspecified) to a university medical centre (Skootsky, Jaeger & Oye 1989). An average of 3.9 TrPs was found in the upper trapezius, sternocleidomastoid and temporalis muscles of 25 patients with chronic tension type headache (Fernandez-de-Las-Penas, C et al. 2006) and a mean of 4.3 in the upper trapezius, sternocleidomastoid and levator scapulae muscles of 20 patients with mechanical neck pain (Fernandez-de-Las-Penas, C., Alonso-Blanco & Miangolarra 2006) Although the examination and reporting procedures used to identify TrPs were not

uniform, making comparisons difficult, these studies lend support to the notion that pain due to TrPs is common in patients with a variety of pain complaints. While circumstantial evidence is mounting to support the idea that myofascial TrPs (ATrP and LTrP) are prevalent in those suffering from musculoskeletal pain, some clinicians have suggested that the prevalence of LTrPs in healthy individuals is even higher (Simons, D., Travell & Simons 1999), or even innately normal (Hong, C. Z. 2004). However, a paucity of experimental data are available to support this idea. In an earlier study, Sola and co-workers (1955) investigated what they described as the occurrence of “hypersensitive spots” in the posterior shoulder muscles of 200 healthy, young military recruits (Sola, Rodenberger & Gettys 1955). It was later suggested by Simons and colleagues (Simons, D. 1997; Simons, D., Travell & Simons 1999), that the “hypersensitive spots” identified in 50 percent of this sample were probably LTrPs. The only other information concerning the occurrence of LTrPs in healthy, pain-free subjects comes from studies that have used control subjects for comparison with patient populations. For example, as mentioned in the previous paragraph Fernandez-de-Las-Penas and colleagues (2006) conducted two studies that reported the numbers of LTrPs in healthy controls, one in relation to patients with chronic tension type headache (Fernandez-de-Las-Penas, C et al. 2006) and the second looking at patients with a mechanical cause of neck pain (Fernandez-de-Las-Penas, C., Alonso-Blanco & Miangolarra 2006). In the first they found an average of 2.0 LTrPs in the upper trapezius, sternocleidomastoid and temporalis muscles of 20 healthy subjects and 1.4 LTrPs in the upper trapezius, sternocleidomastoid and levator scapulae muscles of 25 healthy subjects

(Fernandez-de-Las-Penas, C et al. 2006) in the other. In general, it may be concluded that the available evidence supports the notion that TrPs are a common phenomenon, though the case for LTrPs is less convincing. This situation provided the rationale for the study on LTrP prevalence reported in Chapter three of the present work.

The next section provides information pertaining to the treatment of TrPs, which formed part of the basis upon which the effects LTrPs on MAPs in the shoulder girdle muscles could be confirmed, (Chapter five).

2.1.10 Treatment of Myofascial TrPs

It has been suggested that ice, heat, ultrasound and massage are used in the treatment of TrPs because these modalities might achieve temporary relief of TrP-mediated pain (Fernandez-de-Las-Penas, C et al. 2003). Unfortunately, to date, there have been no controlled studies that support this proposition (Hanten, W. et al. 2000). Treatments such as dry needling or injection with lidocaine (Hong, C. Z. 1994b; Kamanli et al. 2005) or botulinum toxin A (Kamanli et al. 2005), spray (with vapocoolant) and stretch (Jaeger & Reeves 1986), transcutaneous electrical nerve stimulation (TENS) (Graff-Radford et al. 1989) and post-isometric relaxation (Lewit & Simons 1984) have all been investigated for their effectiveness in resolving TrP pain (measured with visual analogue scales - VAS) and/or altering pressure-pain thresholds (PPTs), most often by taking pre and post pain measurements for

comparison. However, these studies had no control groups. Interestingly, Baldry (2002) reported that various dry needling techniques, injections of analgesics and the spray and stretch technique were the most commonly reported forms of TrP treatment in the medical literature (Baldry, P. 2002b).

Therapeutic interventions used to treat TrPs can be divided into three categories:

- (i) *Manual therapies*; including ischemic compression, spray and stretch, strain and counterstrain, muscle 'energy techniques', trigger point pressure release and transverse friction massage.
- (ii) *Needling therapies*; dry needling (superficial and deep), injections (analgesics, corticosteroids, Botox A, vitamin B12).
- (iii) *Other techniques*; thermotherapy, transcutaneous electrical nerve stimulation (TENS), ultrasound therapy, laser therapy, magnetic stimulation therapy, exercise (Fernandez-de-Las-Penas, C et al. 2003) and frequency specific microcurrent (McMakin 2004).

Each of these categories of treatment are now briefly discussed either in the coming sections or presented in various appendices as referred to in the text.

2.1.10.1 *Manual therapies*

A systematic review of the effectiveness of various manual therapies for the treatment of myofascial TrPs conducted by Fernandez-de-Las-Penas and co-workers (2003) revealed few randomised controlled trials (RCTs) on the

subject and none that showed any statistically significant effects beyond placebo. Although no effect beyond placebo was found for any manual therapy, some findings of the trials discussed below may provide a platform upon which to base future, more rigorous work.

In a trial that studied various combinations of exercise, manual therapy, stretching and thermal modalities, Hou and colleagues (2002b) found that all groups that received some form of electrotherapy (eg: TENS, interferential current) as part of the therapy combination, had significantly reduced pain intensity compared to the group that received 'hot pack' intervention (Hou, C. R. et al. 2002). The same authors also found that the combination of hot pack, range of motion exercise, interferential current (IFC) and myofascial release, produced the largest reduction in pain immediately after treatment, though long-term effects were not measured. Similarly, Hanten and colleagues (2000) compared the effects of a home program of self-administered ischemic compression followed by stretching with a control treatment of active range of motion exercises in a group of 40 adults with neck and upper back pain and found a significant increase in PPT for the treatment group, but no difference in total duration of pain (measured with VAS) three days post treatment. In contrast, Edwards and Knowles (2003) reported no difference in TrP pain scores between stretching and control groups and suggested that where TrPs had not been de-activated, stretching might be a pain aggravator, a notion supported by others (Simons, D., Travell & Simons 1999).

In a trial comparing the effects of massage, exercise and ultrasound to massage, exercise and placebo ultrasound in 58 patients with neck and shoulder TrPs (Gam et al. 1998), the TrP number and pain intensity were significantly reduced for both groups receiving massage and exercise, suggesting that ultrasound had no therapeutic effect on neck and shoulder TrPs. Similarly, in a trial that compared Thai Massage combined with stretching with a Swedish massage and stretching combination, Chatchawana and co-workers (2005), found that both groups had significant reductions in pain and disability measures, though there were no differences between treatments (Chatchawana et al. 2005). An immediate improvement within groups, but not between groups, was also reported in a trial comparing the techniques of ischemic compression with transverse friction massage (Fernandez-de-las-Penas et al. 2005).

In summary, from the research and clinical opinion cited, there appears to be no incontrovertible evidence to support the efficacy beyond the effect of placebo of the manual therapies currently used in the management of myofascial pain syndromes caused by TrPs. There is some support for the application of some interventions (spray and stretch, deep pressure, soft tissue massage and ischemic compression) as effective in reducing pressure-pain sensitivity and pain in TrP conditions, however many trials (Fernandez-de-Las-Penas, C et al. 2003; Hong, C. 2006; Hou, C. R. et al. 2002; Rickards 2006) have combined treatments and therefore any improvement in symptoms can only be related to the combined effect as opposed to any single modality.

2.1.10.2 *Needling therapies*

A thorough history of the development of TrP needling therapies has been provided by (Baldry, P. 2002a) starting in the 1940s when Janet Travell began her life-long study of myofascial pain and TrPs, terminology she introduced to the medical world. According to Baldry (2002), she realised that the analgesia produced by injecting procaine into a TrP could not be due to the nerve blocking effect of this drug because the effect lasted too long. She later observed that pain relief of a similar duration could be obtained by inserting a needle into the TrP without injecting any substance, however, not surprisingly, the soreness produced was much greater than occurred when an analgesic was injected (Cummings & White 2001; Hong, C. Z. 1994b; Kamanli et al. 2005; McMillan, Nolan & Kelly 1997), but produced fewer allergic reactions in the patients. In the mid 1950s, Sola, first with Kuitert (Sola & Kuitert 1955) and later with Williams (Sola & Williams 1956), investigated and confirmed the efficacy of alleviating TrP pain by injecting saline into the TrP and still later, Frost's group (Frost, Jessen & Siggaard-Andersen 1980) compared the effects of injecting saline with that of a long-lasting analgesic (mepivacaine). Seventy six percent of the saline recipients had pain relief compared with 57% of the local anaesthetic group, reaffirming Travell's original hypothesis that it was the effect of the needle, as opposed to the substance injected that relieved TrP pain, a finding that has led clinicians to adopt the use of acupuncture needles to 'dry needle' TrPs. To clarify the various needling techniques, Baldry (2002) categorized them as: "*deeply applied techniques*" all involving the direct injection of TrPs either

with some substance (corticosteroids, a non-steroidal anti-inflammatory drugs, local anaesthetics, botulinum A toxin or vitamin B12) or with needle insertion alone (so-called deep dry needling - DDN) and “*superficially applied techniques*” involving needle insertion into the skin and subcutaneous tissues over TrPs with the delivery of an injectable or simply needle insertion alone (superficial dry needling - SDN)

More recently, in a systematic review of the available needling therapies in the management of TrP pain, Cummings and White (2001) confirmed that “wet needling” (injecting drugs) was not therapeutically superior to dry needling of TrPs. The review, which covered the period until 2001, found that though needling therapies improved various measures associated with TrP pathology, they found no evidence that they had an effect beyond placebo in myofascial pain treatment, predominantly due to the fact that a valid placebo needle or needling technique had not been developed. For example, in a randomised, double blind trial, low back strain subjects who received dry needling treatment of TrPs reported it was effective in subjectively reducing TrP pain, though the difference was not statistically significant nor compared to a placebo needling intervention (Garvey, Marks & Weisel 1989). More recently, it has been suggested that needling therapies would be more effective if a LTR is obtained during the treatment (Hong, C. Z. 1994b; Shah, J P 2003).

In summary, it would appear that TrP needling (both DDN and SDN) is an effective treatment given that all studies where TrPs were needled produced

marked, though not placebo controlled, improvement of symptoms (Cummings & White 2001) with the only adverse affect appearing to be that dry needling produced more post-injection soreness when compared to lidocaine (Hong, C. Z. 1994b). The other significant outcome of Cummings and White's work (2001) was to provide impetus for the development of a 'placebo needle' to allow researchers to test the efficacy of any needling techniques beyond placebo (Cummings & White 2001). In this vein, researchers over recent times have endeavoured to develop, validate and employ what might be called sham acupuncture needles in various ways and with various degrees of success. Attempts have ranged from home-made options such as cutting the needle tips off and using a blunt, non-penetrating needle (Huguenin, L. et al. 2005) through to purpose built products (eg: the "Park Sham Device") and it now appears more likely that a sham acupuncture needle provides a valid placebo in acupuncture-naive subjects (Park et al. 2002) as opposed to acupuncture aware subjects (Park et al. 2002; White et al. 2003).

(a) Deep Dry Needling (DDN) of TrPs.

Deep dry needling (DDN) of TrPs involves rapidly inserting a needle, usually an acupuncture needle, into the centre of a TrP in order to elicit one or multiple local twitch responses (LTRs) (see pages 19-21 and 28-31) (Borg-Stein & Simons 2002). The change in the length of the fibres caused by the LTR is thought to stimulate mechanoreceptors, with large diameter, fast-conducting fibres. Many authors in the myofascial needling field (Chu 1997,

2002; Chu & Schwartz 2002; Hong, C. Z. 1994b, 1994a) have postulated that the input from the large diameter sensory afferents blocks the intra-dorsal horn passage of smaller, slower conducting input generated in the TrP nociceptors, resulting in at least short-term alleviation of TrP pain. A number of authors (Chu 2002; Shah, J P 2003) have suggested that the greater the amplitude of the LTR, the greater the pain relief afforded by the needling treatment, opinions based upon clinical observation but yet to be confirmed by experimental evidence. Notwithstanding this deficiency, this postulate has provided a theory to account for the therapeutic relief associated with the LTR and the belief that LTRs may be the key to pain relief, rather than just a diagnostic sign for the localisation of TrPs. For this reason, Hong (1994) and Chu (2002) strongly supported obtaining multiple LTRs in treating TrPs, believing that doing so (by rapidly re-inserting the needle into the TrP region), increased the effectiveness of DDN. However this must be tempered by the fact that in two early studies (Hong, C. Z. 1994b; Hong, C. Z. & Torigoe 1994), the authors found that though both DDN and injection with lidocaine (0.5%) reduced subjective pain, increased the pressure-pain threshold (PPT) of TrPs as well as improved cervical ROM, all subjects who received DDN had significantly more intense post-needling soreness which persisted for longer than pain associated with lidocaine injection. Furthermore, the techniques used to elicit LTRs apparently cause immediate sensory responses, ranging from minor, transient discomfort to considerable initial sharp pain that lingers as a dull ache for up to 48 hours (Hong, C. Z. 1994b). Finally, Baldry (2002) suggested that DDN and in particular, the elicitation of a LTR was liable to cause damage to neighbouring blood vessels and

nerves, and for this reason has been a strong advocate for using superficially applied needling techniques as a general rule, except in the case where there is significant muscle spasm secondary to radiculopathy.

(b) Superficial dry needling (SDN) over TrPs

The mechanism by which SDN produces favourable effects on TrP pain appears to be related to afferent responses caused by stimulation of A-delta nerve fibers from cutaneous receptors secondary to needle insertion into the skin and subcutaneous tissues over a TrP. This is thought to lead to release of opioid peptides in the dorsal horn, which inhibit the intra-dorsal horn transmission of nociceptive information arriving at the spinal cord from group IV afferents from the TrP (Baldry, PE. 2005). Pomeranz (2001) confirmed that needle induced analgesia was opioid peptide-based by administering a known endorphin antagonist, which cancelled the effect of SDN (Pomeranz 2001).

In addition to the mechanical stimulation of A-delta nerve fibers in the skin and subcutaneous tissues, it has been suggested that SDN establishes a low intensity galvanic current as a result of the difference in the electrical potential between the needle and the skin that persists for up to 72 hours after needle removal (Baldry, PE. 2005). If this be true then, SDN may act by briefly stimulating the A-delta fibres mechanically, but also by producing an electric current that might produce these pain-reducing afferent effects for extended periods, concepts that require further investigation.

(c) *Summary of the known effects produced by dry needling of TrPs*

A number of studies have investigated the effects produced by needling TrPs and have found various effects including reduction of the endplate noise (EPN) associated with TrPs, improved local muscle and skin blood flow, reduced TrP pain sensitivity and improved neck range of motion, as outlined in the following paragraph.

Chen and colleagues (2001) investigated the effects of obtaining multiple LTRs in rabbit TrPs in one biceps femoris, using deep dry needling in the opposite side 'normal' muscle as control and found a significant decrease in the EPN due to their treatment (Chen, J. T. et al. 2001). With regard to TrP sensitivity, in a study involving 40 patients with musculoskeletal pain, Edwards and Knowles (2003) found that the pressure-pain threshold (PPT) of TrPs was significantly improved after a three-week follow up for patients who received SDN with active stretching compared with patients who did stretching alone or had no treatment (Edwards & Knowles 2003). In a recent study, Sandberg and colleagues (2005) investigated patterns of blood flow response in the trapezius following needle stimulation (both DDN and SDN) in 19 healthy subjects, 20 patients with fibromyalgia and seven with work-related trapezius myalgia. Where DDN was employed, skin and upper trapezius blood flow was significantly improved in healthy subjects compared with SDN, whereas both needling methods produced a comparable improvement of blood flow in the trapezius and its overlying skin in the fibromyalgia patients. However, in myalgia patients, there were no

differences between needling techniques and generally a reduced blood flow response compared with the fibromyalgia patient group. In addition, positive correlations were found between increased blood flow (both muscular and skin) and increased PPT, cervical ROM and pain experienced due to the stimulation of the needling during the trial. In other words, in terms of pain reduction post-treatment, those who experienced most discomfort during needle stimulation (movement of the needle, possibly eliciting LTRs though not reported) experienced greater treatment responses, suggesting that the intensity of needle stimulation, that is the amount of needle movement, should be taken into consideration when applying dry needling techniques in order to increase the muscle blood flow in chronic pain conditions. Importantly, patients who had greater pain levels pre-needling had less favourable treatment results, pointing to the importance of nociceptor activation as a limiting factor when attempting to increase local blood flow for a therapeutic response (Sandberg et al. 2005). Finally with regard to the effects of TrP dry needling treatment on range of motion (ROM), the literature provides mixed results. Some authors have found an improvement in ROM (Sandberg et al. 2005) after TrPs have been needled either deeply or superficially, while others (Huguenin, L. et al. 2005) have found no significant effects post needling. In this vein, Irnich and colleagues (2002) found that dry needling had no effect on 'motion related pain', suggesting that where movement related pain was the main symptom, dry needling to treat TrPs would be unlikely to improve the condition (Irnich et al. 2002). These contradictory findings suggest that changes in ROM are not reliable indicators of the effectiveness of dry needling for TrP treatment.

In summary, DDN is probably useful under the following conditions or situations:

- To increase blood flow to the muscle and overlying skin in healthy (no pain) subjects (Sandberg et al. 2005), possibly to enhance blood flow to certain muscles prior to sporting endeavour or promote continued recovery from an old injury.
- To elicit LTRs from the TrP region (Hong, C. Z. 1994b).
- Where significant muscle spasm exists secondary to another problem (eg: radiculopathy) (Baldry, P. 2002b).
- Where a patient is a weak reactor to needling therapies and requires a stronger stimulus to elicit a response (Baldry, P. 2002a).
- Where the client and practitioner are both experienced with this more aggressive technique and are not affected by fear (Baldry, P. 2002b; Simons, D., Travell & Simons 1999).

The less invasive SDN approach on the other hand, may be indicated:

- Where the TrP pain is not chronic or is not rated as severe (Sandberg et al. 2005).
- Where other conditions affecting neurophysiology, such as fibromyalgia co-exist (Sandberg et al. 2005).
- For clients who are strong negative reactors to needling therapies and require a more gentle stimulus or where the reaction of the patient is unknown (Baldry, P. 2002b).

- Where the TrP is situated close to important anatomical structures (eg: in the anterior scalene muscles in relationship to the phrenic nerve or subclavian vessels) (Baldry, P. 2002a).

(d) *Relative contra-indications to consider*

The following list summarises what can be considered the main contraindications to the application of DDN or SDN.

- If the patient is undergoing anticoagulation therapy.
- If the patient has taken aspirin within three days of injection.
- If the patient has a needle phobia.

2.1.10.7 TrP injections and Other TrP Therapies

Myofascial TrP injections, including analgesics, botulinum toxin A, anti-inflammatory agents and occasionally vitamin B₁₂ are commonly used by physicians to manage myofascial pain syndromes, as are a broad ranges of electrotherapeutic modalities. Considering the nature of the current work, these techniques were only of peripheral interest and therefore, only a brief outline of the topic has been included in Appendix F (page 289) for the interested reader.

2.1.11 LTrPs: Symptoms, effects and relationships to ATrPs

As was discussed earlier (page 23), based upon a synthesis of the relevant literature, his own research and many years of clinical experience, Simons (2005), concluded that there were fundamental differences between the effects produced by the two basic types of TrP (active and latent). He suggested that ATrPs usually augment sensory phenomena, (manifested as referred pain and tenderness), but at times inhibit them (manifested as referred anaesthesia). Latent TrPs in contrast, were said to commonly amplify or inhibit motor functions of the “parent” muscle and possibly refer these changes in motor behaviour to functionally related muscles. Furthermore, both clinical (Sciotti et al. 2001; Simons, D., Travell & Simons 1999) and biochemical (Shah, J. P. et al. 2005) differences have been found when comparing ATrPs and LTrPs, providing support for the notion that they are quite different entities. These differences notwithstanding, most TrP authorities (Gerwin, R. D. 2005; Hong, C. Z. & Simons 1998; Huguenin, LK 2004; Simons, D. G. 2004b) still appear to consider the LTrP a clinical forerunner of the ATrP. In this vein Simons (2005) felt that the motor effects of LTrPs could profoundly influence muscle coordination which could promote their conversion to ATrPs. More controversially, Hong (2004) suggested that LTrPs might be found in many pain-free skeletal muscles, where they could be “activated” (becoming an ATrPs) by continued noxious stimuli (for example, overload caused by prolonged or repetitive contractions of the muscle). Furthermore, Hong considered that although ATrPs could be “inactivated” (no longer spontaneously painful) through treatment, he felt that

they never fully disappeared but rather converted to latent form. These views prompt a number of questions, particularly given Hong's belief in the ubiquity of LTrPs:

1. How prevalent are LTrPs?
2. What are the deleterious effects on motor function of LTrPs?
3. Do currently recommend treatments de-activate LTrPs ?
4. Are there any positive effects on motor function produced by de-activating LTrPs?

Much of the present work was directed at answering these questions through an investigation of the prevalence of LTrPs in a sample of normal healthy adults (Chapter 3) and investigations of their effects on scapular muscle activation patterns (MAPs) during a common upper extremity motion, (elevation of the arm in the scapular plane) (Chapters 4 and 5) Given the latter, it is important to review the fundamental processes involved in generating scapular plane elevation of the arm to set the stage for the work described in subsequent chapters. First is presented a description of the normal movement and this is followed by discussions of some of the common overuse conditions that may predispose to or result from LTrPs in the scapular muscles

Section 2: Elevation of the Arm in the Scapular Plane

The arm moves in a combination of three planes of motion about three separate axes, allowing great mobility (Downar & Sauers 2005). When elevating the arm (combining abduction at the glenohumeral joint and predominately upward rotation of the scapula), the most common plane of motion during functional tasks, such as those used in daily living and athletic performance, is the scapular plane, defined as lying 30-40° anterior to the coronal plane (Borsa, Timmons & Sauers 2003; Kibler, W. B. 1998a). This movement requires that the contributing motions of the respective bones and joints occur in an optimal sequence so that appropriate loads are apportioned to the contributing tissues in a manner that does not result in functional overload and subsequent injury (Price et al. 2000). The smooth integrated movements of the humerus, scapula and clavicle necessary to elevate the arm were first referred to as “scapulohumeral rhythm” by Codman in 1934 (Codman 1934). A description of scapular plane elevation, including proximal humerus and scapular kinematics, the muscle activity that produces them and the resulting scapulohumeral rhythm, is outlined in the following sections.

2.2.1 Kinematics of the humeral head during elevation of the arm in the scapular plane

The glenohumeral joint possesses six degrees of freedom including three rotations and three translations (Michener, McClure & Karduna 2003). During scapular plane elevation, of particular interest is the relationship maintained

between the humeral head and the glenoid fossa and how it affects the subacromial space, defined as the area between the superior surface of the humeral head and the inferior surface of the coracoacromial arch (acromion, coracoacromial ligament and acromioclavicular (AC) joint) (Neer 1972). In healthy shoulders, the subacromial space with the arm in the anatomical position, has a vertical diameter of between 1 and 1.5cm (Flatow et al. 1994) which decreases slightly during arm elevation movements in healthy shoulders. The latter results in an increase in the contact between the inferior surface of the acromion and the underlying subacromial structures which include the subacromial bursa and the tendons of the long head of the biceps and supraspinatus (Brossmann et al. 1996). The humeral head translates 1-3mm superiorly during the first 30-60° of active elevation in the scapular plane and then remains close to the centre of the glenoid cavity ($\pm 1^\circ$ superior/inferior translation) as the movement continues (Ludewig & Cook 2002). Presumably, via its attachment to the anterior superior aspect of the glenoid labrum, activation of the long head of the biceps decreases both the superior (Pradhan et al. 2000) and anterior translation of the humeral head (Kumar, Satku & Balasubramaniam 1989) as well as reducing the pressure in the subacromial space (Payne et al. 1997), the former helping to maintain the stability of the humeral head both superiorly and anteriorly. However, given the negligible contribution that the Biceps makes to shoulder abduction, particularly with the elbow extended, the importance of these functions in scapular plane elevation is questionable. Even more effective as stabilisers of the humeral head are the teres minor, infraspinatus and subscapularis whose contractions produce vectors that limit the superior translation of the

humeral head generated by the deltoid during early arm elevation. In addition, these muscles generate horizontal forces that compress the humeral head against the glenoid fossa adding further stability (Neumann 2002) According to Flatow and colleagues (1994), the greatest contact between the tendons of the supraspinatus and biceps with the coracoacromial arch occurs in the mid-range of arm elevation (Brossmann et al. 1996; Flatow et al. 1994). Given that the scapula and humerus are both moving while attempting to preserve the functionally important relationship of the humeral head to the glenoid fossa and subacromial space, the position of the scapula and its motions about the thoracic cage, are critical for normal motion and are discussed next.

2.2.2 Scapular kinematics during elevation of the arm in the scapular plane

According to a biomechanical study by Bagg and Forrest (1988) the scapula rotates around a migrating centre of rotation, or instantaneous centre of rotation (ICR), during upward rotation as follows: initially (arm at 0° in the fundamental standing position), the ICR is located at or near the root of the scapular spine. Then as arm elevation in the scapular plane progresses, the ICR begins to migrate toward the region of the AC joint. There is apparently considerable variability of the point in the range of elevation that the scapular ICR begins to move laterally, reportedly anywhere between 60° and 90° of elevation, however, it has been shown that the ICR reaches the AC joint

somewhere between 120-150° of elevation. During the middle phase of elevation, where the ICR is located somewhere in the upper central scapular area, a variable amount of clavicular elevation occurs about the sternoclavicular (SC) joint, coupled with scapular rotation around the AC joint. This concurrent motion is purportedly responsible for the ICR's gradual shift toward the AC joint as arm elevation in the scapular plane continues. Finally, as arm elevation passes 150°, clavicular elevation ceases and the scapular ICR remains at the AC joint (Bagg & Forrest 1988). In summary, these authors suggested that three phases of scapular upward rotation could be described: an initial phase (from 0° until the middle phase began) where the scapular ICR was near the root of the scapular spine, a middle phase (commencing somewhere between 60° and 90° of elevation), where the ICR migrated from the root of the spine toward the AC joint and a final phase (150°-180°), where the ICR remained at the AC joint. Variability in the patterns of electrical activity recorded from the scapular rotator muscles during these movements (Bagg & Forrest 1986), and in the position of the scapular ICR lead Bagg and Forrest (1998) to conclude that there was more than one mechanically efficient strategy for coupling scapula and humeral motion during elevation of the arm in the scapular plane.

The scapula moves on the thoracic cage at what is sometimes referred to as the scapulothoracic articulation, a physiological rather than anatomical joint. In fact, all motions of the scapula occur through combined motions at the SC and AC joints and can be defined by the resultant scapular movements. These motions are listed in most anatomy texts as upward and downward

rotation, protraction (abduction) and retraction (adduction) and elevation and depression with upward rotation the main action occurring during elevation of the arm (Bagg & Forrest 1986). A more detailed description of scapular upward rotation based upon a three-dimensional analysis was provided by van der Helm and Pronk (1995). They described scapular upward rotation as occurring about an anterior-posterior axis (inferior angle moving laterally) accompanied by external rotation (superior-inferior axis, lateral border moving posteriorly); and posterior tilt (medial-lateral axis, inferior angle moving anteriorly). In summary, according to many studies, the scapula demonstrates a pattern of predominantly upward rotation, with lesser degrees of external rotation and posterior tilting during elevation of the arm (Karduna et al. 2001; Ludewig & Cook 2000; Ludewig, Cook & Nawoczenski 1996; McQuade, Hwa Wei & Smidt 1995; van der Helm & Pronk 1995).

In addition to upwardly rotating, the scapula translates upon the rib cage, produced by clavicular rotations about the SC joint, that is, clavicular elevation/depression (superior/inferior rotation) and clavicular protraction/retraction (anterior/posterior rotation). Measurement of the motion of the clavicle at the SC joint provides an opportunity to estimate scapular translations because of the interposed rigid clavicle that joins the scapula at the AC joint (Karduna et al. 2001; McClure et al. 2001). During elevation of the arm in the scapular plane, the clavicle retracts and elevates, causing relative translations of the scapula in superior and posterior directions (McClure et al. 2001; van der Helm & Pronk 1995). In any case, the clavicle, scapula and humerus and the joints they form are positioned and moved by

the muscles that attach them to the ribs, vertebrae and each other (Kibler, W. B. 1998b). An outline of the activity of the scapular positioning muscles and the glenohumeral stabilising muscles during scapular plane elevation forms the section below.

2.2.3 Muscle activation during scapular plane elevation

The muscles responsible for upwardly rotating the scapula include all parts of the trapezius and the lower part of the serratus anterior (Moore 1992). In the early stages of scapular plane elevation when the scapular ICR is located near the root of the spine of the scapula, Bagg and Forrest (1986), using EMG techniques, found that the upper trapezius and lower serratus anterior were strongly activated, probably in accordance with their relatively large moment arms (compared to the middle and lower parts of trapezius) in this phase of the movement. Interestingly, the same authors found that in cases where the ICR shifted laterally at a relatively early stage of arm movement, the mechanical advantage of the lower trapezius increased earlier in the movement, though the upper trapezius remained in a significantly more mechanically favourable position (Bagg & Forrest 1988). The most common pattern in their 1986 study was a gradual increase in electrical activity of the upper trapezius and lower serratus anterior in the early range of elevation, with the lower trapezius remaining relatively quiet until the arm approached the 90° range (Bagg & Forrest 1986).

According to earlier work (Doody, Freedman & Waterland 1970), and later supported by Bagg and Forrest (1988), scapular upward rotation contributes most to elevation of the arm in the middle phase of the movement (average range, 82° to 139°) when the scapular ICR is migrating away from the root of the scapular spine toward the AC joint. During this phase of elevation, the greater tubercle of the humerus can closely approximate the inferior surface of the acromion, particularly in the presence of insufficient activation of external rotator muscles, which necessitates that the acromion continue to be elevated (achieved by upward rotation of the scapula) in order to preserve the subacromial space (Neumann 2002). Another feature of this phase of elevation is the better mechanical advantage enjoyed by upper trapezius and lower serratus anterior for upward rotation (Neumann 2002) compared with the glenohumeral abductors (deltoid and supraspinatus), plus an improving lower trapezius moment arm (Bagg and Forrest, 1988). However, above 90° of elevation, a rapid increase in the activity of the lower trapezius occurs, which was thought to be related to a corresponding reduction in the rate of increase of electrical activity in both the upper trapezius and lower serratus anterior towards the end of the middle phase of elevation (Bagg & Forrest 1986). A possible explanation for the increasing EMG activity, may lie in the increased activation of these muscles to compensate for their worsening length-tension relationships as they continue to shorten (Neumann 2002). However, in any consideration of the changing activity of the muscles, during upward rotation, the need to accommodate for the changing resistance torque of the upper extremity (increasing to 90° and decreasing beyond the horizontal) is an obvious factor.

The third and final phase of scapular plane elevation, defined by a major decrease in the scapular contribution to arm elevation, was said to begin on average at 139° of elevation (Bagg & Forrest 1986). In this position the force generating capability of the upper trapezius is greatly diminished because the scapular ICR approximates the AC joint resulting in both a minimum moment arm and unfavourable length-tension relationship. Bagg and Forrest (1986), suggested that in this phase, the upper trapezius becomes a supporter of the shoulder girdle, opposing downwardly acting forces produced by the weight of the upper extremity and any loads held in the hand. Conversely, the lower trapezius and lower serratus anterior retain good mechanical advantage for preserving upward rotation of the scapula (Bagg & Forrest 1986). Importantly, as mentioned earlier (page 34), Bagg and Forrest (1988) demonstrated that slight differences in the EMG patterns of muscle activation and ICR locations could produce the same ratio of scapulohumeral rhythm, indicating that there must be more than one mechanically efficient strategy for coupling scapula and humeral motion during elevation of the arm in the scapular plane (Bagg & Forrest 1988).

According to Michener and co-workers (2003), an important role of the scapular musculature is to stabilise the scapula in order to maintain the position of the glenoid fossa (Ludewig & Cook 2000; McQuade, Dawson & Smidt 1998; Pascoal et al. 2000). By acting to dynamically stabilise the scapula and therefore dynamically position the glenoid fossa during elevation of the arm in the scapular plane, the upward rotators of the scapula provide

for optimal kinematics of the humeral head, a role primarily performed by the rotator cuff muscles (Kibler, W. B. 1998b).

The rotator cuff muscles, supraspinatus, infraspinatus, teres minor and subscapularis, apart from their rotary actions, act to maintain the congruence between the humeral head and the glenoid fossa by producing a compressive force during glenohumeral movements (Michener, McClure & Karduna 2003). In addition, these muscles impart an inferior translatory force to the head of the humerus which serves to depress it, thereby countering an upward vector produced by the deltoid (particularly during the early phase of the movement (Thompson et al. 1996)) and critically, helping to preserve the subacromial space. In addition, these muscles form part of a force couple with the deltoid, assisting glenohumeral abduction (Neumann 2002). Some authorities (Halder et al. 2001), also attribute depression of the humeral head during elevation of the arm to latissimus dorsi and teres major, though it seems unlikely that these muscles would be strongly activated during elevation of the arm since both are prime adductors of the glenohumeral joint. Perhaps it is as passive restraints that they perform this function. The roles of the rotator cuff muscles are evidenced by the well documented observation of dysfunctional or weak rotator cuff musculature in patients with subacromial impingement (Baltaci 2003; Blevins 1997; Burke, Vangsness & Powers 2002; Corso 1995; Powers et al. 1994), a condition in which their function of providing a smooth trajectory for the humerus during all phases of arm elevation is compromised (Alpert et al. 2000; McMahon et al. 1995), a phenomenon which most likely existed prior to the development of the

shoulder impingement. Furthermore, Payne and colleagues (1997) found that decreased activity in the rotator cuff during elevation of the arm, required the deltoid to increase its contribution for the movement to occur, an adaptation that they proposed could pose problems for maintaining the subacromial space. This notion has been supported by both an artificially induced disruption in the force couple between deltoid and supraspinatus (Chen, S. K. et al. 1999; Deutsch et al. 1996) and in a naturally occurring state of rotator cuff dysfunction (degeneration or muscle tears) (Deutsch et al. 1996; Yamaguchi et al. 2000). Under both conditions, an increase in superior translation of the humeral head occurred. In addition to alterations to humeral head kinematics, Michener and colleagues (2003) suggested that weakness or dysfunction of the rotator cuff could also lead to changes in optimal scapular kinematics, predisposing to compression of the structures of the subacromial space. Such inefficient or disorganised scapular kinematics have been referred to by clinicians as scapular “dyskinesis” (Kibler, W. B. & McMullen 2003), the clinical relevance, causes and effects of which will be outlined after a discussion of the upper extremity kinetic chain and how it contributes to scapular plane elevation.

2.2.4 The kinetic chain of the upper extremity

To complete the discussion of aspects that contribute to the normal functioning of the upper extremity during elevation of the arm in the scapular plane, Kibler’s (1998) “kinetic chain” concept is discussed (Kibler, W. B.

1998b). He described human motion in terms of a series of segments that link to form kinetic chains acting to transfer forces around the body. In this vein, one of the roles of the scapula and the muscles that position it, is to transfer force and kinetic energy developed by the muscles of the lower limbs, trunk, and shoulder girdle to the upper limb as a whole (Kibler, W. B. 1998b). Logically, a deficiency of function in any segment comprising a kinetic chain, could compel changes in the function of linked systems both “upstream” and “downstream” of the original site in an attempt to preserve normal movement. Kibler (1998) suggested that such adaptations could predispose to dysfunction and pain if they changed the loading patterns in any segment sufficiently. In this model then, tissues that alter their normal function are at risk of compensatory overload and treatment of any resulting overuse injury would necessitate addressing the original, sometimes asymptomatic (for example LTrPs), deficiency. In accordance with Kibler’s hypothesis, minor dysfunctions such as muscle weakness or fatigue or altered timing of muscle activation in the scapular positioning muscles, could initiate a process of compensatory adaptation in the next functional segment of the kinetic chain of the upper extremity, involving the glenohumeral joint itself or the rotator cuff muscles that stabilise it. Theoretically, should this process continue the muscles and joints of the forearm and hand could also become involved. Since the focus of the current work was the segment of the kinetic chain that links the torso to the shoulder girdle, namely the scapula and the muscles that position it, the discussion will now turn to dysfunction of the upper extremity chain and associated pathologies, beginning with a description of scapular dyskinesis, its relevance, causes and effects.

2.2.5 Scapular dyskinesia

Scapular dyskinesia is an alteration in the normal position or motion of the scapula that is manifested during coupled scapulohumeral movements (Burkhart, S. S., Morgan & Kibler 2003b). It may occur subsequent to a shoulder girdle injury, but can also result from altered muscle activation patterns secondary to muscle imbalance or tight or weak muscles and frequently associated with inappropriate inhibition in the scapular positioning muscles (Kibler, W. B. & McMullen 2003). Such altered activation patterns can increase the functional deficit associated with shoulder injury by changing the normal motions of the scapula (Kibler, W.B. 2006). Dyskinesia, usually results from a loss of coordinated upward scapular rotation (usually due to muscle weakness, tightness or altered motor control) and the translation associated with scapular retraction, the latter resulting in a rounded shoulder posture (Sevinsky 2006). Sevinsky (2006) went on to explain that once the condition has developed, it is characterised by altered timing and range of upward scapular rotation, excessive anterior tilting of the glenoid, and reduced strength (due to deficits in the ability to activate the rotator cuff muscles fully). As a result of the altered scapular kinematics and posture, scapular dyskinesia is accompanied by altered length-tension relationships of the muscles attached to the scapula, particularly those of the rotator cuff muscles (Liu et al. 1997; Otis et al. 1994), suggesting that a dysfunctional rotator cuff could result from or cause dyskinesia of the scapula. Though its relationship with shoulder dysfunction appears to be non-specific, that is no specific patterns of dyskinesia have been associated with

any specific shoulder ailment (Burkhart, S. S., Morgan & Kibler 2003a), treatment of scapular dyskinesis is currently directed at managing underlying causes, for example pain or inflammation, and re-establishing normal scapular MAPs, usually by rehabilitation programs that restore the function of the kinetic chain (Kibler, W. B., Livingston & Chandler 1997; Kibler, W. B. & McMullen 2003; Kibler, W. B., McMullen & Uhl 2001). These pathophysiologic and pathomechanical alterations of scapula posture and movement can predispose to conditions such as subacromial impingement, or where shoulder joint injury and pain already exist, can perpetuate or increase dysfunction (Kibler, W.B. 2006).

According to some authors (Kibler, W.B. 2006; Sevinsky 2006), the known causes of scapular dyskinesis include:

- (i) Postural abnormality or anatomical disruption such as increased thoracic kyphosis or AC joint injury or anatomic abnormality.
- (ii) Nerve injury to: the spinal accessory nerve (CNXI) resulting in trapezius weakness; the long thoracic nerve, resulting in serratus anterior weakness; the dorsal scapular nerve resulting in rhomboids weakness.
- (iii) Muscular tightness or capsular stiffness. A shortened pectoralis minor and/or short head of biceps increase anterior tilt of the scapula while a shortened pectoralis major restricts posterior clavicular motion affecting normal scapular motion. Anterior capsular stiffness results in an increased upward scapular rotation component of scapulohumeral rhythm as well as decreased posterior scapular tilt. Posterior capsular

stiffness results in the humeral head being positioned more superiorly and anteriorly, predisposing to impingement of the subacromial structures (Lin, Lim & Yang 2006).

- (iv) Muscle imbalance or weakness. The most commonly weakened or inhibited scapular muscles are the serratus anterior, the middle and lower trapezii and the rhomboids. Inhibition (for example, due to myofascial TrPs or pain) manifests as a decreased ability to exert torque and position the scapula and also as disorganisation of normal MAPs.
- (v) Proprioceptive dysfunction arising from noxious stimuli (due, for example to ischemia or inflammation of a muscle, joint effusion or hemarthrosis) in a muscle or a joint, affects both the source tissues but also functionally related muscles, altering sensory information provided by mechanoreceptors that sense the mechanical deformation in soft tissues. This results in inefficient or uncoordinated muscle group activation.

Broadly speaking, the most likely clinical manifestations associated with scapular dyskinesis are latent and active TrPs (the presence of the latter constituting a myofascial pain syndrome (MPS)) in shoulder girdle muscles, rotator cuff overuse or dysfunction and subacromial impingement syndrome, though the cause and effect relationship between such clinical conditions and scapular dyskinesis has been difficult to elucidate (Burkhart, S. S., Morgan & Kibler 2003a). Given the association of TrPs, muscle imbalance and overload, scapular dyskinesis and shoulder joint pathology, the following section reviews the kinematic and muscular contributions that underpin

shoulder impingement syndrome and incorporates a description of rotator cuff dysfunction and its effects.

2.2.6 Subacromial impingement syndrome

Since the term subacromial impingement was first introduced in 1972 (Neer 1972), it has been acknowledged as the most widely recognised mechanism of chronic shoulder pain (Michener, McClure & Karduna 2003; Yanai, Fuss & Fukunaga 2006), accounting for between 44 and 66% of all complaints of shoulder pain during visits to physicians (Michener, McClure & Karduna 2003). This disorder can present in a variety of forms ranging from inflammation to degeneration of the bursa and tendons in the subacromial space which can ultimately result in a full-thickness tear of these tendons with subsequent degenerative joint disease (Michener, McClure & Karduna 2003), likely due at least in part, to the altered scapular kinematics which have been demonstrated in patients with subacromial impingement (Endo et al. 2001; Ludewig & Cook 2000; Lukasiewicz et al. 1999; Warner et al. 1992). For example, Warner, Micheli and colleagues (Warner et al. 1992). used Moire topography¹. to demonstrate a pattern of increased scapular winging in subjects with subacromial impingement, while three-dimensional kinematic analysis in more recent work has demonstrated decreased posterior tilt (Ludewig & Cook 2000; Lukasiewicz et al. 1999), and decreased upward and external rotation (Ludewig & Cook 2000) during arm elevation in patients with subacromial impingement. Optimal scapular upward and external rotation

¹ A method of 3D morphometry in which contour maps demonstrate symmetry of the body.

and posterior tilting serve to elevate the acromion and increase subacromial space, hence disturbances in these movements predispose to impingement as described below.

An “occupational” example of dyskinesia associated with subacromial impingement syndrome was described by Ludewig and Cook (2000) in 52 overhead construction workers. During elevation of the arm, these subjects showed increased EMG activity in both the upper and lower trapezii while the serratus anterior had decreased activity. These muscle patterns were accompanied by decreased upward rotation, increased anterior tilt and increased internal rotation of the scapula (medial border of the scapular moving posteriorly) compared with controls. While it is not clear whether these deviations from normal cause or are the result of impingement, some authors have stated that to maximise the space for the subacromial structures, trapezius and serratus anterior must function “normally” to upwardly rotate the scapula during arm elevation, particularly in the mid-range (60-150°) where subacromial compression is most likely to occur (Brossmann et al. 1996; Flatow et al. 1994). Furthermore, Deutsch and colleagues (1996) found that active elevation of the arm in impingement sufferers increased superior and anterior humeral head translation by 1-1.5mm and by approximately 3mm respectively. A similar increase in superior translation (1–1.5mm) has also been found in patients with rotator cuff tendon degeneration during both active or simulated arm elevation (Deutsch et al. 1996; Thompson et al. 1996; Yamaguchi et al. 2000). Importantly, from the point of view of the current work, superior translations of

a similar or greater magnitude (1-5mm) have also been demonstrated with weakness or fatigue of the deltoid and rotator cuff in healthy subjects during elevation in the scapular plane (Chen, S. K. et al. 1999; Sharkey & Marder 1995) Though the alterations in the magnitude of humeral head translations mentioned may seem small, the space available under normal conditions is only 10-15mm (Flatow et al. 1994) leaving little room for error. Three common clinical presentations associated with scapular dyskinesis and subacromial impingement: forward shoulder posture, posterior shoulder joint tightness and dysfunctional rotator cuff muscles, will now be briefly summarized.

The most common clinical manifestation of scapular dyskinesis is forward shoulder posture which has been defined as a position of protraction and elevation with internal rotation of the scapula, (often referred to as 'scapular winging' in the clinical setting where the medial border of the scapula does not sit flush against the thoracic cage) in company with medial rotation of the humerus (Neumann 2002). The same author suggested that this posture may be produced by, or result from, a combination of tightness of the pectoralis minor and upper trapezius and weakness of the serratus anterior and middle and lower trapezii, the same muscular imbalance pattern that has been implicated in the development of subacromial impingement (Fu, Harner & Klein 1991). Alterations of the scapular resting posture have also been noted in patients with subacromial impingement involving greater anterior tilt of the scapula (Lukasiewicz et al. 1999) and increased scapular winging (Warner et al. 1992) giving these patients a 'slouched' posture. The coupling of this

scapular position with medial rotation of the humerus brings the greater tubercle closer to the coracoacromial arch, reducing the subacromial space and increasing risk of impingement.

Another functional change that can predispose to scapular dyskinesis and subacromial impingement is tightness or stiffness of the posterior shoulder joint capsule (Sevinsky 2006). According to Michener, McClure and colleagues (2003), posterior shoulder joint tightness can cause changes in humeral head kinematics that lead to subacromial impingement. This opinion was supported by a recent study of six patients with posterior capsular stiffness (although posterior capsular stiffness was difficult to isolate from posterior rotator cuff tightness) who were found to have anterior shifts of the humeral head of 2.2-3.4mm during arm elevation movements (Lin, Lim & Yang 2006). Though these patients were not compared with matched controls, the findings add weight to the notion that tightness of the posterior glenohumeral joint structures alters kinematics thereby contributing to subacromial impingement. With regard to rotator cuff muscle function, increased rotator cuff and deltoid activity (EMG) at 120° of abduction in 10 healthy subjects was associated with increased subacromial space (Graichen et al. 1999) and simulated activation of the same muscles based on the parameters measured in 10 human cadaveric shoulders was shown to decrease subacromial pressure during elevation of the arm (Payne et al. 1997). Conversely, a decrease in EMG activity in the infraspinatus, subscapularis and middle deltoid between 60° and 90° of scapular plane elevation has been reported in patients with subacromial impingement

(Reddy et al. 2000), adding further evidence for the importance of the rotator cuff muscles in maintaining normal glenohumeral joint function. The work cited in the foregoing discussion provides good evidence that a large percentage of patients presenting with subacromial impingement have scapular dyskinesis (Kibler, W.B. 2006) and often other shoulder girdle conditions like rotator cuff tendinopathy, bursitis, joint degeneration (Michener, McClure & Karduna 2003), long head of biceps tendinopathy (Yanai, Fuss & Fukunaga 2006) or myofascial pain (Sevinsky 2006). Although these variables are clearly associated with each other, their concurrent presentation does not allow the determination of a causal relationship. However, they do indicate that alterations in the synergies between the shoulder girdle muscles that produce elevation of the arm in the scapular plane are associated with dysfunction and potentially, pathology. Hence it is important to establish what effects LTrPs (which are pain-free neuromuscular lesions (Simons, D., Travell & Simons 1999) commonly found in these muscles (Chapter 3)) may have on muscle action and the preservation of this common upper extremity movement, during two situations that occur regularly during daily tasks: raising the loaded hand above shoulder level with scapular plane elevation, or performing elevation of the arm when fatigued. A brief review of the published literature on these two conditions comprises the following sections.

2.2.7 Effects of load during scapular plane elevation

No studies were found that investigated the effects of load on the timing of muscle activation in shoulder girdle muscles during elevation of the arm in the scapular plane. However it is known that during concentric muscle contraction, muscles contract at a maximum velocity when the external load is negligible and contraction velocity decreases as the external load is increased until an extreme load results in a contraction velocity of zero with respect to the well known "Force Velocity Relationship" (Neumann 2002). In related research, in a study on 16 asymptomatic shoulders, Alpert and colleagues (2000) measured the degree of muscle activation, as opposed to the timing of muscle activation, of the deltoid and rotator cuff muscles in response to various external loads during scapular plane elevation. The authors found that EMG activity of deltoid, supraspinatus and infraspinatus increased in the 0°-90° range and decreased in the 120°-150° range and that the change in activity with increasing load was greater from 0-25% and from 25-50% of maximum load than it was for the change from 50-75% and 75-90% of maximum load. The peak muscle activity for anterior and middle deltoid and supraspinatus and infraspinatus occurred between 30° and 60° of scapular plane elevation. The EMG activity of the posterior deltoid was less than 20% of maximum for all parts of the range of scapular plane elevation with peak activity occurring between 120° and 150°, which was expected given the investigated movement and position of posterior deltoid. The subscapularis and teres minor were most active between 0° and 90°, but only when the external load was greater than 50% of maximum. These data

suggest that deltoid, supraspinatus and infraspinatus are utilised to a greater extent in the first 90° of elevation and show greater increases of activity when lighter loads (25-50% of maximum) are used whereas increased activity from the subscapularis and teres minor are required when the external loads are higher (greater than 50% of maximum). These results imply that deltoid, supraspinatus and infraspinatus are preferentially recruited in response to initial increases in external load, with the subscapularis and teres minor increasing their contributions when load increases above 50% of maximal.

With regard to scapulohumeral rhythm, two studies (Doody, Freedman & Waterland 1970; Michiels & Grevenstein 1995) employing three dimensional analysis at three different loads found no significant influence on scapulohumeral rhythm. In other words muscle activation strategies were unchanged with load variations. Similarly, two later three dimensional studies found that increasing external loads (0-3kg) (de Groot, van Woensel & van der Helm 1999) and (0-4kg) (Pascoal et al. 2000) had no effect on either clavicular or scapular kinematics during scapular plane elevation. However McQuade and Smidt (1998), reported changes to scapulohumeral rhythm produced by maximum resisted (performed against a dynamometer) arm elevation in the scapular plane, suggesting that high external loads can affect scapulohumeral rhythm. In summary, these studies suggest that external loads of four kilograms or less do not alter scapulohumeral rhythm during scapular plane elevation, but maximum loads might.

2.2.8 Effects of fatigue during scapular plane elevation

According to Neumann (2002), fatigue involves a variety of elements located in central and/or peripheral parts of the neuromuscular system. Central fatigue may be affected by psychological factors, such as perceived effort or physiological factors, such as inhibition of pathways that prevent efficient activation of motor neuron pools. Peripheral fatigue on the other hand may result from neuophysiological factors related to action potential propagation in motor nerves and transmission of activation to muscle fibers, for example repetitive activation of motor units may result in a short-term reduction of ACh release, and the muscle fiber cytoplasm may undergo a variety of biochemical changes that reduce force output over time (Fitts & Metzger 2004).

Fatigue has been found to affect three dimensional scapular kinematics during scapular plane elevation. Tsai, McClure et al. (2003) used a repetitive external rotation task to fatigue 30 healthy subjects then measured three rotations (anterior/posterior tilting, upward/downward rotation and internal/external rotation) of the scapula at six points of humeral elevation. They found that fatigue caused increased anterior tilt of the scapular up to 90° of elevation with the greatest anterior tilt occurring at 4°. Fatigue decreased external rotation from zero to 120° of arm elevation with the greatest degree of internal rotation (where the medial border of the scapula lifts posteriorly off the thoracic cage) occurring at 2.4° of arm elevation after fatigue. Upward rotation was significantly reduced during the first 60° of

humeral elevation after fatigue with the greatest decrease occurring at 2.5°. These findings suggest that fatigue has the greatest impact on scapular position at the beginning of scapular plane elevation. Similarly, three additional studies found a significant decrease in posterior tilt in the early part of scapular plane elevation in response to fatigue (Ebaugh, McClure & Karduna 2006; McQuade, Hwa Wei & Smidt 1995; McQuade & Smidt 1998). In a more recent investigation into the effects of fatigue on scapulothoracic and glenohumeral kinematics (Ebaugh, McClure & Karduna 2006), 20 healthy subjects underwent a fatiguing protocol until they could no longer perform a battery of tasks. Median power frequency (MPF) dropped by at least eight percent in all muscles except the lower trapezius, indicating that the upper trapezius, serratus anterior, anterior and posterior deltoid and the infraspinatus muscles had indeed been fatigued by the protocol employed. Compared with the pre-fatigued state, upward rotation increased at the following angles of elevation: 60° (5.3°), 90° (7.4°), 120° (6.4°) and maximum elevation (2.9°). Scapular external rotation increased at the following angles of elevation: 90° (6.4°), 120° (8.2°) and maximal elevation (5.2°) and finally, clavicular retraction increased at 60° (2.6°), 90° (5.4°), 120° (6.4°) and maximal elevation (3.3°). For humeral motion, subjects demonstrated decreased humeral external rotation when fatigued. These findings suggest that greater scapulothoracic motion and less glenohumeral motion occur during scapular plane elevation following muscle fatigue which the authors speculated may have resulted from increased scapular rotator muscle activation in compensation for fatigue in the deltoid and infraspinatus muscles.

Muscular fatigue has been shown to alter motoneuron firing rates during sustained maximal voluntary contractions (Bigland-Ritchie, B. et al. 1983; Bigland-Ritchie, B. & Woods 1984; Duchateau & Hainaut 1985; Viitasalo & Komi 1981) and has also been shown to affect proprioceptive feedback and cortical control (Macefield et al. 1991; Taylor et al. 1996; Taylor, Butler & Gandevia 2000) For example, shoulder proprioception in active repositioning in external rotation of the arm was significantly altered after a fatiguing protocol using an isokinetic dynamometer to maximally resist internal and external rotation of the shoulder (Lee, H. M. et al. 2003). Furthermore, in a study on segmental posture and movement, where seven healthy adults performed a series of fifteen fast wrist flexions and extensions while being instructed to keep a dominant upper limb posture as constant as possible, it was concluded that there was no clear understanding of the mechanisms by which the CNS adapts to fatigue in order to preserve normal movement patterns (Chabran, Maton & Fourment 2002). One way to gain insight into both peripheral and central output related to movement performance, is to record MAPs which reflect the temporal sequence of muscle recruitment. This approach is discussed in relation to elevating the arm in the scapular plane in the following section of this review.

Section 3: Muscle Activation Patterns (MAPs) in the Shoulder Girdle

The timing and sequence of muscle activation can be investigated by electromyographic recordings of MAPs and this approach has been used in various regions of the body including the lower back (Hodges, P. & Richardson, C. 1999; O'Sullivan, P. et al. 1997), the pelvic floor (Smith, Coppieters & Hodges 2006), the neck (Falla, D., Bilenkij & Jull 2004), the knee (Mellor & Hodges 2005) and the shoulder (Wadsworth & Bullock-Saxton 1997). Focusing on the latter, Wadsworth and Bullock-Saxton (1997) used surface EMG (sEMG) to investigate the temporal sequence of muscle recruitment of the upward scapular rotator muscles (upper and lower trapezius and the lower part of serratus anterior) in nine young, elite swimmers with chronic unilateral shoulder impingement and compared them with nine swimmers with healthy shoulders during elevation of the arm in the scapular plane. In the healthy shoulders, the timing of muscle activation for the upward scapular rotators was as follows: upper trapezius was activated 217ms prior to movement start (the arm leaving the side of the body), serratus anterior was activated 53ms after movement start and the lower trapezius was activated last at 349ms after movement start, which correlated with the arm reaching 15° of elevation. No significant differences in MAPs were found between the injured and non-injured sides of the shoulder impingement group, however when the non-injured side of the impingement group was compared to the control group, serratus anterior was significantly delayed in its time of activation in the healthy shoulders of the impingement subjects ($p < 0.05$). Interestingly, no differences were observed in MAPs for

any muscle between the control group and the injured side of the impingement group. The investigators found that the presence of shoulder impingement syndrome significantly increased the variability (as indicated by the standard deviations of onset times) of the timing of activation of all the upward scapular rotator muscles compared to the control group and within the impingement group, serratus anterior was significantly more variable in its time of activation on the injured side compared to the non-injured side (Wadsworth & Bullock-Saxton 1997). In this scenario, there was both a deficiency of function in a proximal segment of the upper limb (altered activation patterns of the upward scapular rotator muscle group) and a chronic, painful condition of the shoulder joint (shoulder impingement syndrome). If Kibler's theory regarding dysfunction in a proximal segment of a kinetic chain is correct (see page 90), then the initial change would be expected in the recruitment patterns of the upward scapular rotator muscle group, possibly leading to changed biomechanics at the glenohumeral joint. In the Wadsworth and Bullock-Saxton (1997) study, however, there was no opportunity to establish a cause and effect relationship between these two variables since all subjects had been diagnosed with shoulder impingement syndrome prior to the investigation. In this situation, pain arising from impingement might explain the findings. Therefore, in order to establish whether deficiency in the muscles of a proximal segment of the upper limb chain is associated with changed function in a more distal segment, the subjects need to be pain-free at the time of investigation, a situation that exists when LTrPs alone constitute the deficiency. Given the dependence of muscle activation and therefore movement performance, on effective

neuromuscular function, the next section of this discussion will focus on aspects of motor control.

2.3.1 Effect of pain on motor activity and control

Examples of changes in motor activity and control include increased activity in some muscle groups and inhibition or weakness in others and pain avoidance motor patterns such as limping, decreased ranges of motion and loss of spinal curves, can also be observed in clinical settings (Sterling, Jull & Wright 2001). Pain has been associated with motor control deficits in the form of muscle inhibition and altered patterns of muscle recruitment, both of which have been shown to affect joint control and have been found in the lumbar spine (Hodges, P. & Richardson 1996; O'Sullivan, P et al. 1997), the cervical spine (Falla, D 2004) and the knee (Mellor & Hodges 2005; Owings & Grabiner 2002; Voight & Wieder 1991). Loss of joint control may leave the subject vulnerable to further injury or be the cause of ongoing pain or recurrence of injury (O'Sullivan, P et al. 1997). A vicious cycle model was proposed by Johansson and Sojka (1991) to explain altered muscle function and loss of joint control in response to painful and non painful conditions alike. At the heart of their proposal was an increase in muscle tension or spasm produced by increased gamma motor neuron (γ -motoneuron) discharge in response to input from receptors in joints, muscles and skin (Johansson & Sojka 1991). Though the influence (if any) of the gamma system in altered joint control remains uncertain because of continuing

debate, especially with respect to afferent input from muscle (Sterling, Jull & Wright 2001), a brief description of the vicious cycle model is now provided in point form

- Stimulation of group III and IV muscle afferents by algogenic substances produced by “distressed” muscle fibers, sensitised nociceptors or from other structures (e.g. joints) reflexively excite both dynamic and static γ -motoneurons (Appelberg et al. 1983). These in turn enhance the activity of primary and secondary muscle spindle afferents that ultimately determine muscle stiffness by the following mechanism.
- Increased activity in primary and secondary muscle spindle afferent input increases excitability in alpha (α) and γ motoneurons projecting back to the muscle increasing both stiffness and metabolite production secondary to the increased muscular contraction, which continues the cycle and leads to further stiffness. (Johansson & Sojka 1991).

In support of this theory, animal studies have shown enhanced ipsilateral activity in primary and secondary spindle afferents after application of chemical mediators such as potassium chloride, lactic acid, bradykinin and serotonin to muscle tissue (Djupsjobacka, Johansson & Bergenheim 1994; Djupsjobacka, M et al. 1995; Djupsjobacka, M. et al. 1995) as well as modulation of secondary spindle afferents after injection of bradykinin into the contralateral muscle (Djupsjobacka, M et al. 1995). Similarly, bradykinin injection of the trapezius and splenius muscles of cats produced excitatory effects in the γ -motoneurons manifested as increased static stretch sensitivity of muscle spindles in both contralateral and ipsilateral muscles (Pedersen et

al. 1997). In addition to chemical stimulation of muscle sensory afferents, it has been suggested that input from the articular afferents of inflamed joints may also increase activity in the γ -efferent system, thereby amplifying any effects of a vicious cycle model (Johansson & Sojka 1991). These findings and ideas notwithstanding, the validity of the vicious cycle model has not yet been established because no increase in α -motoneuron activity has been shown in any relevant study (Graven-Nielsen, Svensson & Arendt-Nielsen 1997; Stohler, Zhang & Lund 1996). In addition, the model does not account for all situations in which muscle tension or spasm exist such as occurs in conjunction with myofascial TrPs. In this case, muscle acidity secondary to inflammation produced by injury or exercise-induced muscle overload (Gerwin, RD, Dommerholt & Shah 2004) is believed to initiate neurophysiological activities (increased CGRP in synaptic cleft, decreased AChE and increased AChRs on the post-synaptic membrane) that results in an increase of acetylcholine-mediated miniature high frequency endplate potentials that act to cause a sustained partial depolarisation of the muscle cell membrane. The explanation of how sarcomere contracture occurs within sarcomeres near the myoneural junction may involve an associated depolarisation of the T tubule leading to Ca^{2+} release from the SR. These events occur in the absence of motor nerve activation of the post-synaptic muscle membrane (Gerwin, RD, Dommerholt & Shah 2004). The TrP makes the muscle feel tense but is not associated with propagated action potentials that would be identified as EMG activity. Simons and Mense (1998) also highlighted the possibility that increased muscle tension may result from

alteration of the visco-elastic properties of the muscles (non-electrical) as opposed to contractile (electrical) changes.

In direct opposition to the a vicious cycle explanation, Lund and colleagues (1991), suggested that pain itself, does not cause muscles to become hyperactive, but in fact, reduces the ability to voluntarily contract muscle fibers (Lund et al. 1991). They reported that when pain was experimentally induced in an animal study, EMG activity of the painful agonist muscle decreased but increased in its antagonist, presumably as a protective strategy to limit the range or velocity of movement. It was suggested by others that these changes in motor output resulted from alterations in the firing pattern of segmental interneurons in the spinal cord or brain stem (Westberg et al. 1997). This interaction (between muscle pain and muscle coordination) was termed the "Pain Adaptation Model" (Lund et al. 1991). However, though loss of voluntary muscle contraction may be caused by pain-mediated inhibition, it has also been shown to occur when pain is not present, as when saline is infused into the knee joint (Shakespeare et al. 1985). Stokes and Young (1984) suggested that knee joint swelling produced by saline infusion resulted in quadriceps inhibition derived from joint afferent inhibition of α -motoneurons and that this was also the cause of atrophy over time (Stokes & Young 1984). Based upon his work on the vertebral column, Panjabi (1992) suggested that deterioration in muscle function, resulting from "disuse, degeneration, disease or injury" to the vertebral column could give rise to inaccurate feedback to neuronal control systems, thereby affecting spinal joint control (Panjabi 1992).

A further mechanism to explain how pain affects motor control was put forward by Sterling, Jull and Wright (2001). They suggested that reflexes mediated by pain, altered patterns of neuromuscular activation, delaying the activation of specific muscles or muscle groups thereby disturbing their synergies. This concept finds support in the work of several authors who demonstrated altered MAPs in the presence of pain including the transversus abdominis and multifidus in subjects with low back pain (Hodges, P. W. & Richardson, C. A. 1999), the deep neck flexors in whiplash patients (Jull 2000) and the upward scapular rotators in subjects with shoulder impingement syndrome (Wadsworth & Bullock-Saxton 1997). Importantly, Hides and Richardson (1994) contended that though such changes might be initiated in the acute phase of an injury, they could persist into the period of chronicity. It has also been suggested that inhibition usually occurs in deep muscles of the joint involved and that these muscles act as joint stabilisers. (Hodges, P. & Richardson 1996). Perhaps lending support to this view, altered patterns of neuromuscular activation were found in a study of low back pain patients in whom selective fatigue of lumbar multifidus (lying deep to longissimus and iliocostalis) was detected using EMG, even though the net extensor torque remained unchanged (Hides, Richardson & Jull 1994). Furthermore, patients with spondylolysis or spondylolisthesis generated greater levels of activity in the superficial rectus abdominis (than controls), to stabilise the spine with abdominal straining manoeuvres which was considered by the authors to represent compensation for “loss” of control of the deep abdominal muscles which normally perform this function (O’Sullivan, P et al. 1997). Similarly, O’Sullivan and colleagues (1997),

suggested that the heightened EMG activity in superficial muscles that they observed in 12 chronic low back pain patients performing an abdominal “drawing in” manoeuvre could represent a measurable compensation for loss of segmental spinal support (O'Sullivan, P. et al. 1997). These findings implicate dysfunction of synergistic muscle control as a specific and important consequence of pain and injury (Sterling, Jull & Wright 2001). Furthermore, when experimental muscle pain was induced in 10 healthy subjects by intra-muscular injection of hypertonic saline into the trapezius, the investigators found that shoulder coordination was adversely influenced and a reorganisation of the pattern of muscle recruitment occurred during work related tasks such as cutting (Madeleine et al. 1999), increasing the evidence available that suggests that pain alters patterns of neuromuscular activation.

The foregoing section demonstrates how pain can affect motor control through several mechanisms and in particular, by impinging on patterns of muscle activation. In contrast, Sterling and co-workers (2001) proposed that changes in motor control systems may occur before the onset of pain via some sort of afferent input that does not register consciously as pain. The consequence of this afferent input, for example due to LTrPs, may be to produce various patterns of reflex inhibition in the CNS and adversely affect motor control systems and decrease the effectiveness of movement. This process may potentially predispose to the development of pain in tissues exposed to changed loads as a consequence of the inefficient muscle activation (Sterling, Jull & Wright 2001). This suggestion aligns well with

Kibler's (1998) proposal i.e. that dysfunction in one segment of a kinetic chain, causes ineffective or inefficient activation of muscles, predisposing muscles in related segments to alter their activation patterns in order to preserve normal movement more distally. Because of the clearly demonstrated effects of pain on muscle activation (as cited above), a pain producing entity like an ATrP would be expected to affect motor control in accordance with models (and material) discussed above including the Vicious Cycle, Pain Adaptation and Altered Patterns of Neuromuscular Activation models. However, whether Kibler's proposal, "holds up" in the face of a non-painful lesion capable of producing sensory input, the LTrP, remains to be seen and was tested in the present investigation using the upper extremity kinetic chain operating in a common motor pattern, elevation of the arm in the scapular plane. Accordingly, whether taking a neurophysiological (Sterling, Jull & Wright 2001) or biomechanical (Kibler, W. B. 1998b) standpoint, it seems logical that the effects of lesions that allow pain-free movement, such as LTrPs, in a proximal segment of the upper extremity may produce effects that alter optimal MAPs and therefore movement efficiency and effectiveness of the entire upper limb. Given that decreased movement efficiency exposes 'compensating' tissues to altered functional loads, the end point of this process may be an overuse injury developing in the compensating structures. Such endpoints in the shoulder girdle include inflammatory or degenerative conditions of the rotator cuff, shoulder impingement syndrome (Michener, McClure & Karduna 2003) or ATrPs in overloaded muscles (Simons, D., Travell & Simons 1999), all conditions that

can cause significant disability and can be difficult to treat, making prevention of this process all the more appealing.

2.3.2 Surface EMG in the measurement of muscle activation patterns

Surface Electromyography (sEMG) of selected shoulder girdle muscles was the technique of choice to determine MAPs in the current work and has been employed by many authors (Christensen 1986; Ebaugh, McClure & Karduna 2005; Elert et al. 2000; Gerdle, Edstrom & Rahm 1993; Hagberg 1981; Hermans & Spaepen 1997; Lucas, Polus & Rich 2004; Lundblad, Elert & Gerdle 1998) to investigate these muscles, though some have used indwelling fine wire bipolar electrodes for the infraspinatus muscle (Ballantyne et al. 1993; Kelly et al. 1996). In a closely related study Wadsworth and Bullock-Saxton (1997), used sEMG to measure the “time of onset” of the upward rotators (upper and lower trapezius; lower part of the serratus anterior) of nine young male swimmers with unilateral shoulder impingement syndrome during scapular plane elevation, with their main finding being that the timing of muscle activation was more variable in subjects with the shoulder condition as compared to matched controls (Wadsworth & Bullock-Saxton 1997). This work and others (referenced though not described, above), provide evidence that sEMG is a useful tool for the measurement of muscle activation patterns of the trapezius, serratus anterior, infraspinatus and middle deltoid during elevation of the arm in the scapular plane.

2.3.3 Development of the research hypotheses

Unless being directly compressed, LTrPs are pain-free neuromuscular lesions that are thought to be prevalent and potentially can become activated to become spontaneously painful ATrPs that might ultimately develop into a recalcitrant Myofascial Pain Syndrome associated with pain and disability. Myofascial TrPs, whether active or latent, are most likely to develop in postural muscles that are exposed to prolonged or repetitive activity (Simons, D., Travell & Simons 1999). Postural muscles that can rotate the scapula, including all parts of the trapezius, serratus anterior, rhomboids major and minor, levator scapulae and pectoralis minor, are known to function in optimally positioning the scapula to facilitate effective transference of forces generated in the legs and torso to the upper extremity in order to move the hand and vice versa (Kibler, W. B. 1998b). The upward scapular rotators are responsible for this scapular positioning during arm elevation movements, the most common being elevation in the scapular plane (Michener, McClure & Karduna 2003).

One measure of motor output that affects movement is MAPs where the temporal sequence of muscle activation is measured using electromyography. Pain, one type of sensory afferent input, is known to affect muscle activation, but the effects of LTrPs, which contribute afferent input that is not perceived as pain, on MAPs have not been investigated. Because LTrPs are pain-free with movement, any effects on MAPs found will not be

due to the presence of pain but presumably will occur in response to the sensory afferent input from the LTrP or other structures.

If LTrPs are common in the scapular positioning muscles (chapter three), it would appear appropriate to investigate their effects on the MAP of functionally related shoulder girdle muscles during this common upper extremity movement. The muscles investigated in this study: upper and lower trapezius and lower serratus anterior (upward rotators of the scapula); the infraspinatus (stabilising function on the humeral head and part of the force couple for arm elevation with the scapular upward rotators) and the middle deltoid (abductor of the arm in the scapular plane) have different functional roles. This will allow study of the effects of LTrPs on the MAP of the upward scapular rotators and also to establish whether there is any alteration to functionally related muscles within the upper extremity kinetic chain in accordance with Kibler's proposal (1998). It is clear from the research reviewed that the effects of LTrPs located in the scapular rotator muscles have the potential to produce effects that may adversely affect scapular positioning and movement of the upper extremity and importantly, to predispose an individual to a significant overuse injury of the shoulder. Therefore the following research hypotheses were formulated:

1. LTrPs occur commonly within the scapular positioning muscles in a group of normal males and females (Chapter 3).

2. LTrPs in the scapular rotator muscles alter muscle activation patterns of these and functionally related muscles during elevation of the arm in the plane of the scapula under each of three conditions (Chapter 4):
 - a. Unloaded
 - b. Loaded
 - c. Fatigued
3. A commonly applied LTrP treatment (Superficial Dry Needling) is an effective means of “removing” LTrPs and restoring normal muscle activation patterns altered by their presence (Chapter 5).

CHAPTER 3

THE PREVALENCE OF LATENT TRIGGER POINTS (LTRPS) IN THE SCAPULAR POSITIONING MUSCLES IN HEALTHY SUBJECTS

3.1 Introduction

Myofascial trigger points (TrPs) are the characteristic clinical sign of Myofascial Pain Syndromes (MPS) that cause regional muscular pain (Simons, D., Travell & Simons 1999). There have been no large epidemiological studies specifically examining the prevalence of TrPs (Baldry, PE 2001), although anecdotal evidence from experienced examiners implies that pain caused by TrPs is a very common phenomenon (Huguenin, LK 2004; McCain 1994), particularly after trauma or sustained muscular fatigue. In support of this view, Rashiq and Galer (Rashiq & Galer 1999) found that 70 percent of 41 patients diagnosed with Complex Regional Pain Syndrome had TrPs in the proximal musculature of the upper limb. Other studies have reported TrPs as a source of pain in 50 percent of patients with temporomandibular disorders (Schiffman et al. 1990), 54 percent of patients presenting with head and neck pain (Friction et al. 1985) and 30 percent of patients presenting with pain (unspecified) to a university medical centre (Skootsky, Jaeger & Oye 1989). Although the examination procedures used to identify TrPs were not uniform, making comparisons difficult, these studies lend support to the notion that pain due to TrPs is common in patients with a variety of pain complaints.

There are two main classifications of TrPs: 'Active' and 'Latent'. According to Simons (2004), an Active myofascial trigger point (ATrP) is a nodule of exquisite spot tenderness in a palpable taut band of skeletal muscle that can produce local or characteristic referred pain both spontaneously or when the

ATrP is compressed. Latent myofascial trigger points (LTrPs), on the other hand, are considered to be associated with muscle stiffness but are not painful unless directly compressed and are thought by some (Bonica 1957; Simons, D., Travell & Simons 1999), to be the clinical 'forerunners' of ATrPs and therefore myofascial pain. Experienced clinicians (Simons, D., Travell & Simons 1999) suggest that during manual palpation, ATrPs produce pain (local and often referred), motor dysfunction (muscle weakness, loss of coordination, decreased work tolerance) and autonomic phenomena (abnormal sweating, persistent lacrimation, excessive salivation, pilomotor activity). When stimulated appropriately, usually by 'snapping palpation' (plucking perpendicular to the muscle fiber direction) or by rapidly inserting a needle into the ATrP, a twitch contraction occurs within the fibers of the taut band containing the ATrP or within the fibers of another muscle with a taut band (Simons, D. G. 2004a). This Local Twitch Response (LTR) is a spinal cord reflex (Hong, C. Z. & Yu 1998) and is said to be the most reliable sign that an ATrP has been identified and effectively treated (Hong, C. Z. 1994b).

When strongly stimulated (increased pressure), clinically silent LTrPs can elicit the clinical signs and symptoms listed above for ATrPs, (Hong, C. Z. 1996), although the responses are usually less pronounced (Simons, D. G. 2004a). With regard to the palpation pressure applied during physical examination of LTrPs, most earlier work relied upon the subjective judgment of experienced examiners to employ a pressure that would not cause pain in normal muscle. However, Hong and co-workers (Hong, C. Z. 1996) found that compression of normal muscle tissue near a LTrP produced referred

pain in 23 percent of subjects examined if there was no regulation of the pressure applied. Therefore it may be helpful to quantify the amount of pressure used to identify a LTrP, in order to decrease the likelihood of false-positives. This idea was supported by Lew and colleagues (Lew, Lewis & Story 1997) and Gerwin and co-workers (1997) and later put into practice by Sciotti's group (2001) who used an algometer to measure the pressure-pain threshold (PPT) of LTrPs in their investigation into the clinical precision of LTrP location in the trapezius muscle. In earlier work, Fischer (1987a, 1987b) used pressure algometry to measure the PPT of normal back and shoulder girdle muscles (Fischer, AA 1987b, 1987a). A mathematical algorithm was then employed to calculate the PPT below which a muscle could be considered abnormal. Fischer (1987a) noted that males and females had different PPT's for the same muscles and that PPT's decreased in a cephalad direction. These values were published (Fischer, AA 1987a) and are displayed in Table 3.1.

Some confusion exists in the TrP literature as to which entity has been examined; ATrPs only or all TrPs, including LTrPs. While most investigations have been conducted in 'patient' populations (meaning the subjects definitely had ATrPs and possibly had LTrPs), there are few if any studies that have specifically investigated LTrPs or their relationship to ATrPs. Hong and Simons (Hong, C. Z. & Simons 1998) suggested that the sub-clinical LTrP could become a pain-causing ATrP if the cause of the LTrP was not addressed. If this is true, it follows that identification and treatment of LTrPs will reduce the incidence of myofascial pain. Given this hypothesis, it is

important to determine whether LTrPs are a common phenomenon in the scapular positioning muscles, a muscle group often subjected to postural overload in subjects who spend prolonged periods in inappropriate sitting postures (Simons, D., Travell & Simons 1999) which, according to the same authors, may increase the likelihood of developing LTrPs (Simons, D., Travell & Simons 1999) In addition, given the negative impact of shoulder disorders on workplace productivity (Svendson et al. 2004) and the importance of the scapular positioning muscles in relation to upper limb function (Kibler, W., McMullen & Uhl 2000), this muscle group is often targeted during rehabilitation programs for patients with chronic shoulder pain due to altered motor control (Wadsworth & Bullock-Saxton 1997).

The aim of the present study was to provide data on the prevalence of LTrPs in the scapular positioning muscles in a sample of normal men and women. This work was a prelude to investigations planned to investigate the effects of LTrPs on muscle activation patterns during scapular plane elevation of the arm.

3.2 Subjects and Methods

Upon gaining approval from the RMIT University Human Research Ethics Committee, 154 university staff and students volunteered to be assessed for joint and muscle dysfunction of the upper back, neck and shoulders. Subjects

were excluded if they had less than 160° of arm elevation, had a positive apprehension test (glenohumeral instability), positive upper limb tension test (neurological dysfunction) or significantly increased thoracic kyphosis (judged by clinical observation). Subjects were also excluded if they reported any pain in the back, neck or either upper limb any time in the week prior to the examination. After this assessment, the scapular positioning muscles of the 137 remaining subjects were examined bilaterally for the clinical characteristics of LTrPs. The muscles examined were the pectoralis minor and serratus anterior (examined lying supine), all parts of the trapezius and rhomboids and the levator scapulae (examined lying prone). All examinations were carried out by the same trained and experienced (12 years) Myotherapist using procedures explained by Simons, Travell and Simons (Simons, D., Travell & Simons 1999) and employed by Lew and colleagues (Lew, Lewis & Story 1997) in their reliability study and briefly described as follows: The subject lay on an examination table in a warm and relaxed state with the upper body disrobed. 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 normal muscle fibers are still slack but the fibers of any taut bands are placed under additional tension, rendering them more easily distinguishable. Next, cross-fiber palpation was used to identify any taut bands (Fig. 3.1), using “flat palpation” (trapping the LTrP between the examiner’s fingertips and underlying bone) for all muscles except the upper trapezius, which was examined using “pincer palpation” (trapping the LTrP between the examiner’s thumb and fingers). 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 manually. In the event of an affirmative response, the PPT of the point was measured with an algometer (Activator Methods Inc., Phoenix, Arizona, USA) (Fig. 3.2) using the procedure validated by Fisher (1987b). If the PPT was less than that of 'normal' muscle tissue (Table 1), the tender point was defined as a LTrP and its position documented on an enlarged body diagram. Pressure-pain threshold measurements were repeated three times and the mean recorded to improve reliability. All PPT measures were taken in quick succession (within approximately 30 seconds) due to the fact that LTrPs can be inactivated by sustained pressure (Hou, C. R. et al. 2002). The order of muscle assessment was randomised for each subject. This LTrP examination process was found to have high intra-examiner reliability (Kappa statistics= 0.71 to 1 muscle dependent; Intra-class correlation coefficient (ICC) for PPTs = .92) using a test/retest protocol with 30 minutes between examinations for the clinician who conducted all of the examinations (see Appendix C). Subjects were also asked if the pain was referred elsewhere before snapping palpation was applied in an attempt to elicit a LTR. When referred pain or a LTR was elicited, the event was documented, and used as additional confirmation of the presence of a LTrP. On the basis of Fischer's work (1987a, 1987b) and other previously cited studies (Gerwin, R. D. et al. 1997; Hong, C. Z. 1996; Lew, Lewis & Story 1997; Sciotti et al. 2001), the definition used to identify a LTrP in the current study became:

A tender point within a palpable taut band of skeletal muscle that had a PPT of less than that expected in normal muscle, with or without referred pain or an LTR.

Table 3.1: Lowest PPT (kg/cm²) at which a muscle can be considered 'normal' (Fischer, AA 1987b).

	Males (kg/cm²)	Females (kg/cm²)
Upper trapezius	2.9	2.0
Scapular muscles	3.6	2.7

Finally, side dominance was determined by asking subjects with which hand they normally wrote.

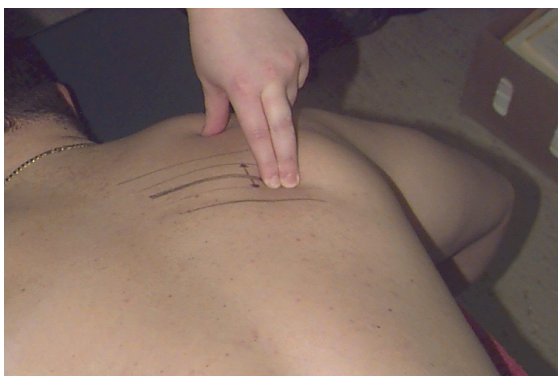


Figure 3.1: Palpation perpendicular to the direction of the muscle fibers to identify the taut band.



Figure 3.2: Using the pressure algometer to measure the PPT of a tender point.

3.3 Statistical Analysis

The number of LTrPs identified and the muscles in which they occurred were tabulated and the means and the percentage of subjects with at least one LTrP determined. Relationships between variables were examined using either Pearson's 'r' (number of LTrPs, age, muscles containing LTrPs) or Point biserial correlations (number of LTrPs, gender, staff member or student and side dominance). Differences between the number of LTrPs in the various muscles were determined using ANOVA. All calculations were made using the Statistical Package for the Social Sciences version 13 and significance was set as $p < 0.05$ for all measurements.

3.4 Results

General characteristics of the sample are presented in Table 3.2. Of the 137 subjects examined (mean age = 34.0 ± 13.2 years; range = 18-60 years), 89.8 percent had at least one LTrP in the scapular positioning muscles (mean= 10.65 ± 6.8 , range=1-27). Of the subjects with LTrPs, 62 percent had more LTrPs on the dominant side, 25 percent had more LTrPs on the non-dominant side and 13 percent had the same number of LTrPs on both sides of the body.

Table 3.2: Demographic data of the sample

	LTrPs N=123 (89.8% of all subjects)		No LTrPs N=14 (10.2% of all subjects)	
	Female N=63	Male N=60	Female N=7	Male N=7
Age	33.0 \pm 13.5	34.8 \pm 13.7	37.4 \pm 8.7	33.7 \pm 9.2
N staff (%)	28 (44%)	32 (53%)	5 (71%)	4 (57%)
N students (%)	35 (56%)	28 (47%)	2 (29%)	3 (43%)
N R-handed (%)	59 (94%)	54 (90%)	7 (100%)	5 (71%)
N L-handed (%)	4 (6%)	6 (10%)	0 (0%)	2 (29%)

N=number of subjects; R-handed=right hand dominant; L-handed=left hand dominant.

Figure 3.3 shows the percentage of subjects who had at least one LTrP in any of the muscles examined. The upper trapezius (78.8 percent), pectoralis minor (77.3 percent), serratus anterior (71.5 percent), lower trapezius (70.4 percent), levator scapulae (68.9 percent) and rhomboids (major and minor together) (65.9 percent) were more likely to have a LTrP than to not have one while middle trapezius was the least likely to have a LTrP (40.7 percent).

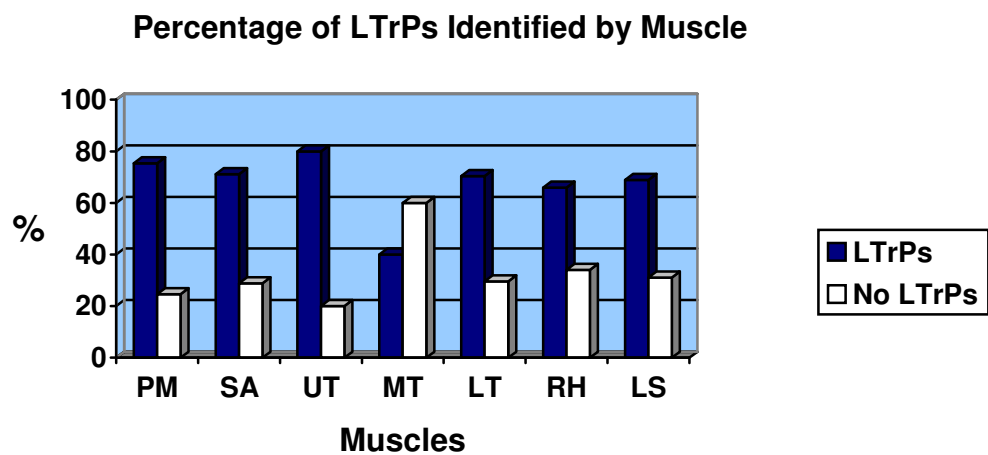


Figure 3.3: Percentage (%) of subjects with LTrPs by muscle. Dark columns are the % with at least 1 LTrP in that muscle. Light columns are the % with no LTrPs. Pectoralis minor (PM), serratus anterior (SA), upper trapezius (UT), middle trapezius (MT), lower trapezius (LT), rhomboids major and minor combined (RH), levator scapulae (LS).

For the subjects who had LTrPs, Table 3.3 displays the mean number for females and males and compares the dominant and non-dominant sides. Because there were no significant differences between the genders for LTrPs (numbers, muscle and side), the table also shows the combined data. The

number of LTrPs was significantly higher ($p < 0.01$) on the dominant side of the body for each muscle investigated.

Table 3.3: Mean number of LTrPs (\pm SD) for females, males, muscles and dominant side of the body (LTrP absent subjects are not included)

	Female	Male	Whole sample
No. LTrPs total	11.10 \pm 5.1	12.37 \pm 5.4	11.72 \pm 6.2
Serr ant D	1.27 \pm 1.0	1.38 \pm 1.1	1.33 \pm 1.1
Serr ant ND	1.08 \pm 1.0	1.18 \pm 0.9	1.13 \pm 0.9
Upp trap D	1.06 \pm 1.2	1.33 \pm 0.6	1.2 \pm 0.7
Upp trap ND	1 \pm 1.3	1.33 \pm 0.7	1.16 \pm 0.8
Pec min D	1.02 \pm 0.7	0.93 \pm 0.8	0.98 \pm 0.7
Pec min ND	0.73 \pm 0.7	0.77 \pm 0.7	0.75 \pm 0.7
Rhoms D	0.89 \pm 0.9	0.95 \pm 0.9	0.92 \pm 0.9
Rhoms ND	0.68 \pm 0.9	0.78 \pm 0.8	0.73 \pm 0.8
Lev scap D	0.9 \pm 1.0	0.88 \pm 0.7	0.89 \pm 0.7
Lev scap ND	0.62 \pm 1.1	0.68 \pm 0.7	0.65 \pm 0.6
Lwr trap D	0.89 \pm 0.6	0.85 \pm 0.7	0.87 \pm 0.7
Lwr trap ND	0.52 \pm 1.1	0.52 \pm 0.6	0.52 \pm 0.6
Mid trap D	0.27 \pm 1.1	0.47 \pm 0.5	0.36 \pm 0.5
Mid trap ND	0.16 \pm 1.4	0.30 \pm 0.5	0.24 \pm 0.4

D=dominant side; ND=non-dominant side; Serr ant=serratus anterior; Upp traps=upper trapezius; Pec min=pectoralis minor; Rhoms=rhomboids major and minor; Lev scap=levator scapulae; Lwr traps=lower trapezius; Mid traps=middle trapezius.

No significant differences were found between gender, age or occupation and number of LTrPs. However, significant differences were identified between the mean number of LTrPs and the muscles in which they occurred (Table 3.4). The results revealed that the serratus anterior and upper trapezius were equally prone to have LTrPs ($p>0.05$) but had significantly more LTrPs than any of the other muscles (all $p<0.01$). Likewise pectoralis minor, rhomboids, levator scapulae and lower trapezius harboured a similar number of LTrPs ($p>0.05$) but all had more than middle trapezius (all $p<0.01$).

Table 3.4: Differences in the number of LTrPs by muscle (mean \pm SD)

	Significantly < ($p<0.001$)	No significant differences in no. LTrPs	Significantly < ($0.001>p<0.01$)	No significant differences in no. LTrPs
Middle trapezius (0.59 ± 0.8)	<	Pectoralis minor (1.72 ± 1.3) Rhomboids (1.65 ± 1.4) Levator scapulae (1.54 ± 1.2) Lower trapezius (1.39 ± 1.0)	<	Serratus anterior (2.46 ± 1.8) Upper trapezius (2.36 ± 1.3)

Two weak but significant point biserial correlations ($p < 0.01$) were found between number of LTrPs and dominant side ($r = 0.14$) and between the number of LTrPs and staff member ($r = 0.16$). A further significant positive correlation ($p < 0.01$) was identified using Pearson's 'r' between the number of LTrPs and age ($r = 0.18$), suggesting that older subjects were more likely to have more LTrPs. No relationship was found between number of LTrPs and gender ($p > 0.05$).

3.5 Discussion of Results

Although a number of studies have investigated the inter-examiner reliability of identifying both ATrPs and LTrPs in specific muscles (Gerwin, R. D. et al. 1997; Lew, Lewis & Story 1997) and the inter-examiner precision in locating LTrPs (Sciotti et al. 2001), there have been no previous studies that have specifically examined the frequency with which LTrPs occur in the scapular positioning muscles.

The objective of the present study was to determine how commonly LTrPs occur within the scapular positioning muscles of ordinary, healthy, pain-free adults. The results confirmed the popular clinical opinion that LTrPs are a common phenomenon with nearly 90 percent of 137 subjects harbouring at least one and often multiple LTrPs in this group of muscles (mean 11.72 for subjects that had LTrPs). In related early work Sola and co-workers (Sola, Rodenberger & Gettys 1955) investigated what they described as the

occurrence of “hypersensitive spots” in the posterior shoulder muscles of 200 healthy, young military recruits. It was later suggested by Simons (Simons, D. 1997) that the “hypersensitive spots” identified in 50 percent of this sample were probably LTrPs. The large discrepancy in occurrence of LTrPs between Sola’s findings (50 percent) and the present study (89.8 percent) may be due to the different populations investigated (active young military recruits compared with university staff and students in the current investigation). In the present study with subjects aged between 18 and 60 years, the correlation between age and the number of LTrPs was weak but significant ($r= 0.18$; $p<0.01$). This may have contributed to a higher occurrence of LTrPs, given subjects in the current study were older (18-60 years versus 18-35 years) and likely to have spent more time in static postures (computer use, desk work and studying), which are thought to predispose to MTrP development (Simons, D., Travell & Simons 1999). Another explanation for the incongruence may lie in the improvement in trigger point examination techniques that have evolved, particularly in the last ten years. It should be emphasised that comparisons between the two studies can at best be speculative given the “suggested” presence of LTrPs in Sola’s study. Some of the same difficulties in comparing the current work with past reports is also evident when considering a more recent study in which Cimbiz et al. (Cimbiz, Beydemir & Mainisaligil 2006) studied 114 university students (mean age = 22.2 years) divided into Myofascial Pain Syndrome (MPS) sufferers and controls for the presence of trigger points in a range of muscles between the occipital and knee regions. The description of trigger points detected in their control group (most comparable with the asymptomatic subjects of the

current study, N=60 range 18-30 years; mean 20.7 years) as 'taut bands and nodules with minimal or no pain', suggests that these were actually LTrPs. They found at least one trigger point in approximately 57 percent of their control subjects with a maximum of five. The trapezius was the most likely muscle to harbour a LTrP (35 percent). Excluding staff members and all subjects over 30 years of age from the data set of the present study (N=70; mean age = 22.9 ± 3.8 years) to provide a better comparison between the two studies, actually resulted in a slight increase in prevalence of LTrPs in the scapular positioning muscles (92.5 percent) for the current sample. Hence, the disparity in the results increased. Perhaps the different diagnostic criteria used to identify a LTrP in the two studies partially explain the difference. In addition, though there were a number of muscles common to both studies, among them, only results for the trapezius were reported in the Cimbiz (2006) publication, making too direct a comparison between the two studies problematic.

In two small clinical investigations dealing with aspects of migraine (Fernandez-de-Las-Penas, C. et al. 2006) and chronic tension-type headache (CTTH) (Fernandez-de-Las-Penas, C et al. 2006), LTrPs were also found in the muscles of the control group subjects. The first study (N=20, suboccipital, upper trapezius, temporalis sternocleidomastoid) found LTrPs in all control subjects (mean 1.7 ± 0.9), while in the second (N=25; upper trapezius, temporalis sternocleidomastoid) the mean was 1.4 LTrPs. However, it was not possible from the data reported in the CTTH subjects to determine the percentage of LTrP occurrence, though it was at least 48

percent. Interestingly in both studies there was no difference between the controls and sufferers in numbers of LTrPs. Furthermore, LTrPs were most often found in the upper trapezius in agreement with both Cimbiz (2006) and the results of the present study.

In terms of the individual muscles examined, pectoralis minor was more likely to have at least one LTrP than serratus anterior (77.3 percent versus 71.5 percent respectively). The seemingly contradictory finding of a greater average number of LTrPs in serratus anterior versus pectoralis minor despite a lower percentage of occurrence, is probably explained by more occasions of multiple LTrPs in the former. The upper trapezius was most likely to contain a LTrP (78.8 percent) but had fewer on average than the serratus anterior (2.36 ± 1.3 versus 2.46 ± 1.8 respectively), probably for the same reason. For the remaining muscles, the percentage of subjects possessing at least one LTrP decreased in the following order: lower trapezius (70.4 percent) levator scapulae (68.9 percent) rhomboids (65.9 percent) but changed when the mean number of LTrPs was considered (rhomboids 1.65 ± 1.4 ; levator scapulae 1.54 ± 1.2 ; lower trapezius 1.39 ± 1.0) The “multiple LTrP explanation” might also account for the change in order though it should be remembered that the differences were not significant. The middle trapezius was least likely to harbour LTrPs (40.7 percent) and where LTrPs were identified, in most cases there was only one (mean= 0.59 ± 0.8).

The largest numbers of LTrPs were found in the serratus anterior and upper trapezius, supporting the accepted clinical view (Simons, D., Travell &

Simons 1999) and available experimental findings (Cimbiz, Beydemir & Mainisaligil 2006; Fernandez-de-Las-Penas, C et al. 2006; Fernandez-de-Las-Penas, C. et al. 2006; Sola, Rodenberger & Gettys 1955) that the upper trapezius frequently develops LTrPs. Simons and collaborators (Simons, D., Travell & Simons 1999) discussed likely structural and functional reasons for this phenomenon and many authors have discussed the significance of trapezius myalgia (Larsson, B. et al. 2000; Larsson, B. et al. 2001; Larsson, R., Oberg & Larsson 1999; Lindman et al. 1991), of which LTrPs may be an early sign (Hong, C. Z. & Simons 1998). The high occurrence in these two muscles (78.5 percent upper trapezius; 71.5 percent, serratus anterior) perhaps reflects the synergy between these two muscles in producing scapular upward rotation which demands precise timing of muscle activation if the movement is to be efficient (Wadsworth & Bullock-Saxton 1997). The reasons for the overall descending order of prevalence: serratus anterior/upper trapezius > pectoralis minor/rhomboids/levator scapulae/lower trapezius > middle trapezius, perhaps also reflects the order of demand placed upon these muscles by common activities which may also be reflected by muscle size or functional capacity.

In all muscles examined for the presence of LTrPs in the current work, subjects were significantly more likely to have a greater number of LTrPs in muscles of the dominant side ($p < 0.05$). According to many authors (Cimbiz, Beydemir & Mainisaligil 2006; Dommerholt 2005; Fernandez-de-las-Penas et al. 2006; Gerwin, R. D. 2005; Simons, D., Travell & Simons 1999), MTrPs can develop due to mechanical loading of the muscle by either a sudden,

sustained or repetitive overload. That there existed a dominant side “preference” for LTrPs seems logical given the greater use of the dominant limb and therefore a greater exposure to conditions that may predispose to their development (fatigue etc).

The reasons for the large disparity in the number of LTrP found in the five studies cited (at least 48 percent (Fernandez-de-Las-Penas, C et al. 2006); 50 percent (Sola, Rodenberger & Gettys 1955); 56.6 percent (Cimbiz, Beydemir & Mainisaligil 2006); 89.8 percent current study; 100 percent (Fernandez-de-Las-Penas, C. et al. 2006)), are multiple (as previously discussed), however, combined, these investigations lend strong support to the notion that LTrPs are common in otherwise healthy people.

3.6 Conclusions

From these results, of the current study it can be concluded that LTrPs in the scapular positioning muscles are common in a sample of normal, healthy adult university students and staff and therefore, likely so in the broader population. Given that LTrPs might develop into ATrPs, which are often identified as the source of pain in patients with pain complaints, it is important to investigate the effects of LTrPs in their own right and also whether treatment of LTrPs affects the future development of ATrPs.

CHAPTER 4

THE EFFECTS OF LTrPs ON MUSCLE ACTIVATION PATTERNS DURING SCAPULAR PLANE ELEVATION

Overview of Chapter

This chapter presents three related experiments designed to establish the effects of LTrPs on the activation patterns of selected scapular muscles and representatives of the rotator cuff group during elevation of the arm in the scapular plane. All were carried out on the same 42 subjects, a subset of the original 154 volunteers described in Chapter 3 who provided data on the prevalence of LTrPs in the scapular rotator muscles. In the first (section 4.1, page 142), MAPs obtained from LTrP-free subjects (Controls) were compared with those obtained from subjects with LTrPs (LTrP group) in the scapular rotator muscles during unloaded scapular plane elevation of the arm. In the second (section 4.2, page 162), the same protocols were followed but with the addition of a load in the form of hand-held weights. The third experiment (section 4.3, page 176) explored the combined affects of fatigue and LTrPs by carrying out scapular plane elevation after fatigue induced by repetition of the test movement while carrying a load. In a fourth experiment, the subject of Chapter five, LTrP subjects were subjected to either an established LTrP treatment (superficial dry needling followed by post-isometric relaxation, see Chapter 2, pages 74-78) or placebo and all of the scapular plane elevation protocols repeated (unloaded, loaded, post-fatigue). In this way, comparisons with pre-treatment conditions and control group results obtained from each experimental condition could be used to both confirm the effects of LTrPs on the criterion measurements (muscle activation patterns (MAPs)) as well as test the efficacy of the treatment. To clarify the sequence of events, a flow diagram has been provided (Figure 4.1.1)

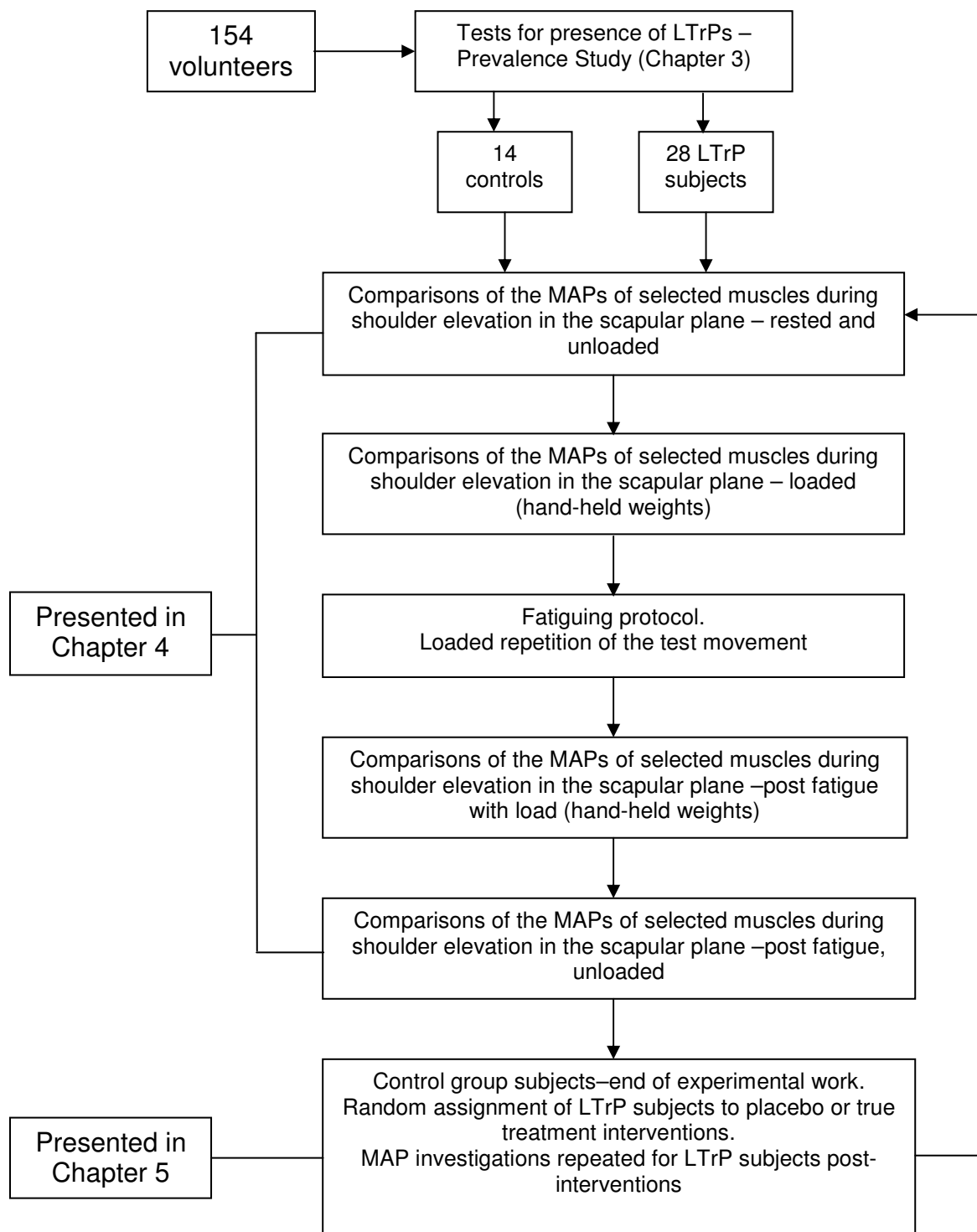


Figure 4.1: Outline of the experimental sequence

In summary, the following questions were addressed:

1. Do LTrPs in the scapular rotator muscles alter their activation patterns (MAPs) during the performance of a common movement (scapular plane elevation of the arm)?
2. What affect (if any), do they have on the activation patterns of muscles placed more distally in the kinetic chain of the upper limb?
3. Does adding resistance to the criterion movement, affect the MAPs displayed by LTrP subjects?
4. Does inducing fatigue in the affected muscles affect the MAPs displayed by LTrP subjects during production of the criterion movement?
5. Does a commonly employed TrP treatment (superficial dry needling see page_) reverse any altered MAPs that might be attributable to the presence of LTrPs (questions 1 and 2. Presented in Chapter 5)?

The current Chapter is divided into three sections, each dealing with one of the experimental conditions presented in Table 4.1.1 and each is more or less self-contained with its own introduction, description of methods, statistics, results, discussion and conclusions. Such an approach means some repetition of material presented in earlier Chapters however, it has the advantage of avoiding constant cross referencing.

Table 4.1.1: Experimental conditions investigated.

Condition	Section
Rested and unloaded	4.1 The effect of LTrPs on MAPs during scapular plane elevation
Rested and loaded	4.2 The effect of LTrPs on MAPs during loaded scapular plane elevation
Post-fatigue, unloaded	4.3 The effect of LTrPs on MAPs post-fatigue during scapular plane elevation
Post-fatigue, loaded	4.3 The effect of LTrPs on MAPs post-fatigue during loaded scapular plane elevation

4.1 The Effects of LTrPs in the Scapular Rotator Muscles on MAPs during Elevation in the Scapular Plane.

4.1.1 Introduction

Many clinicians suggest that it is useful to view the musculoskeletal system as a series of segments linking to form kinetic chains that transfer force in a coordinated manner to produce movement at more distal or proximal segments in any chain (so-called closed and open kinetic chain movements respectively) (Kibler, W. B. 1998b). Kibler (1998) suggested that deficiencies (due for example, to injury, overload, fatigue or TrPs), in proximal segments of such systems, could change the loading patterns in related distal segments and thereby compel changes in muscle recruitment patterns distally as the nervous system sought to preserve normal movement outcomes. If this be true, various musculoskeletal conditions (overload, inflammatory or degenerative) affecting structures in one segment of a kinetic chain, might predispose tissues in other segments to injury/dysfunction because of altered loading patterns (Kibler, W. B. 1998b). For the upper extremity, the scapula and the muscles attaching it to the vertebrae and ribs, (trapezius, serratus anterior, rhomboids, levator scapulae and pectoralis minor), can be considered to constitute the proximal segment linking the trunk to the upper limb (Burkhart, S. S., Morgan & Kibler 2003b, 2003a; Kibler, W. B. 1998b, 1998a; Van der Helm et al. 1992). In order for the scapula to be positioned and moved effectively in its role of force

transference to and from the upper limb, the scapular positioning muscles must be recruited in optimal patterns (MAPs). Hence, any disturbance to the normal pattern of recruitment could be transferred “downstream” promoting abnormal patterns distally, for example in the infraspinatus and rotator cuff, and in consequence, exacerbating the original problem or potentially developing a new problem.

Scapular dyskinesis describes an alteration in the normal position or motion of the scapula during coupled scapulohumeral movements (Burkhart, S. S., Morgan & Kibler 2003b) that is commonly associated with compression of the contents of the subacromial space (subacromial impingement syndrome) which can lead to inflammatory or degenerative changes in these structures as well as to the appearance of TrPs due to overload in muscles attempting to cope with shoulder joint pathology (Brossmann et al. 1996; Burkhart, S.S. 2006; Hebert et al. 2002; Sevinsky 2006). This relatively common upper extremity condition highlights the need to maintain normal scapular muscle control which may be lost if Kibler’s propositions are correct. For example, Wadsworth and Bullock-Saxton (1997) investigated the MAPs of the scapular upward rotator muscles of nine young elite male swimmers with unilateral chronic shoulder impingement syndrome and compared them to matched controls during elevation of the arm in the scapular plane. They found that chronic shoulder pain was associated with an increased variability in the timing of muscle activation in these muscles, however, they were unable to establish a cause and effect relationship since it was impossible to determine which condition (altered MAPs or impingement) occurred first. Importantly

with regard to the present focus on LTrPs, Sterling and colleagues (2001) suggested that changes in motor control could occur through a process of reflex inhibition secondary to non-painful sensory input (mechanoreceptors) which in turn could eventually lead to the development of pain. They felt that this phenomenon might explain the persistent weakness and atrophy observed in the quadriceps muscles after non painful knee damage without effusion (Hurley 1997; Sterling, Jull & Wright 2001). Latent myofascial trigger points (LTrPs) are pain-free neuromuscular lesions that are associated with muscle overload and decreased contractile efficiency (Simons, D., Travell & Simons 1999) and there is evidence that these lesions are common (Simons, D., Travell & Simons 1999) (see also Chapter 3), suggesting that they deserve investigation. The following section presents an investigation into the effects of LTrPs in the scapular rotator muscles on MAPs during scapular plane elevation of the arm in both the muscles harbouring them and those downstream in the upper extremity kinetic chain.

4.1.2 Methods

4.1.2.1 Subjects

The subjects were a subset of those who were recruited for the prevalence study described in Chapter 3. Of the original 154 pain-free volunteers, 14 met the criteria for inclusion in this part of the investigation as “controls” that is,

they were the only subjects with healthy shoulders and no LTrPs (see Chapter 3). From the 140 LTrP sufferers, the first twenty-eight subjects who were assessed as having healthy shoulder girdles and at least one LTrP in the scapular positioning muscles on the dominant side, were invited to participate in the remaining components of the study and in due course, were randomly assigned to receive either treatment (N=14) or sham treatment (N=14) in the final investigations where the effects of superficial dry needling were tested (Chapter 5). Subjects were excluded if they had less than 160° of arm elevation, had a positive apprehension test (glenohumeral instability), positive upper limb tension test (neurological dysfunction) or significantly increased thoracic kyphosis (judged by clinical observation), reported any pain in the back, neck or either upper extremity any time in the week prior to the examination, or harboured LTrPs in the infraspinatus or middle deltoid muscles on the dominant side. Subjects were examined bilaterally for the clinical characteristics of LTrPs in the pectoralis minor, serratus anterior and middle deltoid (examined lying supine), all parts of the trapezius and rhomboids, the levator scapulae and the infraspinatus (examined lying prone). Examinations of infraspinatus and middle deltoid were carried out to ensure that they harboured no LTrPs which could have affected muscle activation patterns either intrinsically and/or in related muscles. All examinations were carried out by the same trained and experienced (12 years) Myotherapist using procedures explained by Simons, Travell and Simons (Simons, D., Travell & Simons 1999) and employed by Lew and colleagues (Lew, Lewis & Story 1997) in their reliability study. A full description of the LTrP examination process was provided in Chapter 3 (page

122). For reasons detailed in Chapter 3 (page 124), the definition used to define a LTrP in the current study became:

A tender point within a palpable taut band of skeletal muscle that had a PPT of less than that expected in normal muscle (see Table 4.1.2), with or without referred pain or an LTR.

Table 4.1.2: Lowest PPT (kg/cm²) at which a muscle can be considered 'normal' (Fischer, 1987).

	Males (kg/cm²)	Females (kg/cm²)
Upper trapezius	2.9	2.0
Scapular muscles	3.6	2.7

All participants gave informed consent and all procedures were approved by the RMIT University Human Research Ethics Committee. Characteristics of the experimental groups are provided in Table 4.1.3.

Table 4.1.3: Characteristics 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	28	Yes	33.86 ± 11.4	12	16

It should be recalled that nearly 90% of the original volunteers (Chapter three) had at least one LTrP (mean =10.65) in the muscles examined and for the present study, all 28 LTrP subjects had at least one LTrP in the scapular rotator muscle group of the dominant arm, however mean number and standard deviation (SD) of LTrPs are described for each scapular positioning muscle in Table 4.1.4.

Table 4.1.4: Mean number and standard deviation of LTrPs by muscle in the LTrP group (N=28) in the dominant upper extremity.

	PM	SA	UT	MT	LT	RH	LS	TOTAL SRM
Mean	0.86	1.75	1.39	0.36	1.00	1.11	0.71	13.50
SD	0.76	1.04	0.69	0.56	0.72	0.96	0.76	4.99
% of subjects	64	82	93	32	79	64	56	100

PM=pectoralis minor; SA=serratus anterior; UT=upper trapezius; MT=middle trapezius; LT=lower trapezius; Rh=rhomboids major and minor combined; LS=levator scapulae; Total SRM=total number of LTrPs in the scapular rotators as a group; SD=standard deviation.

4.1.2.2 *Time of onset of muscle activation*

Surface electromyography (sEMG) was used to measure time of onset of muscle activity for five muscles of the dominant arm, chosen on the basis of accessibility (for sEMG) and their known functions as either scapular rotators, muscles of the rotator cuff group or prime movers for glenohumeral

abduction. Specifically, the upper and lower trapezius and serratus anterior represented upward scapular rotators, the infraspinatus, the rotator cuff group and the middle deltoid, a prime glenohumeral abductor. The infraspinatus and deltoid belong to a functionally different muscle group than the scapular rotators and represent a more distal component of the upper extremity kinetic chain. Importantly, Laursen and colleagues (2003), on the basis of their biomechanical model, (which used predicted EMG activity to establish the roles of selected shoulder muscles), concluded that the glenohumeral stabilising role of the infraspinatus was more important than its role as an external rotator (Laursen, Sogaard & Sjogaard 2003). This factor and the muscle's amenability to sEMG (compared with the specialised abducting role and deep position of supraspinatus), provided the rationale for its selection as a representative of the rotator cuff muscles in the current work.

Bipolar Ag/AgCl electrodes (3M Red Dot) were used and were positioned according to Cram and colleagues (Cram, Kasman & Holtz 1998) using a two centimetre inter-electrode distance. 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=500Hz, high pass 10Hz), rectified then smoothed using a root mean square (RMS) calculation. The sampling speed was 2000 samples/sec. A custom built microswitch was placed on the subject's thigh to align with the 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 the time of onset of muscle activity to be normalised to the start of the movement. The test movement was carried out according to the procedures reported by Wadsworth and Bullock-Saxton (1997). Plane of motion, standing posture, and postural sway were controlled by asking the subjects to look at a target approximately two metres ahead of them positioned at eye level and to lightly brush wooden movement guides (vertical wooden panels set at appropriate angles) while velocity of movement was controlled by asking subjects to move in time with a metronome set at 60 beats per minute, with four beats to raise and four beats to lower the arm, equivalent to 40 degrees per second (Figures 4.1.2 and 4.1.3).

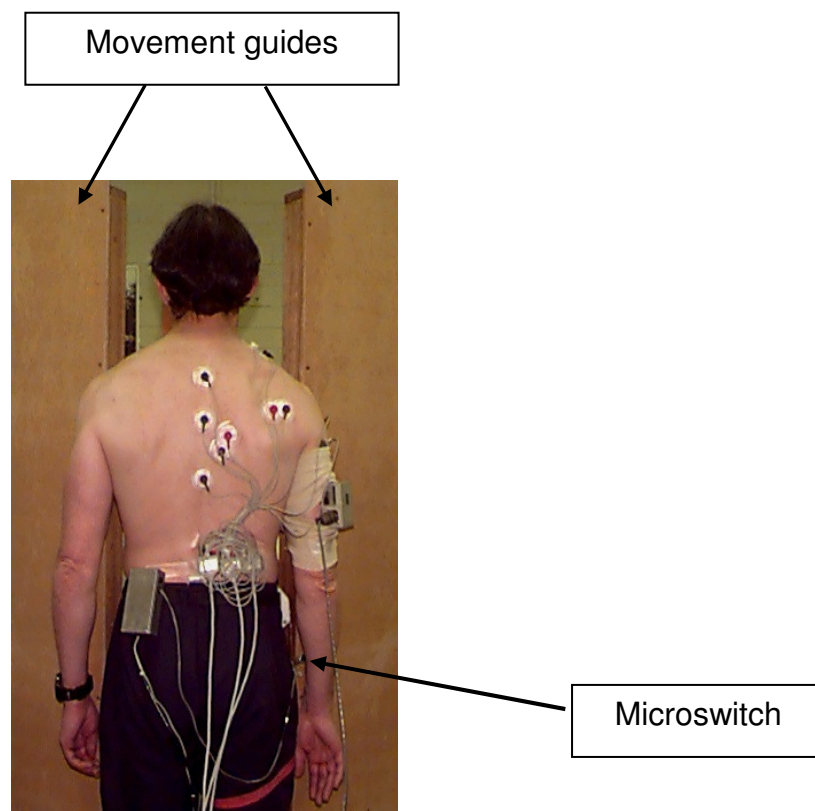


Figure 4.1.2: Starting position of the test movement.

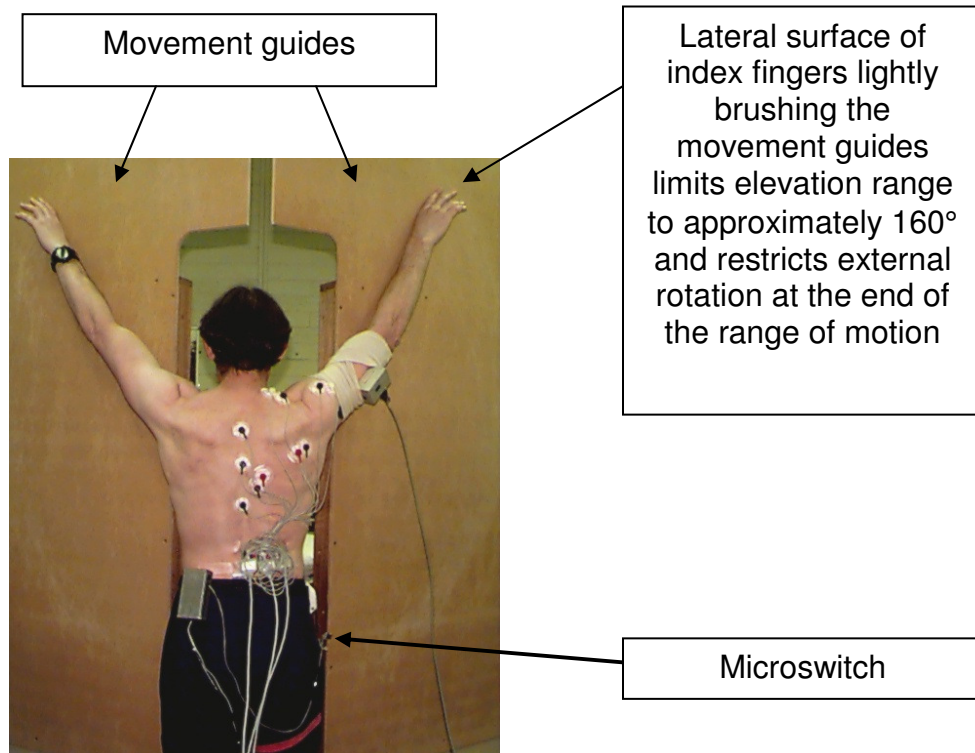


Figure 4.1.3: Performing elevation of the arm in the scapular plane. Note lateral aspect of the index finger remains in contact with the movement guide to restrict external rotation of the shoulder at the top of the movement.

Elevation of the arms in the scapular plane was performed without allowing the subject to externally rotate at the end of the range (Figure 4.1.3). This was accomplished by instructing the subjects to maintain contact of the lateral surface of the index finger with the movement guides throughout the movement. This strategy restricted subjects to 160° of abduction but allowed the infraspinatus to act as a glenohumeral stabiliser rather than as a prime mover for external rotation. Prior to application of sEMG, subjects practised the velocity of movement in time with the metronome until they could reliably

reproduce the required velocity. After adequate rest (5 -10 minutes), subjects performed three trials of the movement in time with the metronome with a four second rest between trials to re-establish a stable electrical baseline. To identify the onset of muscle activity, the algorithm suggested by Hodges and Bui (1996) for a low-noise signal (10ms windows, 1 standard deviation above the baseline and 500hz low pass filter) was employed (Hodges, P. W. & Bui 1996). The time of onset of muscle activity was defined as the time at the start of the first 10ms window whose mean was more than one standard deviation (SD) above the mean of the baseline. The accuracy of this process was confirmed by a visual inspection to ensure the time identified as the beginning of muscle activity was not associated with ECG or other artefact.

4.1.2.3 Statistical Analysis

An independent t-test was used to test for differences in the mean muscle activation times for the control and LTrP groups and the F statistic was used to identify significant differences in the variability of activation times between groups and was calculated by dividing the variance of one group (higher value variance) by the variance of the other group (lower value) and then compared to the appropriate critical value of F. The significance level was set at $p < 0.05$ for all comparisons.

4.1.3 Results

Group data are depicted in Table 4.1.5 and Figure 4.1.4 and show the mean (solid circles) and SD (bars) of activation times for each muscle for both groups. Examples of raw sEMG are provided in Figures 4.1.4 and 4.1.5.

4.1.3.1 Control group MAP

The control group displayed a relatively stable, sequential MAP with all subjects demonstrating the same order of muscle activation which consisted of the upper trapezius (UT) always activated first, on average 115ms prior to movement start. Immediately after the arm left the side of the body, the infraspinatus (Inf) (mean=75ms) and the middle deltoid (MD) (mean=201ms) were activated. The serratus anterior (SA) and lower trapezius (LT) were activated 433ms and 776ms after movement start respectively and displayed more variability in activation times than did the preceding three muscles as evidenced by the length of the SD bars in Figure 4.1.4.

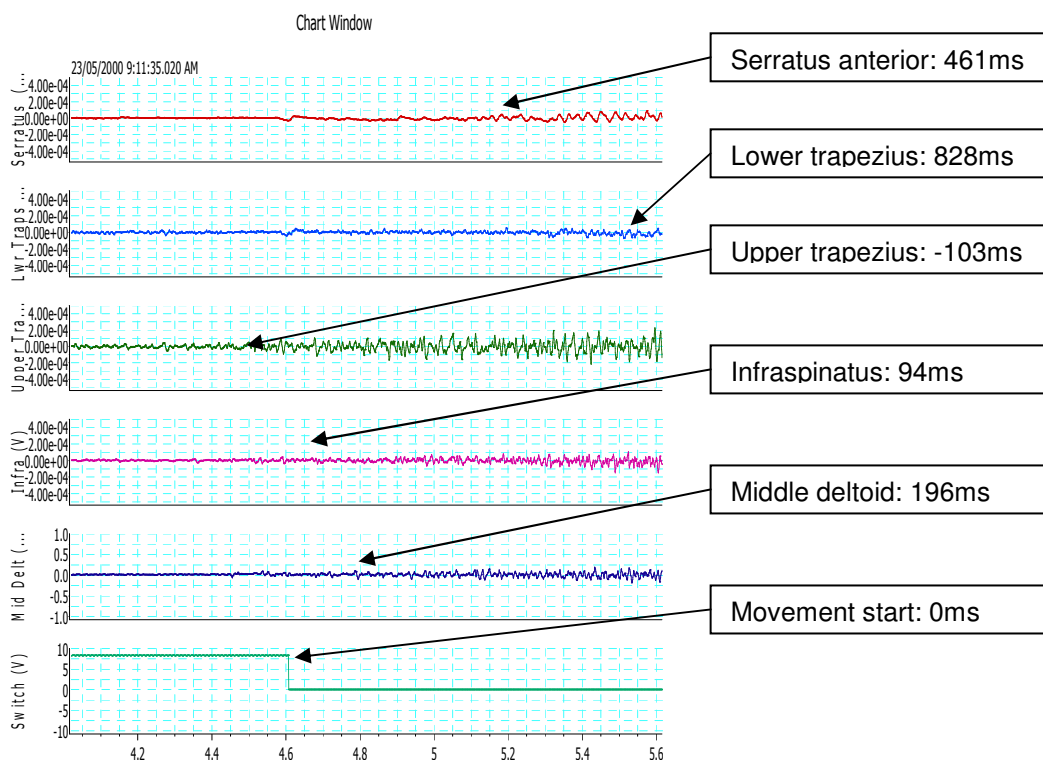


Figure 4.1.4: Raw sEMG from an individual control group subject.

4.1.3.2 LTrP group MAP

In contrast to the control group, the only consistency in the order of muscle activation for the LTrP group was that Inf was activated first in 13 out of 14 subjects (92.9%). Beyond this finding, the order of muscle activation was inconsistent across the group with the most common activation sequence being Inf activated prior to movement start, UT approximately as the arm began to move, then SA, MD and LT after movement start respectively (three out of 14 subjects, 21.4%). With regard to the mean activation pattern for the group, Inf was activated 153ms prior to movement start, followed by UT (27ms), MD (142ms), SA (212ms) and LT (477ms) after movement start respectively.

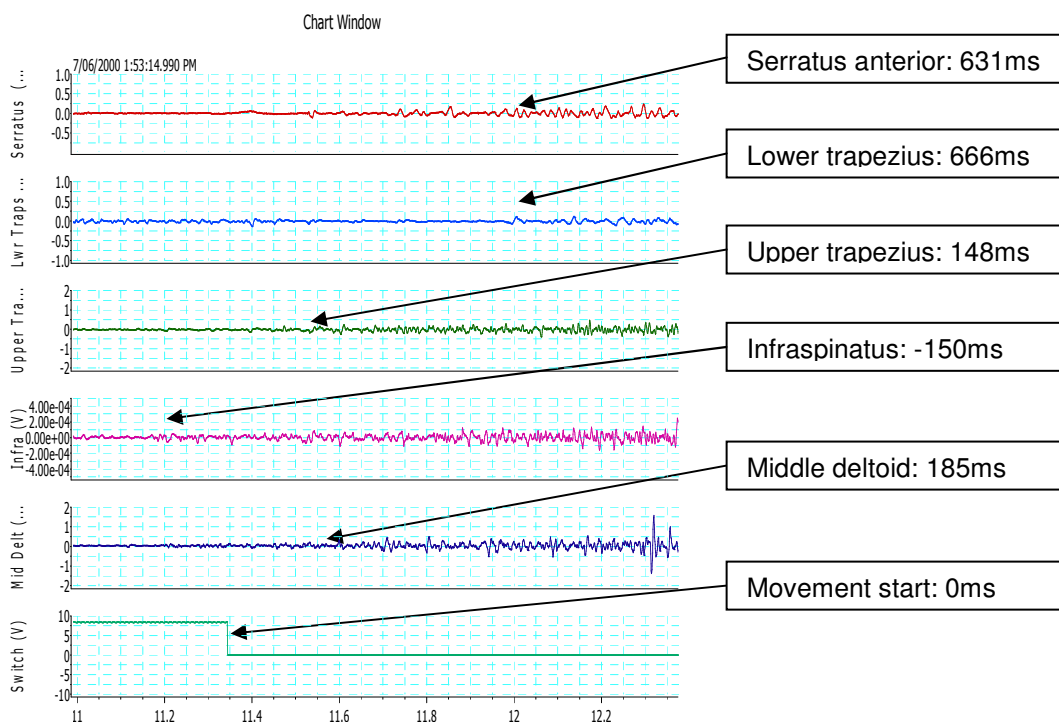


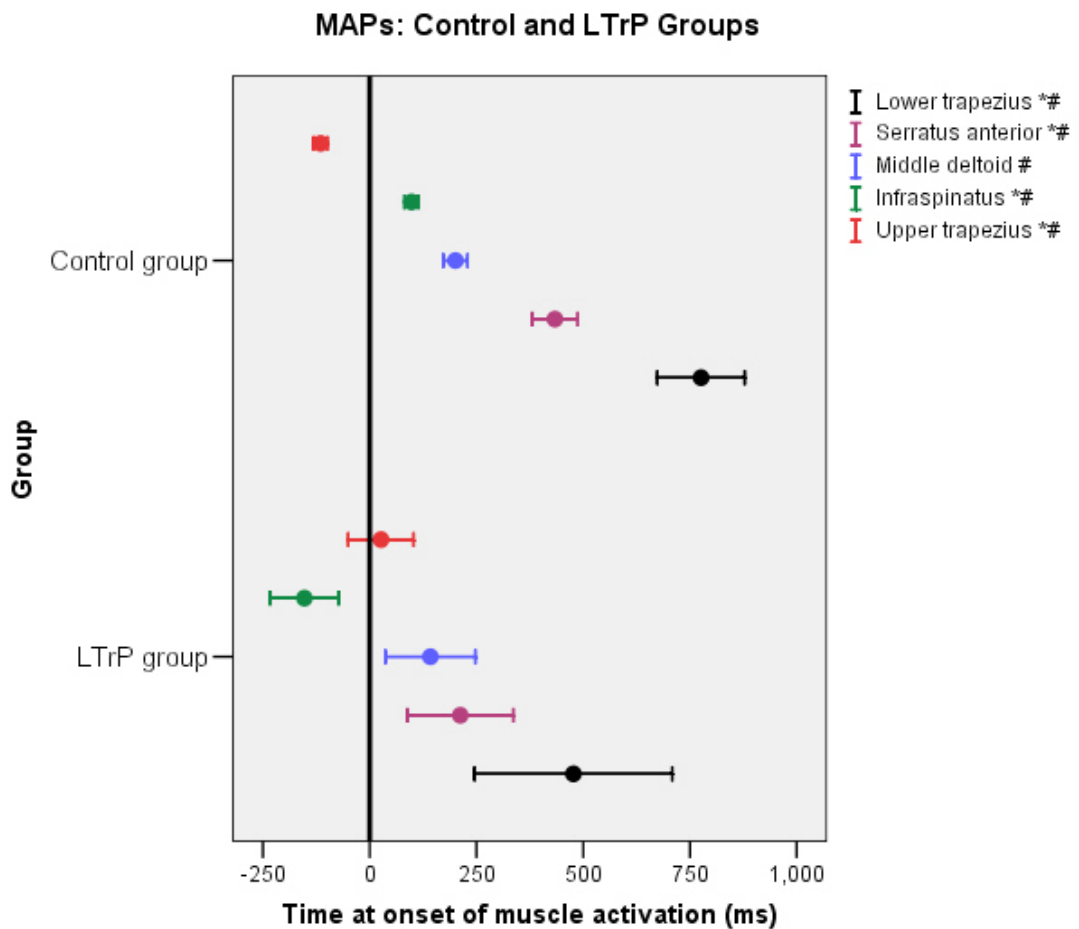
Figure 4.1.5: Raw sEMG from an individual LTrP group subject

Significant differences ($p < 0.05$) between groups for mean activation times were found for all muscles except the MD. In addition, the variability in muscle activation times was significantly greater for all muscles in the LTrP compared with the controls.

Table 4.1.5: Mean muscle activation times for the control and LTrP groups in the rested state.

	UT	Inf	MD	SA	LT
Control	-115	75	201	434	776
LTrPs	27* [#]	-153* [#]	142 [#]	212* [#]	477* [#]

* significant difference in activation times. [#] significant difference in the variability of activation times ($p < 0.05$). UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius.



*significant difference in time of activation. # significant difference in the variability of activation times ($p>0.05$).

Figure 4.1.6: The effects of LTrPs on MAPs during scapular plane elevation (mean and SD displayed). Time '0' is the time at which the arm left the side of the body as measured by the microswitch.

4.1.4 Discussion of Results

In this study the control group displayed a relatively stable and sequential MAP with the UT consistently activated before movement start. As shown by Bagg and Forrest (1986), the instantaneous centre of rotation (ICR) of the

scapula is located near the root of the spine of the scapula during the initial phase of elevation of the arm in the scapular plane (the first 60-90°), giving the UT an advantageous moment arm and length-tension relationship for elevating the lateral clavicle and acromion, perhaps to maintain/increase the subacromial space guarding against superior translation of the humeral head (Bagg & Forrest 1986; Graichen et al. 1999). The initial activity (at -115ms) in the UT was not enough to cause the arm to leave the side of the body and thus activate the microswitch, suggesting this initial UT activation was aimed specifically at elevating the acromion, rather than the entire arm. The Inf was activated 75ms after movement start (on average), perhaps in keeping with its primary roles in the early phase of scapular plane elevation: resisting superior and anterior translation of the humerus by compressing the head against the glenoid fossa (thereby preventing the excessive translations associated with subacromial impingement syndrome) and contributing to a force couple with the other rotator cuff muscles for abduction of the humerus (Halder et al. 2001; Sharkey & Marder 1995). Interestingly, the MD was not activated until some 200ms after the arm left the side of the body, adding support to the notion that other muscles such as the remaining members of the abduction force couple (Inf, supraspinatus and subscapularis), are responsible for initiating the movement of the arm from the side of the body (Michener, McClure & Karduna 2003). Although only five muscle activation times were analysed and no kinematic data were collected in this study, based on the movement speed of 40°/sec, it can be estimated that at 200ms post movement start (MD activation), the arm would be in the vicinity of 8° of abduction. In contrast, the Inf was activated on average within 75ms of

movement start, which places the arm within the first 3° of abduction at the time. According to Liu and colleagues (1997), the supraspinatus has its peak moment arm at approximately 30° of scapular plane elevation, suggesting that the supraspinatus might not be in a position to initiate arm movement on its own (Liu et al. 1997), however length/tension factors should also be considered. Given the importance of coordinated movement of the functional segments of the upper extremity to facilitate efficient scapular plane elevation (Kibler, W. B. 1998b), it appears most likely that the coordinated actions of the UT and rotator cuff combine to produce the first few degrees of arm elevation while acting to preserve the subacromial space, though onset data from other rotator cuff muscles would be useful to confirm this proposal. The early activations of UT and Inf immediately prior to and after movement start respectively, indicate that these muscles play important roles in initiating elevation of the arm in the scapular plane compared with the much later onsets for SA and LT (433ms and 776ms after movement start respectively). These data support an early report by Bagg and Forrest (1988) who found that SA and LT probably have their most favourable combination of moment arm (Bagg & Forrest 1986) and length/tension relationship (Neumann 2002) once the glenoid has rotated superiorly and the ICR of the scapula had migrated laterally (Bagg & Forrest 1988).

A significantly different ($p < 0.05$) temporal sequence of muscle activation was found in both the scapular rotators and shoulder muscles when LTrPs were present in the scapular rotator muscles, suggesting that LTrPs do indeed affect MAPs in the “parent” muscle group and functionally related muscles in

the upper extremity chain. This contention was further supported by the significantly greater variability in muscle activation times, indicative of less consistency in activation patterns, for all five muscles in the LTrP group. Interestingly, increased variability of MAPs is a feature that has often been associated with muscle fatigue (Chabran, Maton & Fourment 2002) and joint injury (Wadsworth & Bullock-Saxton 1997). This finding (greater variability in muscle activation times) indicates that LTrPs force an alteration in the strategy used by the CNS to elevate the arm and perhaps these patterns represent coping behaviours associated with decreased movement efficiency. In fact, the only “reliable” aspect of the LTrP group pattern was that the Inf was commonly activated first (92.9% of trials), and the UT just prior to or immediately after movement start (± 90 ms from movement start in 57.1% of trials). Alternatively, perhaps the descending commands remain the same, but they meet motoneurons that are unable to respond appropriately due to inhibitory influences set in train by the presence of LTrPs with a resultant change in order of activation (Taylor, Butler & Gandevia 2000). Some of the potential mechanisms for these propositions are presented in Chapter 5, page 220.

Given the high likelihood of LTrP group subjects having a LTrP in the UT (93% of 28 otherwise asymptomatic subjects, Table 4.1.4, page 147) it is important to note that when the UT contained a LTrP, the UT was activated at approximately the same time as the arm began to move from the side of the body, whereas when this muscle was LTrP-free (control group), it was clearly activated before the arm began to move. 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. Furthermore, where LTrPs existed in the scapular positioning muscles, the Inf was activated 153ms before the arm left 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, an interesting possibility given the high prevalence of rotator cuff overload and tendinitis in many countries (Netherlands (van der Windt et al. 1995), Britain (Ostor et al. 2005) and Australia (Green, Buchbinder & Hetrick 2003). Perhaps increased duration of activation of Inf along with an altered, and possibly less effective MAP overall, due to LTrPs, contributes to the occurrence of this phenomenon?

Due to the fact that the human musculoskeletal system is a redundant system with more muscles involved in the generation of joint torque than the number of degrees of freedom of the joint (Bernstein 1967), humans can generate the same joint torque with numerous combinations of MAPs (Yao, Acosta & Dewald 2006). Therefore, individuals can use different MAPs to achieve the same motor task with varying degrees of efficiency. In the only other study to date to specifically measure MAPs in the scapular rotator muscles, Wadsworth and Bullock-Saxton (1997) investigated the temporal sequence of recruitment of the upward scapular rotator muscles in nine competitive young freestyle swimmers with unilateral shoulder pain including

signs of impingement and compared them to matched controls. In the control group, they found that UT was activated 217ms prior to movement start and the SA and LT were activated 53ms (approximately 2° of abduction based on movement speed) and 349ms (approximately 15° of abduction based on movement speed) after movement start respectively (Wadsworth & Bullock-Saxton 1997). These authors reported control group mean activation times in their study that differed from those of the control group in the present study (UT -217 Vs -115; SA 53ms Vs 433ms and LT 349ms Vs 776ms), but the order of activation for the scapular rotator muscles was the same (UT prior to movement start, followed by SA, then LT after movement start). These differences may be due to the different populations investigated (young competitive male swimmers (mean age=19.3years) Vs female and male university staff or students (mean age=35.6years)) or the fact that the former study defined the muscle as 'activated' when the EMG trace was more than five percent higher than the baseline, whereas in the current study, a different algorithm was employed to determine onset (10ms 'sliding window' with 1SD above the baseline). In both studies visual verification of the trace was used to eliminate artifact. Interestingly, though the activation times differed between the respective control groups (Wadsworth and Bullock-Saxton and the current study), there were no significant differences in the variability of onset times (found by squaring the SD to calculate the variance, then dividing one group variance by the other to calculate the F statistic) for any of the muscles common to the two studies (Table 4.1.6). In addition, the experimental groups in both studies demonstrated significantly increased

variability in activation times when compared with their respective control groups.

Table 4.1.6: Comparison of mean activation times (\pm SD) for the upward scapular rotator muscles during scapular plane elevation between the Wadsworth and Bullock-Saxton (1997) study and the current study.

Studies and groups	UT	SA	LT
Wadsworth and Bullock-Saxton: control MAP (ms)	-217 \pm 110	53 \pm 478	349 \pm 340
Current study: control MAP (ms)	-115 \pm 28	433 \pm 93	776 \pm 177
Wadsworth and Bullock-Saxton: SIS MAP (ms)	-138	182	498
Current study: LTrP MAP (ms)	27 \pm 132	212 \pm 215	477 \pm 401

UT=upper trapezius, SA=serratus anterior, LT=lower trapezius, SIS=shoulder impingement syndrome, MAP=muscle activation pattern, (ms)=milliseconds. '-' = muscle activated prior to the arm leaving the side of the body. Standard deviations for the SIS group of the Wadsworth and Bullock-Saxton (1997) study were not available.

4.1.5 Conclusions

The presence of LTrPs in the scapular rotator muscles was associated with changes in motor control patterns in the absence of pain, manifested as altered activation times and increased variability of muscle activation

patterns. Such changes may predispose individuals to increased risk of subacromial impingement, overuse of the infraspinatus due to earlier activation and decreased efficiency of movement with earlier onset of fatigue during scapular plane elevation.

LTrPs in the scapular rotator muscles do alter the timing and decrease the consistency of the MAPs of this muscle group and functionally related muscles more distally placed in the upper extremity chain. These findings occurred in the absence of pain and may have implications for overuse syndromes (rotator cuff), shoulder impingement syndrome, and less effective motor control in “overhead” movement patterns in general.

Having found evidence that LTrPs have significant effects on the timing and consistency of MAPs in unloaded motion, section 4.2 details an investigation into the effects of LTrPs in the scapular rotators during elevation of the arm in the scapular plane holding a light load, replicating a movement task that may be performed in many daily work and sporting activities.

4.2 The Effects of LTrPs in the Scapular Rotator Muscles on MAPs during Loaded Elevation in the Scapular Plane.

4.2.1 Introduction

While maximal external loads have been found to alter scapulohumeral rhythm during arm elevation in the scapular plane in healthy subjects (McQuade & Smidt 1998), two three-dimensional studies found that light loads of 0-3kg, (de Groot, van Woensel & van der Helm 1999) and 0-4kg (Pascoal et al. 2000) had no affect on clavicular or scapular kinematics during scapular plane elevation. However, none of these data shed light on what influence LTrPs might have on MAPs when light loads are lifted. Having found evidence that LTrPs have significant affects on the timing and consistency of MAPs in unloaded motion (section 4.1, page 152), the following study was carried out to test the proposition that light loads would increase the degree of dysfunction produced by LTrPs in the scapular rotators during execution of the same common movement pattern, elevation of the arm in the scapular plane.

4.2.12.1 Questions addressed:

1. Do loads commonly encountered in daily activities alter the MAPs of functionally related muscles during scapular plane elevation in 'LTrP-free population'?

2. Do LTrPs in the scapular rotator muscles produce different MAPs during scapular plane elevation in response to the same light loads?

4.2.2 Methods

4.2.2.1 Subjects and Procedures

For this experiment, the same subjects formed the control and LTrPs groups (Section 4.1) respectively and sEMG was recorded in the loaded experimental condition approximately five minutes after the unloaded data were collected, during which time the subjects rested with electrodes still in place. The procedures used in this experimental condition were almost identical to those reported in section 4.1 (page 144), with the exception that subjects were asked to hold one of two hand-weights (1.3kgs or 4kgs), while performing elevation in the scapular plane. The loads were chosen on the basis of subject feedback during pilot testing, when 1.3kg and 4kg hand-weights were selected by most females and males respectively, when asked to choose a weight that they regularly lifted. Therefore these loads were considered to be representative of those that might be encountered in activities of daily living or work-related tasks for the subjects in the current study. To maintain scapular plane motion, this time, the end of the weight was brushed along the movement guides rather than the index finger (Figure 4.2.5).

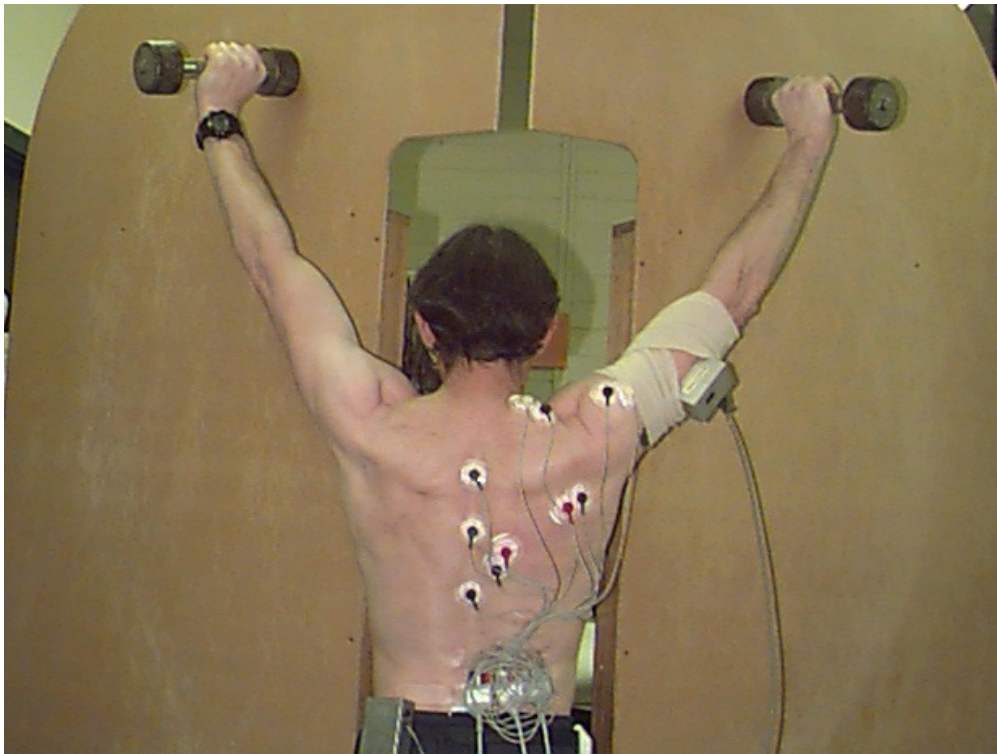


Figure 4.2.1: Loaded elevation in the scapular plane with hand-held weights gently brushing the movement guides to control plane of motion.

4.2.2.2 Statistical Analysis

To test for the effects of load on the MAPs during the test movement in both the control and LTrP groups, paired t-tests were employed and the comparison was made with the data collected in the first experiment (see section 4.1, page 151), where the same subjects (in control and LTrP groups) performed unloaded elevation in the scapular plane. To test for the effects of LTrPs on MAPs during loaded scapular plane elevation, an independent t-test was employed to examine differences in the mean activation times of the control and LTrP groups for all muscles. F statistics were used to compare

the variability in the muscle activation times both within and between groups. The significance level was again set at $p < 0.05$ for all comparisons.

4.2.3 Results

4.2.3.1 Control group MAPs (within group comparisons)

The muscle activation patterns for the control group subjects under both unloaded and loaded conditions are displayed in Table 4.2.1 and Figure 4.2.2. Paired t-tests indicated that although the order of muscle activation was the same under both conditions, the timing of activation was significantly different ($p < 0.05$), with all five muscles activated earlier under load. In terms of the stability of the MAPs as indicated by comparisons of the standard deviations (via F statistics) for muscle onset times for each muscle under the two conditions, the UT was more variable, and the MD less variable under load (both $p < 0.05$). There were no other significant differences for the effects of load in the control group.

Table 4.2.1: Mean times of muscle activation for the control and LTrP groups for the unloaded and loaded conditions.

MAPs	UT	Inf	MD	SA	LT
Control unloaded MAP	-115	75	201	434	776
Control loaded MAP	-191	-57	-6	316	536
LTrP unloaded MAP	27	-153	142	212	477
LTrP loaded MAP	-57	-244	25	91	343

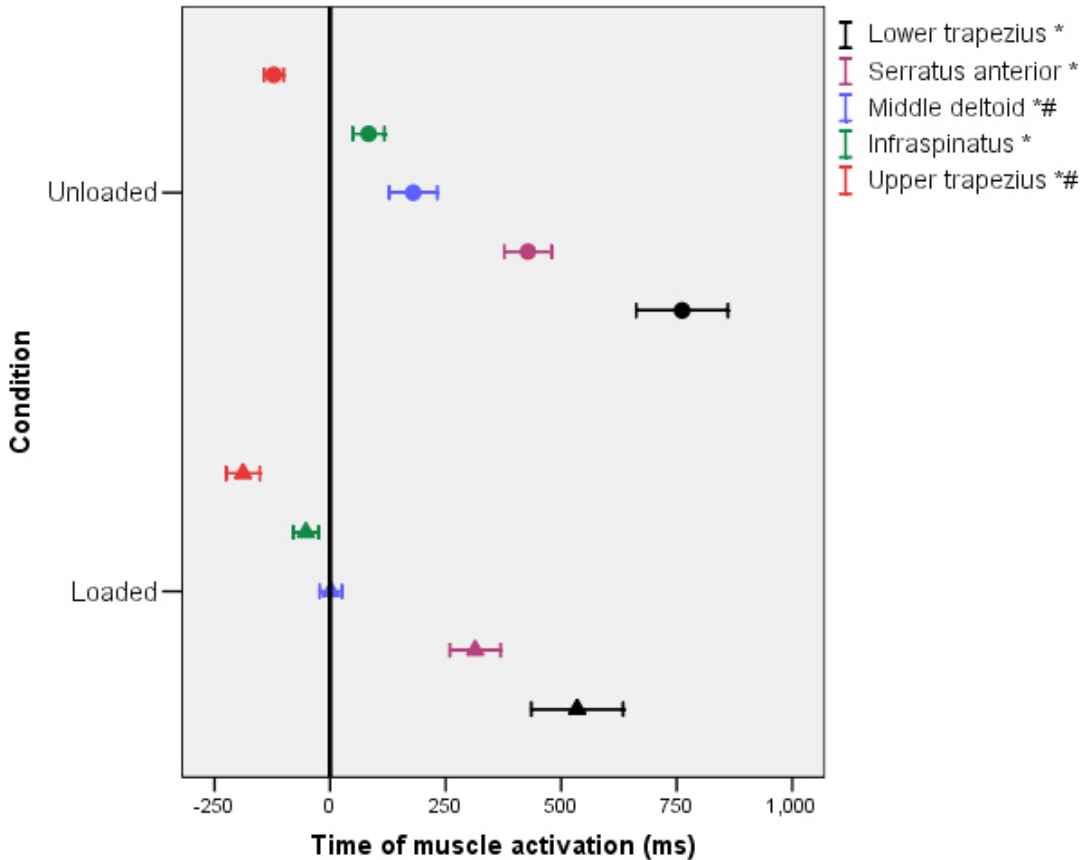
UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius, '-'=muscle activated prior to movement start.

Table 4.2.2: Differences in mean activation times between groups, comparing the unloaded and loaded conditions

Differences in activation times	UT	Inf	MD	SA	LT
Difference control unloaded Vs loaded	-76	-132	-207	-118	-240
Difference LTrP unloaded Vs loaded	-84	-91	-117	-121	-134
Difference control Vs LTrP unloaded	+142	-228	-59	-222	-299
Difference control Vs LTrP loaded	+134	-187	+31	-225	-193

UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius. ‘-‘=muscle activated earlier in the group named second (loaded condition or in the LTrP group compared with the control group respectively); ‘+‘=muscle activated later in the group named second (loaded condition or in the LTrP group compared with the control group respectively).

MAPs: Control Group Unloaded Vs Loaded



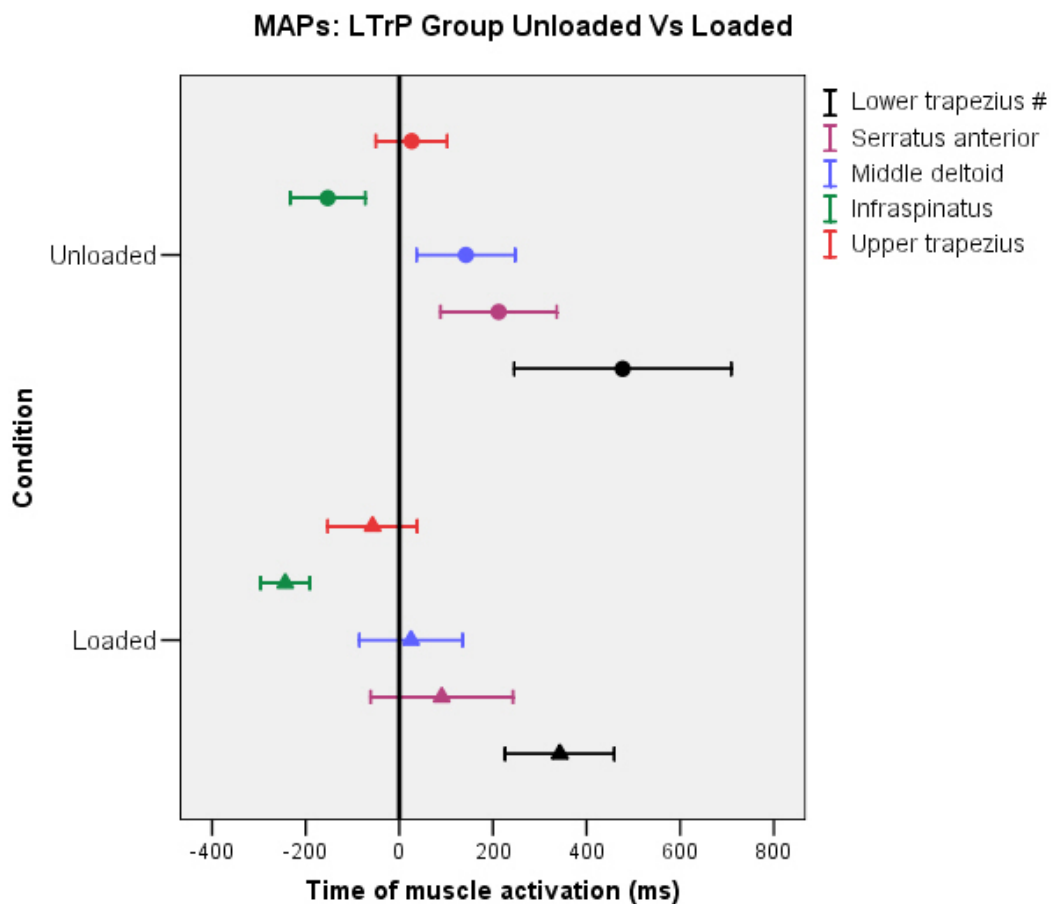
*significant difference in activation time. # significant difference in the variability of activation times (p<0.05)

Figure 4.2.2: The effects of load on the MAP of the control group (mean and SD displayed). Time ‘0’ is the time at which the arm left the side of the body as measured by the microswitch.

4.2.3.2 *LTrP group MAPs (within group comparisons)*

The muscle activation patterns for the LTrP group under both unloaded and loaded conditions are displayed in Table 4.2.1 and Figure 4.2.3. Analyses failed to reveal any changes in the order or the timing of muscle activation in response to load (p>0.05). Though not statistically significant, all muscles

were activated earlier than in the unloaded condition: UT [27ms, -57ms, 84ms]; Inf [-153ms, -244ms, 91ms]; MD [142ms, 25ms, 117ms]; SA [212ms, 91ms, 121ms]; LT [477ms, 343ms, 134ms] (Table 4.2.2). The only significant difference identified was a reduction in the variability for the time at which LT was activated under the loaded condition ($p < 0.05$).



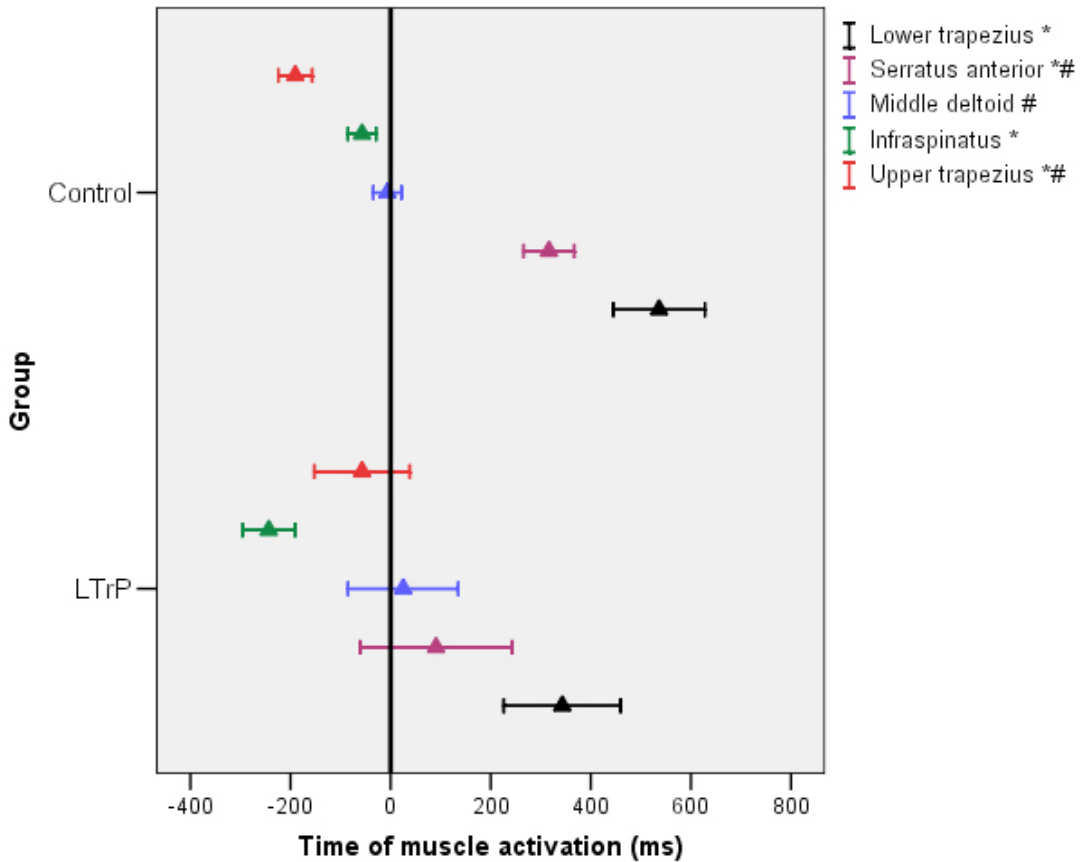
*significant difference in time of activation. # significant difference in variability of activation times ($p < 0.05$).

Figure 4.2.3: The effects of load on the MAP of the LTrP group (mean and SD displayed). Time '0' is the time at which the arm left the side of the body as measured by the microswitch.

4.2.3.3 Controls Vs LTrP group under load (between group comparisons)

It should be remembered that the two groups had already been compared for the unloaded condition (section 4.1), hence the results presented here relate only to comparisons between the groups when a load was applied. Results for activation patterns between the groups when lifting a light weight are presented in Tables 4.2.1 and 4.2.2 and Figure 4.2.4. Under this condition, independent t-tests revealed significant differences for all muscles with the exception of MD. As occurred in the unloaded situation, (see section 4.1.3, p. 155), the LTrP group had a different order of muscle activation with the UT being activated significantly later and the Inf, SA and LT being activated significantly earlier. F statistics revealed significantly more variability for the UT, MD and SA in the LTrP group in the loaded condition, reminiscent of the findings for the unloaded situation (Figure 4.1.4, page 154).

MAPs: Control Vs LTrP Groups with Load



*significant difference in activation times. # significant difference in variability of onset times (p<0.05).

Figure 4.2.4: The effects of LTrPs on MAPs during loaded scapular plane elevation (mean and SD displayed). Time '0' is the time at which the arm left the side of the body as measured by the microswitch.

4.2.4 Discussion of Results

This study investigated the effects of light loads on the MAPs of related shoulder girdle muscles during elevation in the scapular plane in healthy controls and subjects with LTrPs in the scapular rotator muscles. Loads were chosen to represent those that might be lifted during everyday tasks and did not cause subjects undue strain. Under load, the control group demonstrated the same order of muscle activation as they had without load however, all muscles were activated significantly earlier. The UT, Inf and MD were all activated prior to the start of the movement, whereas, only the UT was activated prior to movement start in the unloaded condition. In consequence, it was speculated that when even a light load is imposed, earlier activation is needed to preserve a predetermined movement strategy. The SA and LT were activated 118ms and 214ms earlier respectively in the loaded condition, suggesting that the controlled upward rotation these muscles provide the scapula during the early phases of elevation of the arm, is required earlier with greater external load. Alternatively, earlier activation of muscles in general might be a means of increasing muscle stiffness in anticipation of an applied load that would otherwise result in an unwanted change in scapula position (much in the way the muscles of the lower limb contract in anticipation of foot contact in walking, running or falling (Neumann 2002)). In a study of 16 asymptomatic shoulders, Alpert and colleagues (2000) measured the degree of muscle activation, as opposed to the timing of muscle activation, of the deltoid and rotator cuff muscles in response to various external loads during scapular plane elevation. The authors found that EMG

activity of deltoid, supraspinatus and infraspinatus increased in the 0°-90° range, with peak activity for anterior and middle deltoid, supraspinatus and infraspinatus occurring between 30° and 60° of scapular plane elevation. Furthermore, the change in activity with increasing load was greater between 0-25% and 25-50% of maximum load, than it was for heavier external loads (Alpert et al. 2000). These findings imply that lighter loads (< 50% of maximum) also require significant increases in muscle activation to perform scapular plane elevation, despite the fact that light loads (0-4kgs) have not been found to have any effect on clavicular and scapular kinematics (de Groot 1999; Pascoal et al. 2000), or scapulohumeral rhythm (Doody, Freedman & Waterland 1970; Michiels & Grevenstein 1995). These data and the findings for the control group in the current study, suggest that “healthy” individuals alter the timing and degree of activation of shoulder girdle muscles in order to maintain optimal kinematics and scapulohumeral rhythm when attempting to lift light loads during scapular plane elevation.

The LTrP group demonstrated the same sequence of muscle activation whether the arm was loaded or not, that is Inf activated prior to movement start, the UT approximately as the arm left the side of the body, followed by the MD, SA and LT all after movement start. Reminiscent of the control group responses to load, all LTrP muscles were activated earlier when the arm was loaded, but none significantly so. Perhaps the failure to observe significant changes was due to the large variability in activation times displayed for all LTrP muscles both within and between subjects for both conditions. However, only the LT had a significant change in variability actually having a

more stable activation time under load, a result not likely explained by a learning effect since no other muscle responded in this way.

When comparing the MAPs of the control and LTrP groups during loaded scapular plane elevation (Table 4.2.2), a number of significant timing changes were found: the Inf, SA and LT were activated earlier (187ms, 225ms and 193ms respectively) in the LTrP group, while the UT was activated 134ms later and the MD at approximately the same time in the two groups. In fact, the differences in the activation times between groups were similar when the unloaded and loaded conditions were compared (Table 4.2.2) suggesting that light loads make little difference to the sequence of muscle activation. However, loading was associated with a change in the temporal pattern of activation with all control muscles activated earlier and though not significant, a trend for the same phenomenon was observed in the LTrP group. Interestingly, though the variability between the groups for onset times was significantly greater in all LTrP muscles without load, under loading only three of the five muscles (UT, MD and SA muscle) demonstrated this phenomenon. It seems unlikely that this finding can be explained by increased stability of activation under loaded conditions in the LTrP subjects, but rather, by the fact that variability increased in the control group as well (only UT significantly so) (Figures 4.2.2 and 4.2.4 - see SD bars). As was the case in the unloaded condition, it appears that aside from the consistent early activation of the Inf in the LTrP subjects, the remainder of the MAP was so inconsistent that the addition of external load did not result in additional variability in activation times across the group. Rather than suggesting that

light loads do not adversely affect the MAPs of subjects with LTrPs in the scapular rotator muscles, the results may imply that the presence of LTrPs results in chaotic MAPs whether load is added or not. Supporting this concept, Wadsworth and Bullock-Saxton (1997) found greater variability of muscle activation times in patients with shoulder impingement syndrome compared with healthy controls (Wadsworth & Bullock-Saxton 1997).

4.2.5 Conclusions

In healthy individuals (no LTrPs), performing scapular plane elevation, the sequence of muscle activation in the scapular rotators and related muscles of the shoulder joint, remains relatively stable, but the muscles are activated earlier when light loads are imposed. The presence of LTrPs in the scapular rotator muscles is associated with changes in motor control during scapular plane elevation, but the addition of external loads does not amplify the changes seen in unloaded movement. To reiterate the conclusions of section 4.1, these findings occurred in the absence of pain and clinically, may have implications for overuse syndromes (rotator cuff), shoulder impingement syndrome, and a generally less effective motor control in “overhead” movement.

An additional situation regularly encountered through daily, work and sporting activities is repetitive elevation of the arms, which can bring about a level of

fatigue associated with decreased movement performance. Section 4.3 describes an experiment which investigated the effects of LTrPs in the scapular rotator muscles on the MAP of related shoulder girdle muscles after fatiguing repetitive elevations of the arms in the scapular plane.

4.3 The effect of LTrPs in the scapular rotator muscles on MAPs during elevation in the scapular plane after fatigue.

4.3.1 Introduction

Muscle fatigue resulting from repetitive overload can lead to overuse injuries of the upper limb (Weldon & Richardson 2001) that may result in lost productivity and quality of life (Visser & van Dieen 2006). Initially, muscular fatigue is associated with decreased movement efficiency (Myers, J. B. et al. 1999; Sterner, Pincivero & Lephart 1998) and efficient movement of the upper extremity obviously relies upon the effective coordination of the scapular and shoulder joint muscles to dynamically position the scapula and the arm.

A search of the literature failed to find any investigations into the effects of fatigue on MAPs during scapular plane elevation in normal subjects nor those with LTrPs in the scapular rotator muscles. Given the ubiquity of overuse injuries in the shoulder (second only to low back pain (Langford 1994)), their common association with overhead motion (Blevins 1997; Scoville et al. 1997; van der Hoeven & Kibler 2006) and the fact that LTrPs have the ability to disrupt MAPs (Sections 4.1 and 4.2, pages 152 and 165 respectively), a study was undertaken to elucidate the effects of fatigue on the activation patterns of key muscles of the shoulder girdle during scapular plane elevation. This work was also undertaken to build upon and add to the findings reported in previous sections of this Chapter that have provided

evidence that LTrPs induce significant effects on the timing of muscle contraction, effects that in themselves might be exacerbated or indeed contribute to, muscular fatigue. The results of the current section will be compared to findings reported in sections 4.1 and 4.2.

4.3.1.1 Questions Addressed:

1. Does fatiguing repetitive movement such as may occur commonly in daily activities, alter the normal activation patterns of functionally related muscles during scapular plane elevation?
2. Considering that LTrPs have been shown to alter the timing of muscle activation compared with those who do not have LTrPs (Sections 4.1 and 4.2), will fatigue produce a different pattern of activation in the scapular rotator muscles as well as those more distally placed during scapular plane elevation?

4.3.2 Methods

4.3.2.1 Subjects and Procedures

For this experiment, the same subjects formed the control and LTrPs groups respectively and the post-fatigue sEMG was recorded on the same day, after the unloaded and loaded experiments. The procedures used for this experimental condition were the same as described in section 4.1, but in addition, subjects underwent a fatiguing protocol, then performed three trials

of loaded scapular plane elevation followed by another three trials of this movement unloaded to establish the MAPs of each group after fatiguing movement.

4.3.2.2 *Fatiguing protocol*

In this experiment, control and LTrP subjects were asked to hold a hand-weight (females 1.3kg and males 4kg), while performing scapular plane elevation in time with a metronome set at 60 beats per minute (subjects took four seconds to reach the top of the movement, that is, 160° of elevation moving at approximately 40° per second, and four seconds to return to the starting position), a speed judged to be attainable without resort to anaerobic metabolism (Ogita, Onodera & Tabata 1999). Fatigue was deemed to have occurred when subjects could no longer maintain the cadence. The same relatively light weights were used to duplicate the loaded movement tested earlier (section 4.2), but also to decrease the time taken to fatigue. Subjects were then allowed four seconds rest holding the weights in the starting position to obtain a stable baseline for sEMG and then performed six more trials representing the post-fatigue state, the first three with weights (post-fatigue, loaded), the final three trials without (post-fatigue, unloaded). Again a four second rest was provided between trials (holding the weights for the 'loaded' trials and without any load for the last three trials) to collect baseline sEMG signals (see Figure 4.3.1 for the experimental sequence of sEMG recordings).

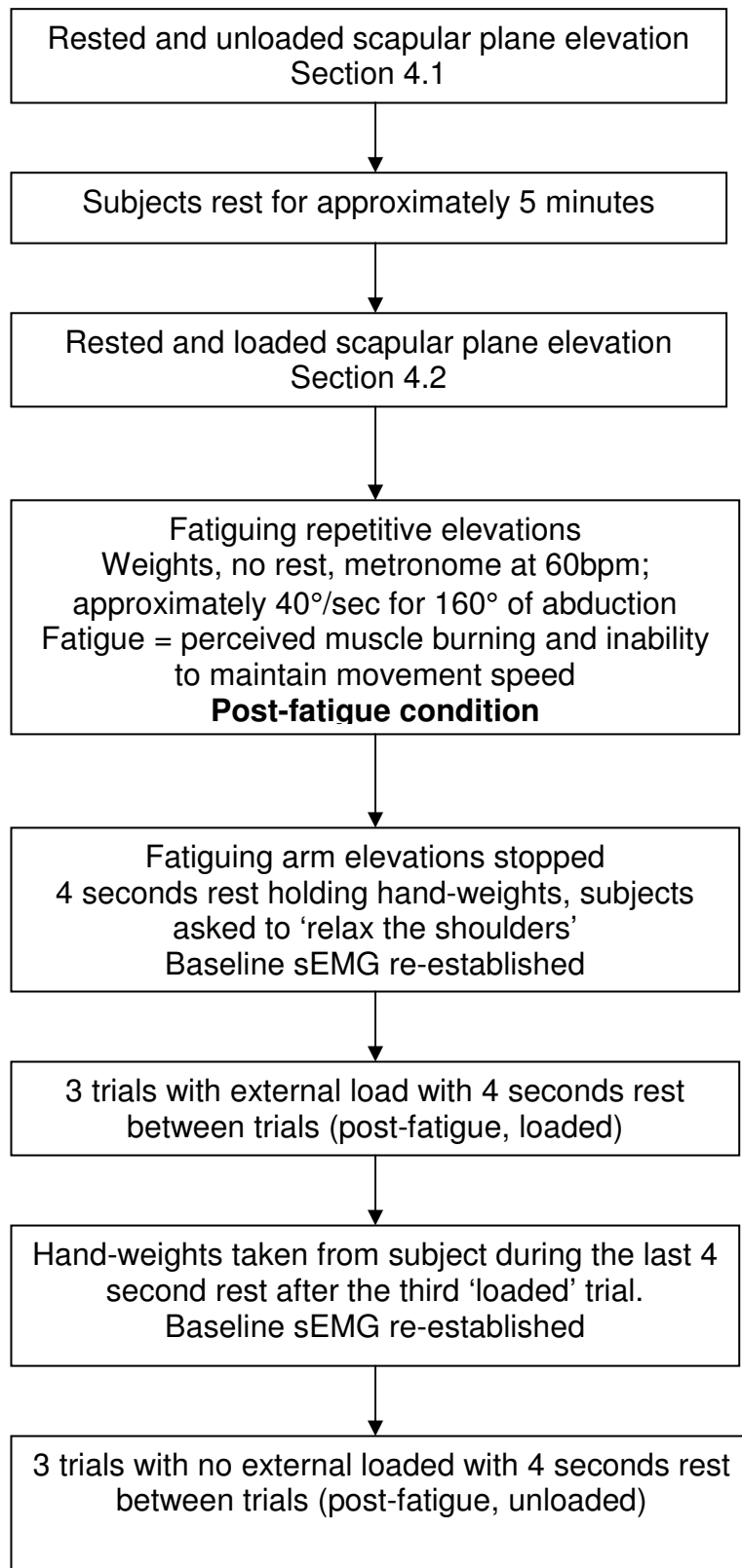


Figure 4.3.1: Experimental sequence and timing of sEMG recordings to test the effects of fatigue on MAPs during scapular plane elevation

4.3.2.3 *Statistical Analysis*

To test for the effects of fatigue on the MAPs during the test movement in both the control and LTrP groups, paired t-tests were employed and the comparison was made with the data collected in the first two experiments (see sections 4.1: rested and unloaded, page 152 and 4.2: rested and loaded page 165), where the same subjects from the control and LTrP groups performed unloaded, then loaded elevation in the scapular plane respectively, prior to the fatiguing protocol. To test for the effects of LTrPs on MAPs during post-fatigue unloaded and loaded scapular plane elevation, independent t-tests were employed to test for differences in the mean activation times of the control and LTrP groups for all muscles. F statistics were once again used to compare the variability in the muscle activation times between conditions or groups and the significance level was set at $p < 0.05$ for all comparisons.

4.3.3 **Results**

4.3.3.1 *Control group MAPs (within group comparisons): unloaded motion*

The muscle activation patterns for the control group subjects under both rested and fatigued conditions without load are displayed in Table 4.3.1 and Figure 4.3.2. Though the order of muscle activation was preserved, except for the UT, paired t-tests revealed significant differences in all other muscle

activation times post fatigue as follows: UT was activated 30ms later and the Inf, MD, SA and LT were activated 95ms, 198ms, 190ms and 437ms earlier respectively (Table 4.3.2). In addition, activation times were significantly more variable for the UT, Inf and MD post-fatigue.

Table 4.3.1: Mean times of muscle activation for the control and LTrP groups for the rested and fatigued conditions with or without load.

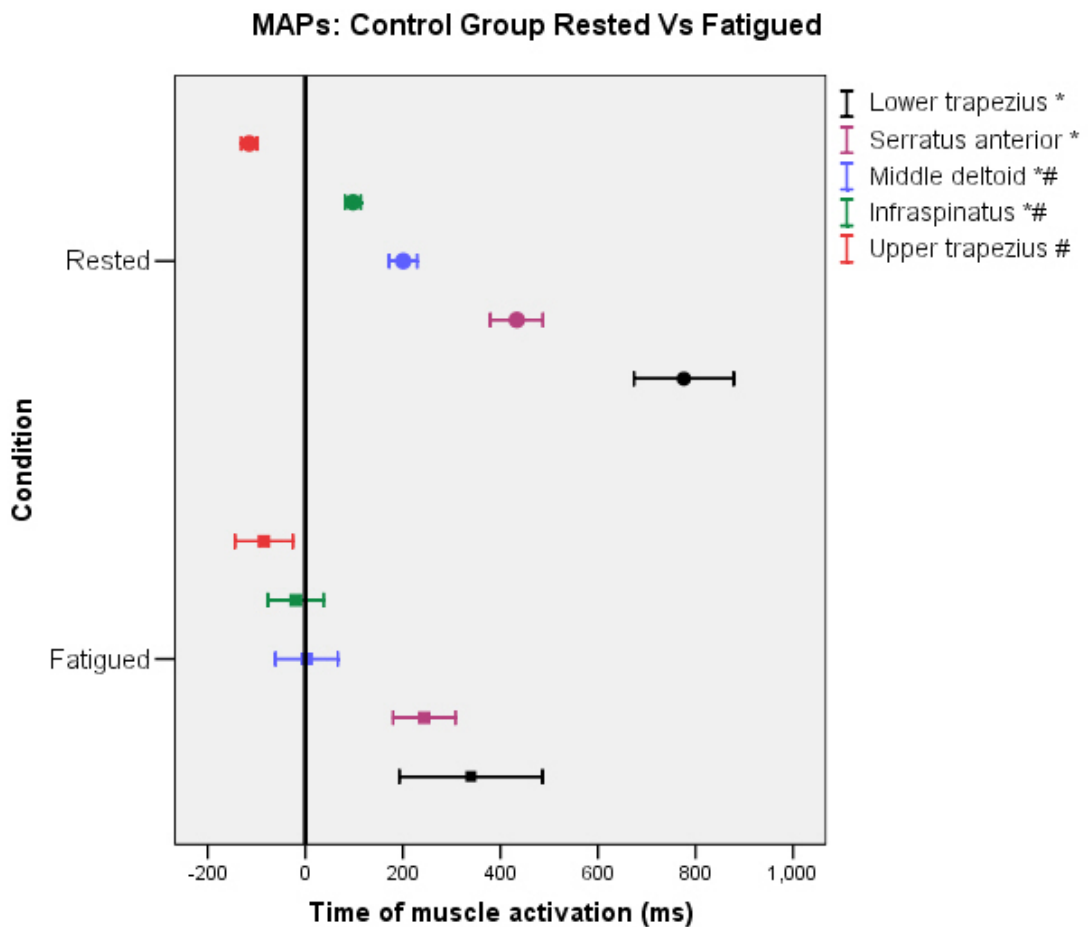
MAPs	UT	Inf	MD	SA	LT
Control rested, unloaded	-115	75	201	434	776
Control fatigued, unloaded	-85	-20	3	244	339
Control rested, loaded	-191	-57	-6	316	536
Control fatigued, loaded	-134	-54	-20	223	363
LTrP rested, unloaded	27	-153	142	212	477
LTrP fatigued, unloaded	30	-54	155	218	541
LTrP rested, loaded	-57	-244	25	91	343
LTrP fatigued, loaded	-41	-149	23	95	276

UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius; '-'=muscle activation time prior to movement start.

Table 4.3.2: Differences in mean activation times between groups, comparing the rested and fatigued conditions, with or without external load.

Differences in activation times	UT	Inf	MD	SA	LT
Difference control rested Vs fatigued, unloaded	+30	-95	-198	-190	-437
Difference control rested Vs fatigued, loaded	+57	+3	-14	-93	-173
Difference control fatigued, unloaded Vs loaded	-49	-34	-17	-21	-24
Difference LTrP rested Vs fatigued, unloaded	+3	+99	+13	+6	+54
Difference LTrP rested Vs fatigued, loaded	+16	+95	-2	+4	-67
Difference LTrP fatigued, unloaded Vs loaded	-71	-95	-132	-123	-265
Difference control Vs LTrP, fatigued, unloaded	+115	-34	+152	-26	+202
Difference control Vs LTrP, fatigued, loaded	+93	-95	+43	-128	-87

UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius, ‘-‘=muscle activated earlier in the group named second (fatigued condition or in the LTrP group compared with the control group respectively); ‘+‘=muscle activated later in the group named second (fatigued condition or in the LTrP group compared with the control group respectively).



*significant difference in activation times. # significant difference in the variability of activation times ($p < 0.05$).

Figure 4.3.2: Normal MAP rested Vs fatigued with no load during scapular plane elevation (mean and SD displayed). Time '0' is the time at which the arm left the side of the body as measured by the microswitch.

4.3.3.2 Control group MAPs (within group comparisons): loaded motion

When the load was added, subjects continued to recruit the muscles in the same order, but the UT was activated significantly later (57ms) and the SA (93ms) and LT (173ms) were both activated significantly earlier. The Inf and MD showed no difference in activation times post-fatigue. Variability in

activation times was unchanged by fatigue (Tables 4.3.1 and 4.3.2 and Figure 4.3.3).

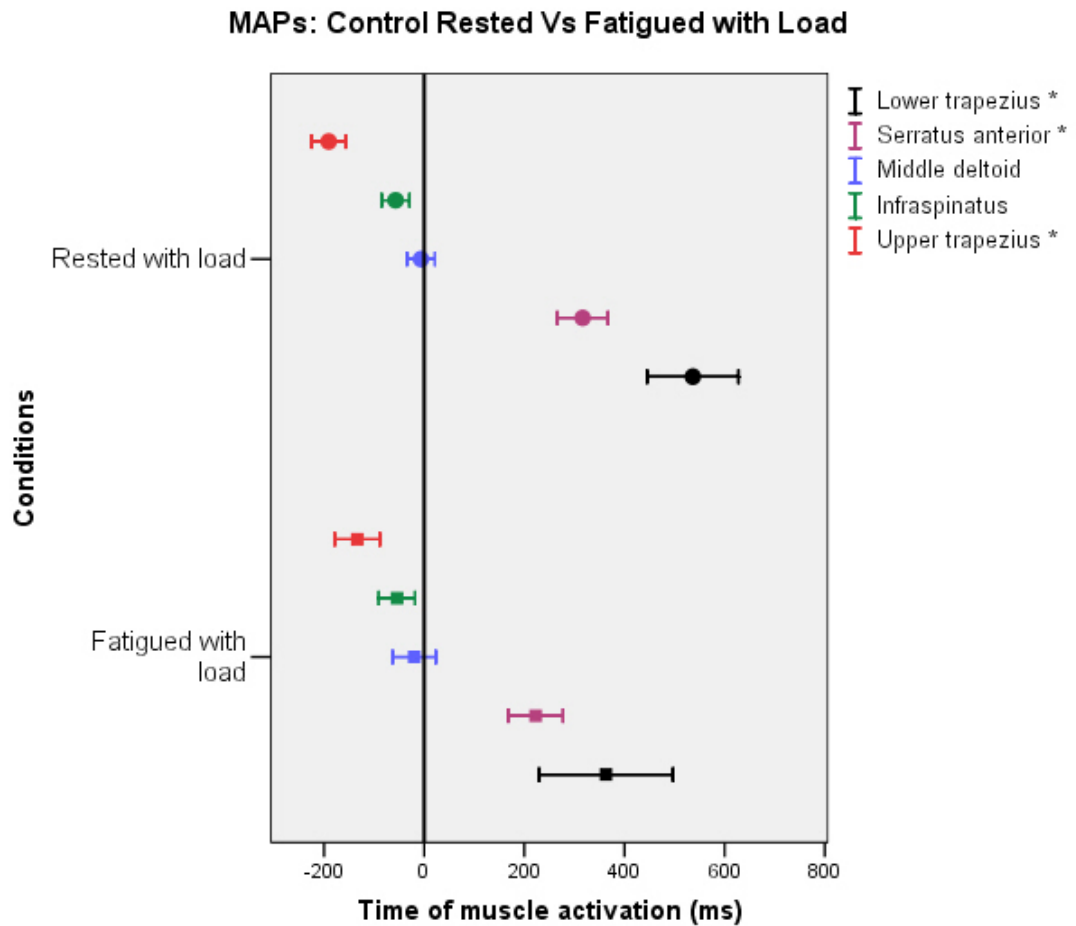


Figure 4.3.3: Normal MAP rested Vs fatigued during loaded scapular plane elevation (mean and SD displayed). Time '0' is the time at which the arm left the side of the body as measured by the microswitch.

4.3.3.3 LTrP group MAPs (within group comparisons): unloaded motion

For the LTrP group, as was observed with controls under the same conditions, the order of activation was the same as the rested state (Inf, UT,

MD, SA, LT), however, the Inf was activated significantly later (99ms) though still prior to movement start (Table 4.3.1 and Figure 4.3.4). Variability of muscle activation times was not changed by fatigue when the movement was performed without load.

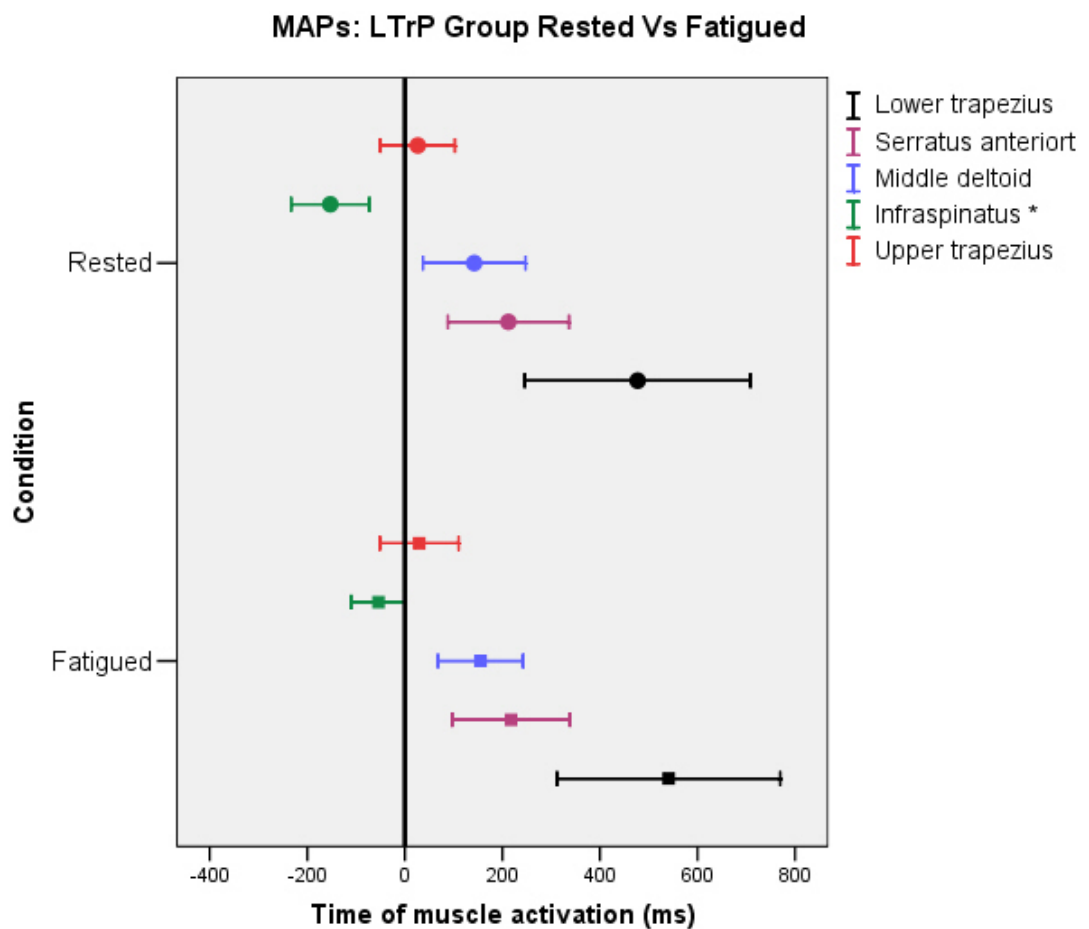
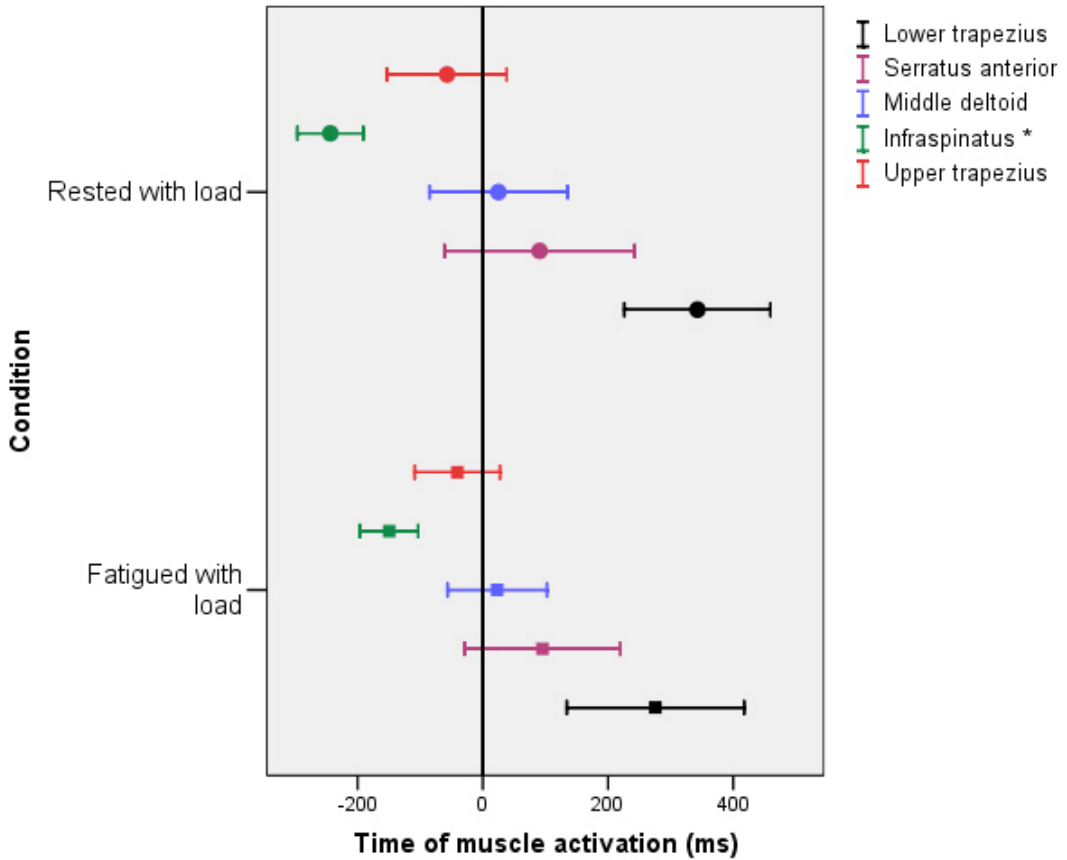


Figure 4.3.4: LTrP MAP rested Vs fatigued with no load during scapular plane elevation (mean and SD displayed). Time '0' is the time at which the arm left the side of the body as measured by the microswitch.

4.3.3.4 *LTrP group MAPs (within group comparisons): loaded motion*

When loaded motion was performed post-fatigue, for LTrP subjects, the sequence of activation was the same as found in both the rested state and the post-fatigue unloaded trial. The only difference observed in sequencing was the later activation of Inf (95ms) in the fatigued state. Large standard deviations in activation times were observed under both conditions (rested and loaded compared with fatigued and loaded) for this group, but the differences were not significant. There was also some 'condensation' or 'compression' of the MAP post-fatigue for the LTrP group with the whole sequence completed in a shorter time span (on average, 587ms rested with load Vs 425ms fatigued with load), that is the first muscle (Inf) was activated later (95ms) and the last muscle (LT) was activated earlier (67ms) for this group post-fatigue (Tables 4.3.1 and 4.3.2 and Figure 4.3.5).

MAPs: LTrP Group Rested Vs Fatigued with Load



*significant difference in activation time. # significant difference in variability of activation times ($p < 0.05$).

Figure 4.3.5: LTrP MAP rested Vs fatigued during loaded scapular plane elevation (mean and SD displayed). Time ‘0’ is the time at which the arm left the side of the body as measured by the microswitch.

4.3.3.5 Controls Vs LTrP group post-fatigue (between group comparisons): unloaded motion

A comparison between the control and LTrP MAPs during unloaded scapular plane elevation revealed two different muscle activation sequences post-fatigue. The control group sequence was UT then Inf both prior to movement start, MD as the movement started, then SA and LT after movement start,

whereas the LTrP group activated the Inf prior to movement start, the UT just after movement start, then the MD, SA and finally the LT (table 4.3.1 and Figure 4.3.6). With regard to the mean times of muscle activation between groups, the UT and MD muscles were activated significantly later in the LTrP group (115ms and 152ms respectively, Table 4.3.2) and only the SA was significantly more variable in activation time in the LTrP group. The control group MAP was more condensed than the LTrP group MAP (424ms Vs 595ms; time taken for all muscles to become activated).

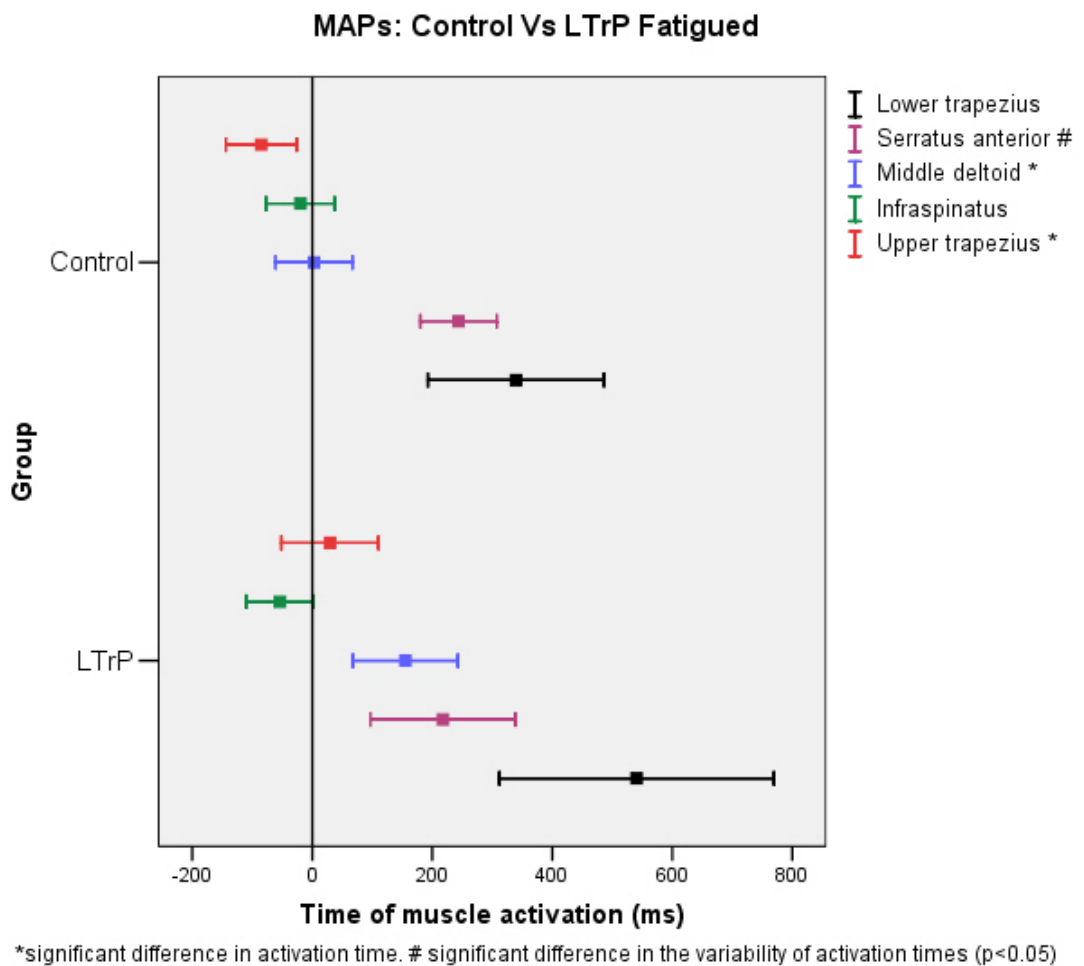


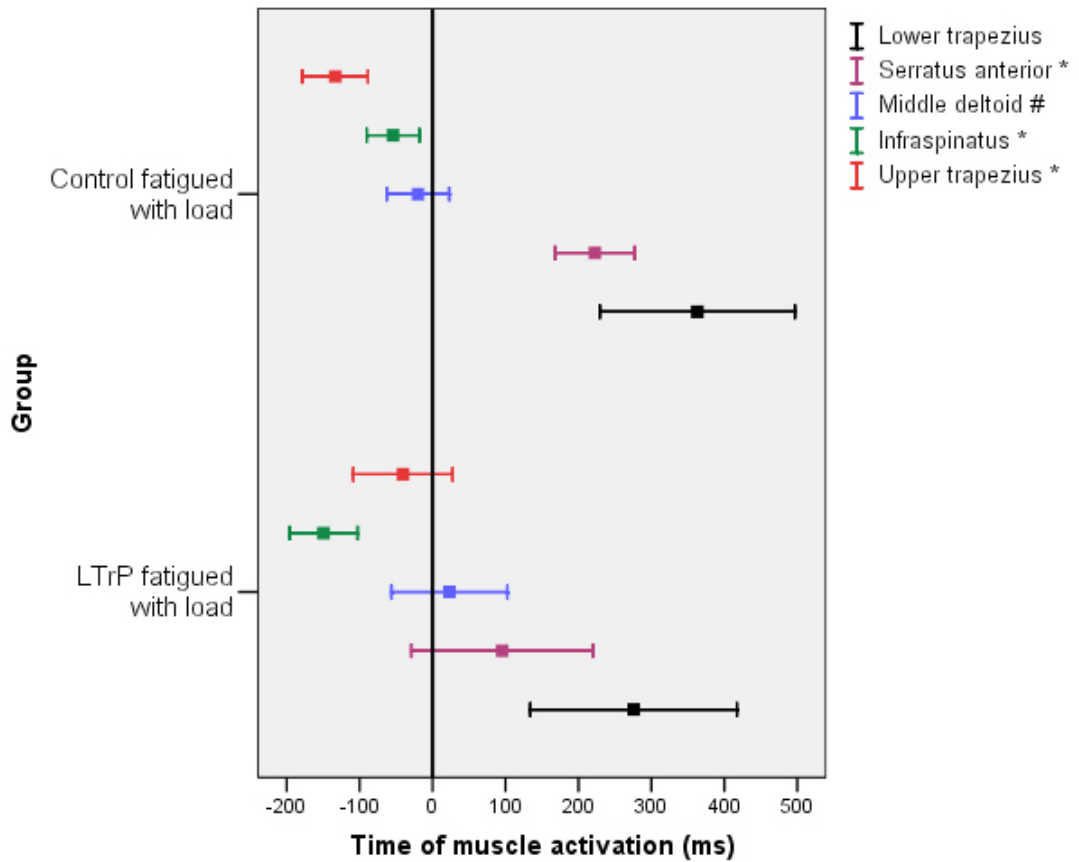
Figure 4.3.6: Comparison of normal to LTrP MAPs post-fatigue with no load during scapular plane elevation (mean and SD displayed). Time '0'

is the time at which the arm left the side of the body as measured by the microswitch.

4.3.3.6 *Controls Vs LTrP group post-fatigue (between group comparisons): loaded motion*

Comparisons between the control and LTrP groups for post-fatigue loaded scapular plane elevation revealed two different activation sequences. The control group sequence was UT then Inf then MD prior to movement start, then SA and LT after movement start, whereas the LTrP group activated the Inf then UT prior to movement start, the MD just after movement start, then the SA and finally the LT after movement start (Table 4.3.1 and Figure 4.3.7). With regard to the mean times of muscle activation, the UT was activated significantly later (93ms) and the Inf and SA muscles were activated significantly earlier (95ms and 128ms respectively, Table 4.3.2) in the LTrP group and only the MD (in LTrP group) showed significantly more variation in time of activation. The total time taken for all muscles to become active was similar for both groups in the loaded post-fatigued state (497ms Vs 425ms respectively).

MAPS: Control Vs LTrPs, Fatigued with Load



*significant difference in activation times. # significant difference in variability of activation times ($p < 0.05$).

Figure 4.3.7: Comparison of normal to LTrP post-fatigue during loaded scapular plane elevation (mean and SD displayed). Time '0' is the time at which the arm left the side of the body as measured by the microswitch.

4.3.4 Discussion of results

This study investigated the effects of fatigue caused by repetitive loaded elevations in the scapular plane, on the MAPs of related shoulder girdle muscles during the same movement in healthy controls and subjects with

LTrPs in the scapular rotator muscles. Muscle activation patterns were recorded post-fatigue under two conditions: unloaded and loaded (females 1.3kg and males 4kg), thought to represent loads that individuals might experience during home and work related tasks and similar to loads applied in previous kinematic studies (de Groot, van Woensel & van der Helm 1999; Pascoal et al. 2000) and studies investigating the effects of load on the scapulohumeral rhythm (McQuade & Smidt 1998). Fatigue was defined as an inability to maintain a cadence of 40°/second over a 160° range of elevation in the scapular plane (4 beats or seconds going up, then 4 beats/seconds coming down), and in all cases, was accompanied by subjective reports of varying degrees of a 'muscle burning' sensation. This definition of fatigue was associated with a decreased efficiency of movement rather than an inability to move at all, and was chosen to represent a level of fatigue that individuals might experience during common daily tasks.

4.3.4.1 Effects of fatigue on MAPs of healthy subjects

In the current study, the order of muscles activated post-fatigue in the control group was the same as in the rested state, however the timing of activation was condensed, that is, the UT was activated slightly later (not significant) and the remaining muscles were activated significantly earlier, perhaps in an attempt to increase scapulothoracic motions in relation to glenohumeral motions, in order to maintain the congruity of the glenohumeral joint and preserve the subacromial space, as supported by previous studies as

follows. Two kinematic studies found that shoulder muscle fatigue resulted in increased upward rotation of the scapula, although one study found twice the increase in this motion (Ebaugh, McClure & Karduna 2006) as the other (McQuade, Dawson & Smidt 1998), possibly due to the former study employing a fatiguing protocol of longer duration than the latter. Conversely, two further studies reported decreased upward scapular rotation after shoulder muscle fatigue (McQuade, Hwa Wei & Smidt 1995; Tsai, McClure & Karduna 2003), though the different fatiguing protocols and small and different populations used, make direct comparisons problematic. External rotation of the scapula, defined in their work as the lateral border moving posteriorly, was increased in the Ebaugh et al. study (2006) but decreased in the Tsai et al. study (2003) after fatigue. Possible explanations for these contrary findings may lie in the specific fatiguing of the humeral external rotators by Tsai and colleagues, whereas Ebaugh et al. achieved generalised shoulder fatigue, suggesting that patterns of altered scapular kinematics may be dependent on the group or groups of muscles fatigued.

As discussed in section 4.1 (page 154), it appeared that optimal timing of activation of the UT, Inf and MD muscles was important in the initiation of scapular plane elevation in healthy subjects. Post-fatigue, these three muscles had significantly more variability in their timing of action, implying that when fatigued, healthy subjects may have initiated elevation in the scapular plane with decreased efficiency, with possible implications for maintaining favourable subacromial space. Scapulothoracic kinematic changes such as increased upward and external rotation may be viewed as

an attempt to optimise the relationship between the glenoid fossa, coracoacromial arch and the humeral head to compensate for a decrease in external rotation of the humerus, preserving the subacromial space and decreasing pressure of the humeral head on the subacromial structures (Kibler, W. B. & McMullen 2003; Ludewig & Cook 2000). Ebaugh and colleagues (2006) proposed that increased scapulothoracic motions were a response to altered glenohumeral motion, believed to be a direct result of fatigue in the external rotator muscles of the humerus in their study (Ebaugh, McClure & Karduna 2006). Furthermore, when light external load was added by asking subjects to hold hand-weights in the current study, UT was activated significantly later and SA and LT were activated significantly earlier, resulting in an even more condensed MAP when healthy subjects were loaded and fatigued, though surprisingly, no differences were found in the variability of activation times in this state. One further factor complicating any comparisons between studies is the fact that in the present work, external rotation of the shoulder was eliminated by using the movement guides, hence the muscle roles may have been different where “uncontrolled” scapular plane elevation was carried out.

4.3.4.2 *Neural mechanisms associated with fatigue in healthy subjects*

Since this was not a mechanistic study, the descriptions below have been necessarily brief and are meant to encourage discussion and further experimental work in this area. In an attempt to explain the neural mechanism(s) that may underpin the effects of fatiguing repetitive movements on MAPs of healthy subjects during elevation of the arm, an understanding of the motor control systems is provided. Recall that a motor command for movement is initiated at high levels of the CNS and this command is processed at progressively lower levels of the CNS and finally at segmental levels in the spinal cord, where it is resolved into the muscles and motor units that are recruited to action for the proposed movement. Output from the CNS occurs through the final common pathway (co-activation of the α and γ motoneurons) and determines the level of activation of individual muscles (Rothwell 1994). Feedback for the system occurs through muscle receptors and their afferent fibers, that is, muscle spindle Ia and II fibers, Golgi tendon organ (GTO) group Ib fibers, Group II non-spindle fibers (low threshold mechanoreceptors), group III and IV fibers (high threshold mechanoreceptors and nociceptors sensitive to algescic substances and ischemia) and in turn, these affect neuromuscular control and joint function (Myers, J. B. et al. 1999). As fatigue develops, extracellular metabolites accumulate and pH decreases in the contracting muscles (Fischer, M. & Schafer 2005; Windhorst et al. 1997). With regard to the effects of this tissue state, the proposal of Myers and colleagues (1999) and the thoughts of Mense (1997) might provide insight. In combination, these authors suggested

that when muscles fatigue, intramuscular afferents responsible for providing proprioceptive feedback (Myers, J. B. et al. 1999) and warning the CNS that where tissues may be approaching structural or functional limits (Mense 1997), are stimulated and alter neuromuscular responses reflected in MAPs. Broadly speaking, this may occur through two neural mechanisms: one affecting central commands and generating “sub-optimal” descending signals and a second operating through spinal cord reflexes, as discussed below.

4.3.4.3 *Central and Sub-Optimal Descending Commands*

A number of authors have suggested that group III and IV muscle afferents from nociceptors are stimulated secondary to fatigue and might act supraspinally to impair voluntary descending drive, inhibiting activation of affected α -motoneurons, though the mechanism by which this occurs has not been proven as yet (Gandevia 2001; Taylor et al. 1996; Taylor, Todd & Gandevia 2006). In addition, Renshaw cells may play a role in this process. These neurons are stimulated by collateral branches from the axons of α -motoneurons and inhibit “their own” α -motoneurons, (“recurrent inhibition”) as well as other α -motoneurons. However, they also receive direct supraspinal input that facilitates their inhibitory actions (Gandevia 2001) and through their wider projections, Renshaw cells have the capacity to influence the α and γ motoneurons of homonymous and heteronymous muscles, the Ia inhibitory interneuron and other Renshaw cells (Rothwell 1994). Because of these connections, supraspinal input to Renshaw cells, may provide a mechanism

through which group III and IV inputs can influence MAPs and motor control during muscle fatigue.

4.3.4.4 *Spinal cord reflexes*

(i) *Facilitation of α -motoneurons secondary to group III and IV stimulation of fusimotor drive*

In the event that central commands and descending signals remain unaltered from the proposed motor program initiated at higher CNS centres, these commands can arrive at the appropriate spinal segment where group III and IV afferent input has increased motoneuron excitability, possibly via the following mechanism/pathway (Gandevia 2001). As muscle fatigues, nociceptors are activated by the fatigue-induced accumulation of metabolites as well as increased acidity leading to group III and IV afferent discharge. These afferents synapse with γ -motoneurons and excite predominantly static gamma efferents that cause contraction of spindle intrafusal fibres and increased spindle primary (Ia) and secondary (II) afferent firing, resulting in facilitation of both α and γ motoneurons (Appelberg et al. 1982, 1983; Djupsjobacka, Johansson & Bergenheim 1994; Djupsjobacka, M et al. 1995; Djupsjobacka, M. et al. 1995; Fischer, M. & Schafer 2005) (Figure 4.3.8). Accordingly, the Ia excitatory input to the homonymous and heteronymous α -motoneurons results in facilitation of agonist and synergist muscles. The stimulation of the homonymous and heteronymous γ -motoneurons by Ia and II spindle discharge feeds back in to the fusimotor system and drives the

continued facilitation of motoneurons supplying these muscles.(Gladden, Jankowska & Czarkowska-Bauch 1998). As Rothwell (1994) explains, the degree of γ activation dictates the degree of 'stiffness' of the muscle spindle and therefore its mechanical sensitivity and subsequent level of spindle afferent input into the CNS. In addition, the spindle Ia fiber synapses with the Ia inhibitory interneuron, which results in inhibition of the antagonist muscle as the agonist is contracting (Rothwell 1994) and acts to facilitate movement. With regard to the MAPs for healthy subjects in the current study, in the unloaded condition, all muscles were activated significantly earlier aside from the UT, however, in the loaded state only SA and LT activation was earlier than when subjects were rested. Although the level of muscle activation (signalled by sEMG amplitude) is obviously different from the timing of muscle activation, these phenomena could have affected each other due to the criteria used to identify the time of muscle activation in the current study. For example, an increased α -motoneuron firing rate (due to increased fusimotor drive secondary to group III and IV afferent input) may have resulted in a sEMG amplitude that was large enough to reach one SD greater than the baseline mean amplitude more quickly, potentially resulting in the muscle being identified as 'active' sooner (e.g. Inf, MD, SA and LT in the unloaded, post-fatigue state in the current study). Conversely, a decrease in α -motoneuron firing rate (for example, due to recurrent inhibition by Renshaw cells) would probably necessitate both increased temporal and spatial summation to reach a certain force output, potentially increasing the time needed to activate motor units and producing an apparent delay in the activation times (e.g. UT in the unloaded, post-fatigue state in the current

study). Furthermore, different muscles may have fatigued at different rates leading to varying amounts of interstitial concentrations of nociceptor activating substances present at any given moment resulting in varying amounts of fusimotor drive to α -motoneurons and producing the increased variability of muscle activation seen in control group subjects post-fatigue.

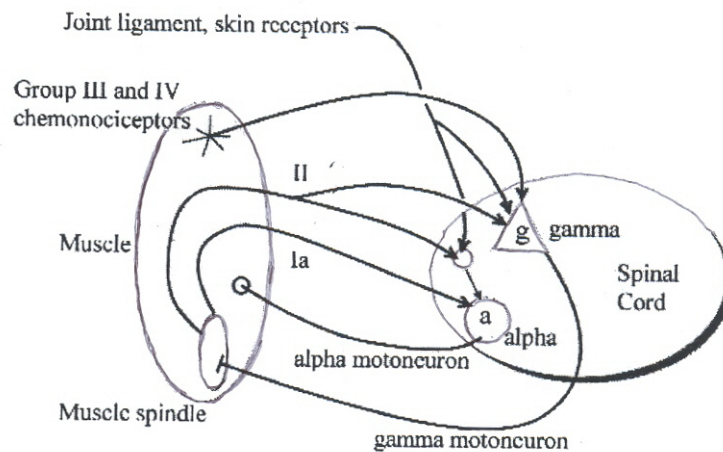


Figure: 4.3.8: Gamma motor positive feedback loops. Reproduced from Knutson (2000).

(ii) *Inhibition of α -motoneurons secondary to group III and IV afferent discharge*

As outlined above, group III and IV muscle afferent input can lead to reflex activation of γ -motoneurons (Djupsjobacka, Johansson & Bergenheim 1994; Djupsjobacka, M. et al. 1995), producing fusimotor drive that facilitates and provides ongoing support for contraction of the agonist and synergistic

muscles and inhibits the antagonist(s) (Hagbarth et al. 1986; Rothwell 1994; Sjolander & Johansson 1995). However, it has been suggested that muscle activation is often inhibited secondary to fatigue and/or pain, when group III and IV nociceptors have become sensitised rather than just activated due to short-term exposure to metabolites). Evidence for this phenomenon was demonstrated by Rossi and colleagues (1999) who chemically induced tonic muscle nociceptive discharge from the extensor digitorum brevis muscle and produced a decrease in the size of the monosynaptic Ia excitatory post-synaptic potentials of soleus motoneurons (Rossi, Decchi & Ginanneschi 1999). In an attempt to explain this result, Thunberg and co-workers (2002), suggested that the process may occur via presynaptic inhibition of the Ia terminals in association with group III and IV afferent input, implying that where fusimotor drive or other reflex effects had enhanced the excitability of α -motoneurons, the effectiveness of the connection between the primary spindle afferent and α -motoneurons could be reduced, ultimately resulting in decreased α -motoneuron discharge (Thunberg et al. 2002). However, Rossi's group did not establish whether the presynaptic inhibition occurred via segmental or supraspinal pathways nor whether the muscle nociceptive volley directly facilitated these presynaptic pathways or removed some tonic inhibition of them (Rossi, Decchi & Ginanneschi 1999). In relation to the current study, this process may have contributed to findings of delayed or variable muscle activation which were observed in the UT (significantly delayed activation when fatigued with external load) and the UT, Inf and MD (significantly more variable in activation time post-fatigue) in healthy subjects.

In addition to the potential for pre-synaptic inhibition of the Ia terminals to inhibit motoneurons, the intrinsic properties of the α -motoneurons themselves can also be modulated by group III and IV input secondary to fatigue. Windhorst and co-workers (1997) substantiated this phenomenon in a study on decerebrate and “mostly” spinalised cats exposed to intra-arterial injections of BK and 5-HT (serotonin). They found that group III and IV afferent reflex action on afterhyperpolarisation (AHP) of motoneurons (an intrinsic property of motoneurons associated with inhibition) may occur through “classical transmitters” altering motoneuron inputs or by altering α -motoneuron responsiveness through modulation of intrinsic properties, including AHP or altering ionic conductance via neurotransmitters, neuromodulators or “neuro-active peptides” (Windhorst et al. 1997). With regard to the control group subjects affected by fatigue in the current study, the UT was activated later, possibly indicating inhibition of this muscle, in both the unloaded (non-significant) and loaded states. Given more muscles in the control group responded with earlier activation, this may suggest that with relatively low levels of fatigue (defined in this study as an inability to perform arm elevations at a required speed as opposed to neuromuscular fatigue), the initial neural response may be one of facilitation of motoneurons. Perhaps because UT was the first muscle active in the sequence of muscle activation, it fatigued more (being active for more of the movement with less opportunity for dispersal of metabolic products) (Taylor, Butler & Gandevia 2000). It was the only muscle that showed signs of inhibition (if that is what can be interpreted from the responses), perhaps demonstrating that as fatigue increases in dynamic movements, inhibition of motoneurons may

begin to occur. It should be emphasized that duration of muscle activation was not measured in the current study so such an explanation would require further analysis of the signals.

It should be recalled that the current study was not designed to investigate mechanisms underpinning fatigue, hence the previous section was prepared as a brief review of this topic to allow some speculation on the findings of the control group post-fatigue. In fact, according to Taylor and co-workers (2000), mechanisms that result in the alteration of motoneuron firing associated with fatigue can apparently, be contributed to by any of the following processes:

- Presynaptic inhibition of Ia muscle spindle afferent discharge.
- Changing patterns of transmission from afferents to motoneurons reflex.
- Inhibition or disfacilitation in response to altered afferent input.
- Changes in the descending drive to the motoneuron pool.
- Changes to the intrinsic properties of the motoneurons.
- Recurrent (Renshaw cell) inhibition.

However, the complex interactions and exact involvement of each of these elements in any given circumstance remains a topic for ongoing investigation (Taylor, Butler & Gandevia 2000). In a concluding remark, Taylor and colleagues (2000) suggested that although group III and IV afferents are activated and sensitised in fatigued and ischemic muscle, other afferents, from Golgi tendon organs or those group III and non-spindle group II afferents which respond when muscles contract, could be used to moderate voluntary drive to respond to variations in force output from the muscle

(Taylor, Butler & Gandevia 2000), These supraspinal effects represent yet another aspect of fatigue relevant to explaining the mechanisms by which healthy subjects alter their MAPs in response to fatigue.

In summary, the work cited suggests that changes to muscle afferent discharge in response to fatigue may alter α -motorneuron activity directly (via spinal cord reflexes at the relevant spinal segmental level) or indirectly at supraspinal sites, altering both the descending command to α -motoneurons and their intrinsic properties in healthy subjects. The next section discusses the MAPs of the LTrP group post-fatigue and the potential effects and underpinning mechanisms.

4.3.4.5 Effects of fatigue on MAPs of LTrP subjects

For the LTrP group, fatiguing arm elevations in the scapular plane caused only a minor alteration in the MAPs compared with the pre-fatigue trials, regardless of whether the movement was performed unloaded or loaded, that is, the Inf muscle was activated significantly later post-fatigue. There were no significant differences in the variability of muscle activation times induced by fatigue, perhaps due to the already large standard deviations (representing variability), of activation times that existed in the rested states associated with the very inconsistent order of muscle activation these subjects employed in either state. When unloaded and compared with the control group, the

LTrP subjects activated the UT (115ms) and the MD (152ms) later when fatigued, potentially compromising the subacromial space and placing the subacromial structures at increased risk of compression, inflammation or ultimately, degeneration over time. Perhaps in an adaptive role aimed at minimising subacromial compression (due to the delayed activation of UT) and helping to initiate abduction (due to the late activation of MD), the Inf was activated earlier in LTrP subjects (compared with controls) in fatigue (non-significant compared with controls), though not as early as at rest where the difference in activation time was significantly earlier compared to both the control group at rest and their own results post-fatigue. It appears that fatiguing activity altered the strategy used by the LTrP group at rest, where the earlier activation of Inf may have been in an attempt to optimise glenohumeral joint mechanics. If the acromion was not elevated early in the movement and the glenohumeral kinematics are altered, when fatigued the Inf was perhaps less capable of performing this role (indicated by its later activation in fatigue in LTrP subjects). As discussed above in relation to healthy subjects, the earlier activation of Inf might be explained by reference to fatigue-induced inhibitory responses mediated by group III and IV afferents via several avenues: sub-optimal descending commands; recurrent inhibition via facilitation of Renshaw cells; pre-synaptic inhibition of spindle Ia afferent terminals or altered intrinsic properties of the affected motoneurons (Bigland-Ritchie, B. R. et al. 1986; Garland 1991; Woods, Furbush & Bigland-Ritchie 1987). With the addition of light loads, though the mean times of activation were generally earlier (as was the case when subjects were rested and loaded, Section 4.2, Pages 168-171), the order of activation remained the

same as the rested state for both the control and LTrP groups. This result suggests that the overall strategies (perhaps preserving subacromial space in the control group (optimal)) or reacting to sub-optimal glenohumeral/scapulothoracic kinematic interactions in the LTrP) employed prior to fatigue remained in force, but muscles were activated earlier to generate the increased force required to lift the added mass (Neumann 2002).

Interestingly, the only significant change brought about by repetitive fatiguing arm elevations for the LTrP group (within group comparison) was that Inf was activated later than when rested (but still earlier than in the control group fatigued). Given that Inf was LTrP-free, its delayed activation post-fatigue was most likely due to a decreased ability to adapt to the inefficient glenohumeral joint mechanics suggested to exist in LTrP subjects or an inhibitory effect of LTrPs located in functionally related muscles (Eg: UT, SA or LT). The relatively repeatable responses of the LTrP-containing muscles (UT, SA and LT) whether at rest or post-fatigue, suggests that either fatigue has less effect on the MAPs of subjects with LTrPs in the scapular rotator muscles or that LTrPs produce a fatigue-like MAP in the rested state that fatigue does not appreciably alter. The results of Chapter five (Section 5.3, page 246) will elucidate which of these two suggestions represents the more feasible explanation.

4.3.5 Conclusions

1. For healthy subjects, fatigue is associated with a ‘condensing’ of the MAP where the muscles activated subsequent to the UT were activated earlier and increased variability of activation times compared with the rested state.
2. Fatigue induces very little change to the MAP of LTrP-sufferers beyond those generated during “rested movement”.

As described in Sections 4.1, 4.2 and 4.3, the presence of LTrPs in the scapular rotator muscles was associated with a number of effects on MAPs during scapular plane elevation under conditions that occur commonly in daily life. In an attempt to establish a cause and effect relationship between LTrPs and altered MAPs, the next chapter reports on the effects of ‘removing’ LTrPs from the scapular rotator muscles on MAPs recorded under the same experimental conditions (unloaded, loaded, fatigued).

CHAPTER 5

THE EFFECTS OF TREATING LTrPs ON MAPS DURING SCAPULAR PLANE ELEVATION

Overview of Chapter

This chapter presents and discusses the MAPs derived from the test muscles of LTrP subjects during scapular plane elevation under each of the conditions previously described (unloaded, loaded, fatigued) but after either LTrP treatment or no treatment (sham treatment). The experiment was designed to establish what happens to the MAPs of the muscles under investigation, when LTrPs are “removed” from affected muscles and thereby strengthen any conclusions drawn about their effects on motor control. To revise the experimental sequence, a flow diagram from Chapter four is again presented below (Figure 5.1.1) and briefly described next. All MAP data were derived from the same 42 subjects who participated in the studies presented in Chapter four, where the 14 members of the control group had undergone sEMG of the scapular upward rotators and related shoulder muscles as previously described (Chapter 4), and in so doing, provided normative MAPs for these muscles during scapular plane elevation under unloaded, loaded and fatigued conditions. The LTrP group (N=28) underwent the same protocols as the control subjects (Chapter 4) so that comparisons of their MAPs with controls could be made. The current chapter describes the results of the same protocols performed on the LTrP subjects after random assignment to one of two interventions: ‘treatment’ of LTrPs represented by superficial dry needling (SDN) followed by post-isometric relaxation (PIR) stretching applied to all muscles in which LTrPs had been identified (n=14), or “placebo treatment” in which sham ultrasound was employed leaving LTrPs ‘intact’ (N=14). It should be emphasised that the entire experimental

program for any given subject (experiments described in chapters 3 – 5) was carried out on the same day, including the initial screening for ‘normal’ shoulder girdles; examination for LTrPs, sEMG for the various conditions of scapular plane elevation (unloaded, loaded, fatigued) and subsequently the interventions for LTrP subjects (treatment or sham) followed by a repetition of the various shoulder elevation protocols. The entire process took approximately 90 minutes per subject.

The effects of “removing” LTrPs on MAPs were determined by a number of comparisons:

1. MAPs in treated versus untreated LTrP subjects under each of the scapular plane elevation conditions.
2. MAPs in treated LTrP subjects versus controls (Chapter 4)

It was hypothesised that “removing” LTrPs (using a recognised treatment) would change the timing of muscle activation in these subjects towards the patterns observed in “normals” thereby providing strong evidence that the MAPs observed in these subjects were indeed the result of LTrPs. The format of this chapter is the same as used in Chapter four with regard to the three experimental conditions under which MAPs were investigated, that is unloaded (Section 5.1), with external load in the hand (Section 5.2) and after fatiguing arm elevations (Section 5.3). As in Chapter four, each section has its own introduction, description of methods, results, discussion and conclusions to minimise the need for constant cross referencing.

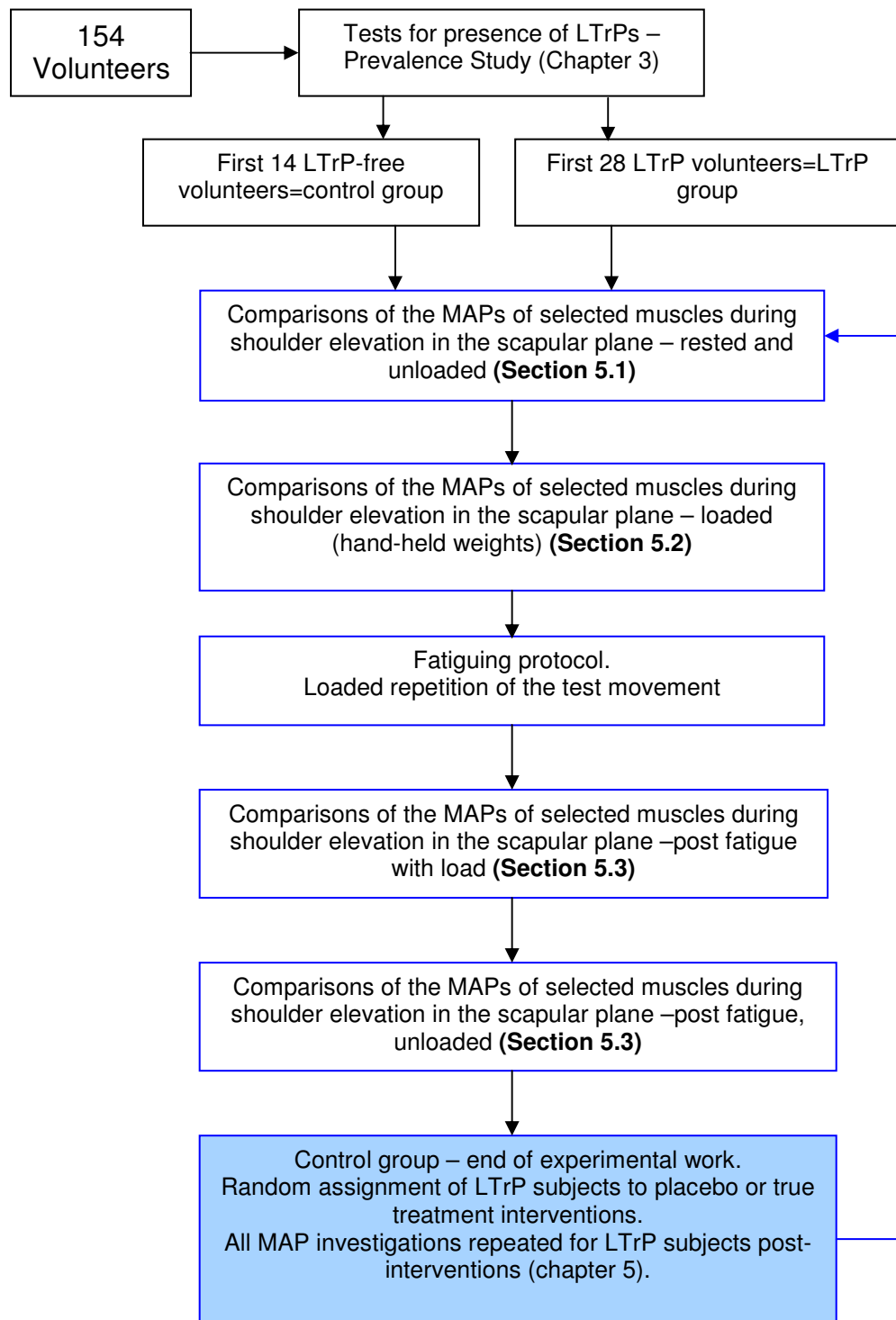


Figure 5.1.1: Outline of the experimental sequence. The contents of Chapter 5 begin from the treatment interventions (coloured blue).

5.1 The Effects of Removing LTrPs from the Scapular Rotator Muscles on MAPs during Scapular Plane Elevation

5.1.1 Introduction

Though the presence of pain has been shown to be capable of altering patterns of neuromuscular activation, there is also some evidence that non-painful stimuli can have similar effects (Sterling, Jull & Wright 2001) for example where the quadriceps muscles can be inhibited secondary to non-painful knee joint effusion (Shakespeare et al. 1985; Stokes & Young 1984). Latent TrPs are pain-free at rest or during movement, but do elicit pain when compressed (Simons, D., Travell & Simons 1999), presumably due to the presence of increased interstitial concentrations of nociceptor sensitising substances resulting from the presence of LTrPs. Greater concentrations of such substances at the sites of ATrPs are thought to explain the different pain responses in these two forms of trigger point (Shah, J. P. et al. 2005). In Chapter four it was established that LTrPs in the scapular rotator muscles are associated with altered MAPs during scapular plane elevation of the arm under unloaded, loaded and fatigued conditions, when compared with healthy subjects (control group). It is possible that such altered MAPs could lead to scapular dyskinesis (Kibler, W. B. & McMullen 2003) and thereby predispose to rotator cuff problems or overuse (Weldon & Richardson 2001) and/or subacromial impingement syndrome (Lewis, Wright & Green 2005; Michener, McClure & Karduna 2003). In contrast, identification of a mal-

adaptive MAP or associated scapular dyskinesis, prior to the development of a painful shoulder condition, could provide an opportunity to prevent progression to a painful injury and optimise movement performance of the upper extremity (Kibler, W. B. 1998b).

Because this chapter describes what happens to MAPs after the treatment of LTrPs, a very brief rationale for the chosen treatment is now provided – the reader is referred to Chapter two (page 66) and Appendix F (page 289) for further information on this topic. There are many TrP treatment modalities that work through a variety of mechanisms with varying degrees of efficacy, however, based upon the limited experimental data available, SDN (which requires the application of stretching after needle removal for best results (Edwards & Knowles 2003)) provided the most feasible approach due to its proven effectiveness (Cummings & White 2001; Sandberg et al. 2005), and provision of a relatively localised stimulus with little involvement of neighbouring tissues (Simons, D., Travell & Simons 1999). This is an important consideration given that any mechanical, thermal or chemical stimulus produces input to the CNS with the potential to influence motor output reflected in MAPs (Gandevia, McCloskey & Burke 1992). Furthermore, this technique requires no injectable and no need for medically qualified personnel. Section 5.1 which follows, describes the work carried out to answer the first two questions presented in the introductory pages of this Chapter, namely:

1. Does removing LTrPs (absence of the clinical signs of LTrPs) from the scapular rotator muscles alter the activation patterns (MAPs) of these

muscles during the performance of a common movement (scapular plane elevation of the arm)?

2. What affect (if any), does removing LTrPs have on the activation patterns of muscles placed more distally in the kinetic chain of the upper extremity?

5.1.2 Methods

5.1.2.1 Subjects and procedures

As stated in the chapter overview, the experiments described below were carried out on the same 42 subjects described in Chapter four (14 control and 28 LTrP subjects). However, this time the LTrP subjects were randomly assigned to receive either treatment (N=14) or sham treatment (N=14) followed by determination of MAPs during scapular plane elevation under the same conditions as described elsewhere (unloaded, loaded, post-fatigue). Table 5.1.1 presents the relevant characteristics of each group.

Table 5.1.1: Characteristics 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

5.1.2 Treatment group protocol

After completing the sEMG protocols described in Chapter 4, the LTrP treatment subjects then received superficial dry needling (SDN) using 30mm long, 0.30mm gauge acupuncture needles (Hwato Ultraclean, Acuneeds, Camberwell, Victoria, Australia) followed by passive muscle stretch to remove LTrPs (a validated LTrP treatment: (Chen, J. T. et al. 2001; Cummings & White 2001; Lewit & Simons 1984; Sandberg et al. 2005)). Surface EMG electrodes were left in situ following the completion of the previous protocol (fatigue affects) wherever this didn't interfere with the respective interventions. However on the rare occasions where removal was necessary, electrode positions were marked to allow new electrodes to be placed as near as possible to the original position.

The treatment protocol is described in detail as follows.

1. With the subject lying prone, Superficial dry needling, which consisted of cleaning the skin with an antiseptic wipe (70% alcohol, 30% water), then

inserting the acupuncture needle to a depth of 5-10mm at LTrP sites. With the subject lying prone, SDN was performed on any LTrPs in the lower trapezius (LT), middle trapezius (MT), rhomboids (Rh), levator scapulae (LS) and upper trapezius (UT), first on the right, then the left side of the body.

2. With the subject lying supine, SDN (as described above) was performed on LTrPs located in the pectoralis minor (PM), serratus anterior (SA) and UT (if the LTrPs was in the more anterior fibers of the muscle).
3. Following SDN, PIR stretching was performed for the PM and SA (usually with the subject supine), the LT, MT and Rh (usually with the subject in a side-lying position) and the LT and UT (usually with the subject in a seated position), as recommended by Lewit (personal communication, 2000)
4. After the completion of the SDN and PIR, subjects stood and the following active movements were performed gently, to full range three times each: neck rotations, shoulder shrugs, arm circles (forwards and back, with the elbows flexed) and horizontal flexion and extension to encourage the treated muscles to move through a full range of motion as suggested by Simons and colleagues (1999).
5. The subjects were then re-examined for LTrPs in all the scapular rotator muscles, the infraspinatus and the middle deltoid. Where the clinical signs of LTrPs were still present, or the PPT as measured with an algometer was still less than that expected for normal muscle tissue (see Table 3.1, page 125), LTrPs were re-treated (two of 14 subjects) until all muscles of interest were considered to be LTrP-free. Because PPTs increased despite extra LTrP treatment (two subjects), such additional treatment was

not expected to have adversely affected them or their subsequent performances in the tests.

All treated subjects then repeated the scapular plane elevation protocols (unloaded, loaded and post-fatigue) that had been undertaken earlier and described in Chapter four.

5.1.3 Sham treatment group protocol

After completing the sEMG investigations described in Chapter 4 (scapular plane elevation unloaded, loaded and following fatigue) the 'LTrP placebo' subjects received a sham ultrasound treatment which had no effect on their LTrPs. This was confirmed by the presence of the clinical signs of LTrPs and PPTs indicative of abnormal muscle tissue (Table 3.1, page 125) following placebo "treatment". An ultrasound machine commonly used to treat muscles was plugged into a fake power outlet on the wall and a false power switch turned on. The front of the ultrasound machine was turned away from the subject to conceal the fact that there was no indication of activity. Subjects were told 'pulsed' ultrasound was being used and were allowed to believe that the aim of the experiment was to investigate two different treatment interventions (SDN with PIR and pulsed ultrasound). Because of the aim of sham treatment, very light pressure was applied with a very slow stroking motion of the ultrasound transducer across the skin surface. The sham ultrasound protocol is described in detail as follows.

1. Ultrasound conducting gel was applied to all identified LTrPs and the ultrasound head was applied to the area over the LTrP for a period of three minutes. The PM and SA were 'treated' supine, then the subjects moved to a prone position and the UT, LS, MT, Rh and LT were 'treated'. The only functioning part of the ultrasound machine was a bell that rang to indicate that the three minutes of treatment had concluded. The sham nature of the treatment was revealed to subjects at the end of the experiment (that is, after the various scapular plane elevation protocols) and all indicated that they had believed that the treatment was real.
2. At the conclusion of the sham ultrasound treatment, the clinical signs indicating the presence of LTrPs remained in all subjects and there was no significant difference between the PPTs of these subjects pre or post sham ultrasound treatment, indicating that LTrPs remained.

5.1.4 Surface EMG protocols

After treatment, 'actual' or 'sham', all LTrP subjects repeated the sEMG protocols (unloaded, loaded, unloaded-post fatigue, loaded post fatigue) as described in detail in Chapter 4 (page 144) and depicted in Figure 5.1.1 (page 208). For each subject, the "treatments" (actual or sham) took approximately 20 minutes and the "post treatment" sEMG recordings of scapular plane elevation under the various conditions, occurred approximately 30 minutes after the pre-treatment sEMG evaluation finished during which time, all subjects were ostensibly resting.

5.1.5 Statistical analysis

Paired t-tests were employed to compare the mean times of muscle activation of the LTrPs subjects pre and post “treatment” while an independent t-test was used to compare the MAPs of treated LTrP subjects (LTrPs removed) with the control group. Again, F statistics were used to compare the variability of activation times for each muscle between the LTrP subjects in their various states and the control group as appropriate. The significance level was set at $p < 0.05$ for all comparisons.

5.1.3 Results

5.1.3.1 *LTrP subjects pre and post treatment (within group comparisons): unloaded motion*

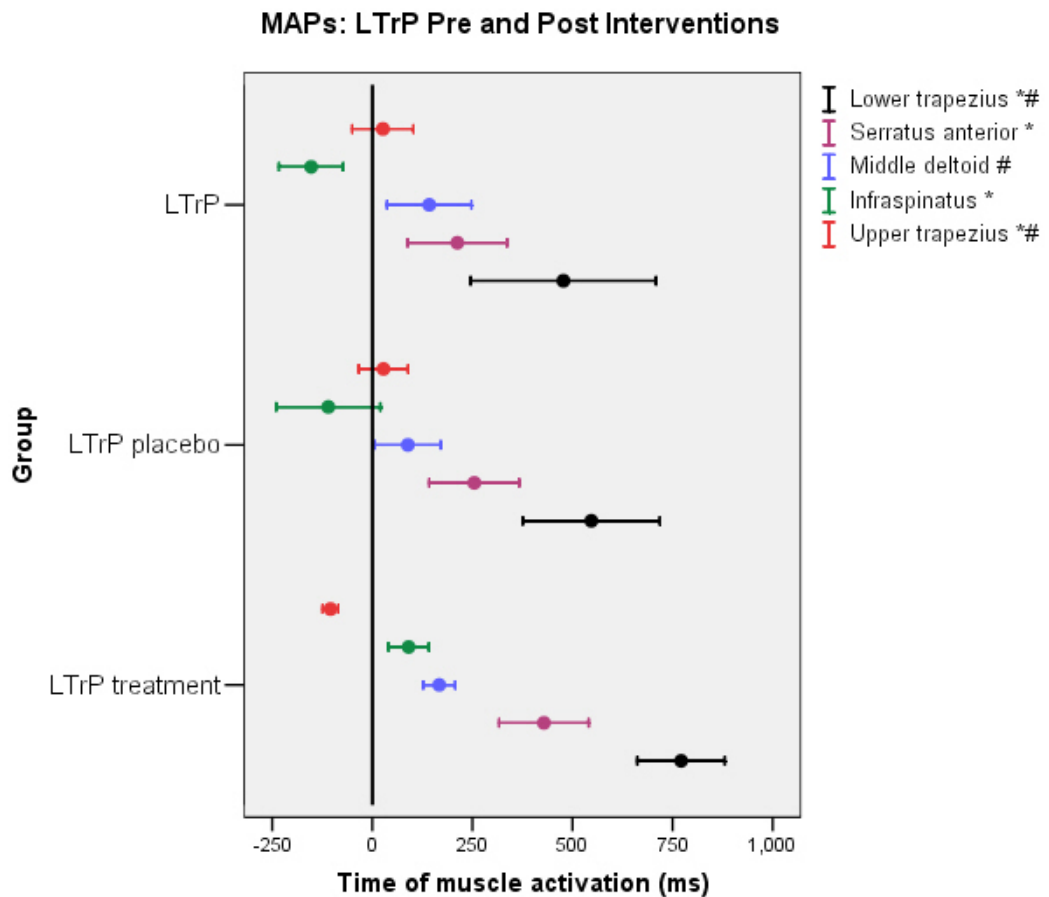
Group data are depicted in Table 5.1.2 and Figure 5.1.2 below and show the mean activation times (solid circles) and SD of activation times (bars) for each muscle for unloaded scapular plane elevation. The LTrP group receiving sham treatment, had the same order of muscle activation before and after sham treatment with no significant differences occurring for the timing of any muscle as follows: Inf activated prior to movement start, the UT immediately as the arm moves, then the MD, SA and LT after movement start respectively. In contrast, the LTrP group who received SDN and PIR stretching to remove LTrPs, displayed a ‘normalised’ order of muscle

activation, that is, UT prior to movement start, followed by Inf, MD, SA and LT activated sequentially after movement start. In these subjects, the UT was activated significantly earlier and the Inf, SA and LT were activated significantly later. Finally, the variability in activation times for the group receiving actual treatment decreased significantly post treatment for all muscles except the Inf and SA.

Table 5.1.2: Mean muscle activation times for the LTrP subjects prior to and after placebo and treatment interventions in the rested state.

	UT	Inf	MD	SA	LT
LTrP present	27	-153	142	212	477
LTrP placebo	28	-110	89	254	547
LTrP treatment	-105*#	91*	167#	429*	771*#

* significant difference in activation time between LTrPs present compared with LTrPs absent. # significant difference in the variability of activation times between LTrPs present compared with LTrPs absent (p<0.05). UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius.



*significant difference in activation times. # significant difference in variability of activation times ($p < 0.05$).

Figure 5.1.2 Mean muscle activation times for the LTrP subjects prior to and after placebo and treatment interventions in the rested state (mean and SD displayed).

5.1.3.2 Control Vs LTrPs removed (between group comparisons): unloaded motion

Table 5.1.3 and Figure 5.1.3 display the mean activation times for the unloaded condition for the control group and the post-treatment LTrP subjects (LTrPs removed). As can be seen, there were no significant

differences between groups for activation times for any muscle and only the Inf was more variable in the post-treatment group.

Table 5.1.3: Mean muscle activation times for the control group compared with the LTrP subjects who had their LTrPs removed in the unloaded state.

	UT	Inf	MD	SA	LT
Control	-115	75	201	434	776
LTrPs absent	-105	71#	167	429	771

* significant difference in activation times. # significant difference in the variability of activation times ($p < 0.05$). UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius.

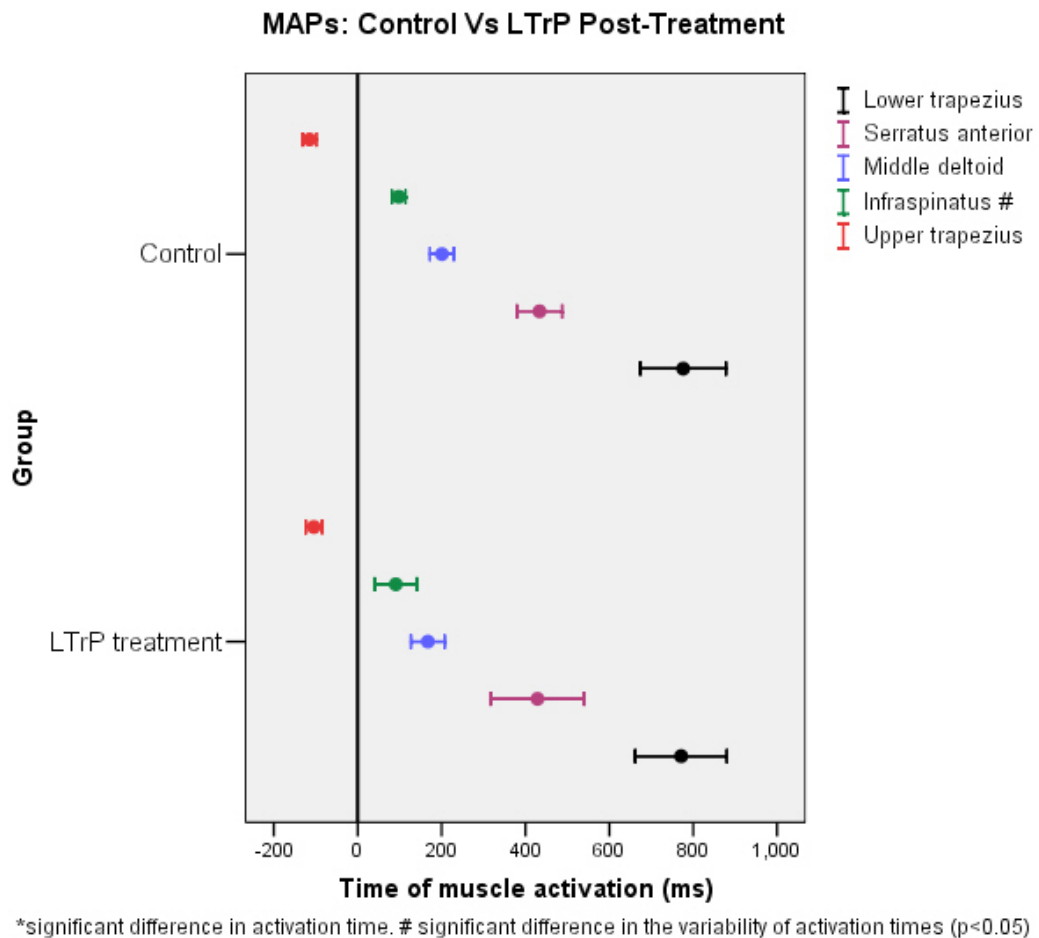


Figure 5.1.3: Mean muscle activation times for the control group compared with the LTrP subjects that had their LTrPs removed in the unloaded state (mean and SD displayed).

5.1.4 Discussion of Results

The results confirm that LTrPs in the scapular rotator muscles alter their activation patterns and also those of functionally related muscles, (infraspinatus, middle deltoid), by altering mean activation times and increasing their variability. This finding supports the proposal of Sterling and

colleagues (2001) who hypothesised that reflex inhibition of motoneurons secondary to some kind of afferent input not perceived as painful, could cause changes in neuromuscular control in humans (Shakespeare et al. 1985; Sterling, Jull & Wright 2001; Stokes & Young 1984). In addition, these findings support Kibler's hypothesis that dysfunction in a proximal segment of a kinetic chain (LTrPs in the scapular muscles in this instance), can affect the function of related segments (infraspinatus of the rotator cuff, middle deltoid as an abductor of the arm) in order to move the most distal segment, in this case, the hand (Kibler, W. B. 1998b) .

With regard to motor control, Neilson (1993) suggested that the CNS contains circuitry able to judge and adapt the accuracy of motor commands by comparison between the command signals (internal movement model) despatched and their consequences (through sensory feedback). In this model, if the inputs show good correlation with the internal reference, the established motor program can 'play out' without alteration by the CNS (Neilson 1993). For the control group in the present study, though timing varied slightly, the same order of muscle activation occurred in all subjects, that is, UT immediately prior to movement start, Inf and MD immediately after movement start, then SA and LT respectively, suggesting that sensory inputs in control subjects were well correlated with the internal model for elevation of the arm in the scapular plane with a consistent MAP resulting. Later, Neilson (2005) also hypothesised that the adaptive CNS circuitry was able to independently extract varying sensory and motor signals and continually alter motor commands in a feedforward process designed to replicate the pre-

planned movement as closely as possible (Neilson & Neilson 2005). In a more recent review, van Vliet and Heneghan (2006) stated that feedforward control incorporates inputs occurring immediately prior to the beginning of a movement and is an ongoing process during the movement (van Vliet & Heneghan 2006). For the LTrP group, the UT was the muscle most likely to contain a LTrP (93% of 28 subjects) and in contrast to the control group where it was always activated first, the UT was never activated first in the LTrP group. Many authors have reported on the specific purpose of the UT as an initiator of elevation of the acromion early in scapular plane elevation of the arm (Bagg & Forrest 1986, 1988; Ludewig & Cook 2000; Ludewig, Cook & Nawoczenski 1996; Matias & Pascoal 2006; Wadsworth & Bullock-Saxton 1997). Accepting that UT plays a specific role and is therefore activated early in scapular plane elevation as part of an optimal activation pattern, where some factor (LTrPs) interferes with this timing, feedforward processing may have altered the rest of the MAP in an attempt to sustain the overall movement. Support for what might be referred to as a “compromised movement pattern” generated through feedforward processing in the face of pain has previously been demonstrated in the “sub-optimal” MAPs recorded for low back pain patients (Hodges, P. W. 2001; Hodges, P. W. et al. 2003), chronic neck pain sufferers (Falla, D., Bilenkij & Jull 2004), patients with long standing groin pain (Cowan et al. 2004) and those with patellofemoral pain (Cowan et al. 2003), but to the author’s knowledge, has not yet been demonstrated in a LTrP population, who feel pain when their LTrPs are directly compressed but are pain-free during movement. Given the possibility that an optimal MAP for scapular plane elevation depends upon early

activation of UT, understanding how the presence of a LTrP can delay its activation seems crucial to restoring optimal kinematics.

With regard to comprehending how LTrPs may affect muscle activation, Shah and colleagues (2005) found increased interstitial concentrations of nociceptor activating substances, specifically substance P (SP) and calcitonin gene-related peptide (CGRP) in UT muscles containing both active and latent TrPs compared to TrP-free muscles (active>latent>normals; N=3 in each group; $p<0.02$). Given that ATrPs are spontaneously painful, the presence of pain-associated neuropeptides might be expected. However, the presence of these endogenous substances, usually produced in response to fatigue, pain or inflammation, at LTrP sites in greater concentrations than in normal tissue, implies that group III and IV afferent input could occur at rest and stimulate neural pathways similar to those expected where muscle is fatigued, painful or inflamed. These pathways were described in Chapter 4 (page 193) as neural mechanisms that underpin fatigue in healthy subjects but are presented again below in point form for ease of reading (for underpinning references, see Chapter 4, page 193).

5.1.4.1 *Facilitation of α -motoneurons secondary to group III and IV stimulation of fusimotor drive*

- Nociceptors are exposed to activating algescic substances and stimulate increased group III and IV afferent discharge. This leads to excitation of γ -motoneurons, especially static γ -motoneurons, which produces increased static (predominantly) and dynamic sensitivity to stretch. The increased spindle Ia and II input facilitates homonymous and heteronymous α and γ motoneurons and Ia inhibitory interneurons resulting in activation of agonist and synergist muscles and inhibition of antagonist muscle(s).

5.1.4.2 *Inhibition of α -motoneurons secondary to group III and IV afferent input*

- Where fusimotor drive secondary to group III and IV afferent discharge facilitates agonist and synergist muscles and inhibits antagonists via the pathway outlined immediately above, the addition of pre-synaptic inhibition of the Ia fiber terminal results in a reduced excitatory input to the α and γ motoneurons and resultant relative depression of muscles usually activated or inhibited via this pathway.
- Secondly, group III and IV afferent input may act at supraspinal sites, resulting in sub-optimal descending excitation of α -motoneurons or

may cause recurrent inhibition of multiple motoneurons by facilitating the inhibitory affects of Renshaw cells.

A neural process not explored in Chapter four involves the potential effects of increased tension of the muscle fibers forming a LTrP taut band on the fusimotor system. Underpinning evidence comes from an earlier study performed by Macefield and colleagues (1991). In this study, subjects performed a low intensity (30% of MVC) isometric contraction for one minute while muscle spindle afferent discharge was measured. Interestingly, the authors found a significant decrease in spindle output that occurred approximately 10 seconds into the sustained contraction which further decreased to half the initial discharge rate by one minute (Macefield et al. 1991). Although the current study used isotonic muscle contractions when measuring MAPs during the test movement, the nature of a LTrP taut band may somewhat replicate a low intensity isometric contraction of the involved fibers when the muscle is at rest. Thus, muscle spindles situated in parallel or in close association with a LTrP taut band while the muscle is at rest may produce a decreased afferent discharge into the CNS “disfacilitating motoneurons” as in Macefield and colleagues (1991) study. Likewise, the presence of LTrPs, with their associated nociceptor activating substances resulting in group III and IV afferent firing and taut band with contracted sarcomeres secondary to increased ACh acting on the exposed muscle cell membrane (see Chapter 2, pages 23-28; 50), potentially replicates a low intensity sustained isometric contraction of the muscle fibers forming the taut band at rest. If this concept is accurate, muscle fibers forming the LTrP taut

band might be associated with decreased spindle afferent discharge with the muscle at rest, resulting in disfacilitation of motoneurons and a decreased ability to activate agonist and synergistic muscles or inhibit antagonists. Speculating further, this may be one mechanism that potentially results in altered or variable MAPs when performing isotonic contractions in association with a low intensity isometric contraction of part of the muscle (taut band) and this potential disfacilitation of motoneurons might explain the variable timing of muscle activation in the current study.

In addition to the effects of the tension of the taut band, Rothwell (1994) reported that muscle spindles are approximately 10mm in length, indicating that one spindle receptor would span approximately 3,700 sarcomeres, where sarcomeres were at optimal length (2.69 μ m according to (Zuurbier et al. 1995)). Rothwell (1994) also reported that spindle function was affected by their position within the muscle. Given they are preferentially sensitive to small increases in stretch (Matthews & Stein 1969; Poppele & Bowman 1970) (for example, a 50 μ m stretch in a decerebrate cat soleus muscle significantly increased spindle afferent discharge (Rothwell 1994)), it follows that they may be activated by the lengthening of sarcomeres that occurs in parts of muscle fibers adjacent to LTrP contraction knots. Conversely, spindles in the endplate zone that may be situated over sections of a muscle fiber that remain shortened in the presence of a LTrP contraction knot, could be “unloaded” and decrease their afferent output. If so, decreased spindle afferent output from these muscle spindles, with associated disfacilitation of gamma and synergistic α -motoneurons, may decrease α -motoneuron drive

to homonymous and heteronymous muscles, ultimately reducing the excitatory drive for activation of these muscles. Interestingly, according to these potential responses, it might be expected that where LTrPs exist, muscle spindle output would be decreased from spindles within the LTrP region (due to the decreased lengthening ability assumed in this part of the muscle fiber), with the opposite response away from the LTrP. Such “opposing” outputs have the potential to “compromise” the proprioceptive feedback provided from multiple spindles in LTrP-affected muscles with affects on the timing of that muscle’s activation. The possibility that spindles can be adapted to muscle fiber length by the fusimotor system (or by intrafusal creep of the dynamic bag sensory region) might not mean that any proprioceptive disturbance would be transient since even if the affected intrafusal fibres are adapted to the length change of their muscle fibers, their output would not be consistent with the length of the unaffected fibres. In addition since the muscle fibers from different motor units mix within any volume of muscle, it is possible that any influence these “dysfunctional” spindles have, could play upon motoneurons controlling different motor units and therefore affect a broader cross section of the motoneuron pool. Though these notions are nothing more than conjecture at this stage, they might provide a basis for further investigation into the effects of muscle spindle input from LTrP affected muscles and ultimately, motor control and movement performance. In addition to neural mechanisms that may alter MAPs, intrinsic properties of LTrP-affected muscle fibers may also contribute to the efficacy of muscle activation. An attempt to explain these potential interactions forms the next section of this discussion.

5.1.4.3 *The intrinsic properties of LTrP-affected muscles*

As suggested above, in addition to neural mechanisms, the intrinsic properties (length/tension relationship of individual muscle fibers and their sarcomeres, contractile efficiency etc) of a specific muscle may contribute to its ability to contract (Gandevia, 2001). A LTrP contraction knot consists of multiple shortened sarcomeres in closely related muscle fibers, which force adjacent sarcomeres within the LTrP-involved muscle fibers to increase in length in an attempt to preserve the normal length of the entire fibre (Simons, D., Travell & Simons 1999). Muscle fiber configuration of this type could result in mechanical failure or disruption of normal contraction of the contractile elements due to loss of overlap of actin and myosin in the lengthened sarcomeres and active insufficiency of the shortened sarcomeres, which when occurring in multiple muscle fibers simultaneously, may reduce the force generating capacity of the entire muscle (Denoth et al. 2002). It has been known for some time that sarcomere lengths within normal muscle fibres are not homogeneous, leading to differing length/tension conditions throughout a fibre (Telley, Denoth & Ranatunga 2003). However, the effects of LTrPs just described could be expected to increase this heterogeneity with unknown effects on force production and perhaps on recruitment. As an additional consideration, sarcomeres that remain shortened would be expected to have altered cross bridge attachments/detachments mechanics, which may affect the relaxation time of the contractile elements of affected muscle fibers. There are also increase in the static tension (as evidenced by the taut band) of the muscle fiber and this

possibly alters the stiffness of affected fibers. A decreased relaxation rate (increased time taken for cross bridge detachment) is thought to increase muscle twitch duration and ultimately decrease the contractile speed in sustained isometric contractions (Bigland-Ritchie, B. et al. 1983; Bigland-Ritchie, B. & Woods 1984). However, no information could be found regarding the specific effects of decreased contractile speed during contractions of changing length. Reduced contractile capacity via either mechanism (loss of contractile efficiency due to severely shortened and consequently lengthened sarcomeres or reduced contractile speed secondary to altered cross bridge mechanics) could result in an initially reduced sEMG amplitude until additional motor units are activated, possibly delaying the time identified as the onset of muscle activation (defined as one SD above the mean baseline amplitude in the current study). Though these thoughts are speculative and further investigation is necessary, this possible mechanism (if it exists) could have contributed to the later activation of the UT in LTrP subjects demonstrated in the present study.

Importantly, Mense (1997) pointed out that the goal of nociceptor activation was to provide timely feedback to the CNS regarding the structural or functional limits of tissues (Mense 1997). As described above, nociceptive input from muscles affects muscle spindles (Knutson 2000) and probably descending central commands (Taylor, Todd & Gandevia 2006), providing multiple neural processes that could result in either excitation or inhibition of muscle activation and alteration of the associated MAP (Bigland-Ritchie, B. R. et al. 1986; Garland 1991; Woods, Furbush & Bigland-Ritchie 1987).

Though nociceptive activating substances at greater concentrations than found in normal tissue have been found at LTrPs (SP and CGRP in a sample of three) and LTrP-affected muscle tissue has a pressure-pain threshold lower than would be expected in normal muscle (see Chapter 3, page ? and (Hong, C. Z. et al. 1997)), it is difficult to determine whether interstitial concentrations of nociceptor activating substances associated with low levels of fatigue (see discussion Section 4.3, page 190) or LTrPs, can also invoke reflex inhibition of motoneurons. To provide insight into the effects of the concentration of nociceptor activating substances a study performed by Farina and colleagues (2004) may be helpful. These authors stimulated nociceptive afferents by injection of hypertonic saline into the muscles of humans subjects, thus experimentally activating group III and IV afferent fibers, and found decreased motoneuron firing rates when subjects produced a contraction that was 10% of MVC (low intensity sustained isometric contraction). Furthermore, the decrease was inversely correlated with both the amount of nociceptive input (as judged by the concentration of hypertonic saline injected) and the subjectively scored pain intensity (Farina, D. et al. 2004). Combining the results of Shah's group (2005) with the findings of Farina's (2004) suggests that with low intensity sustained isometric contraction, (perhaps similar to the situation created by a LTrP taut band), lower concentrations of nociceptor activating substances (LTrPs) produce less decrease in motoneuron discharge while greater concentrations (ATrPs) are associated with greater disfacilitation of motoneurons. Therefore, it is reasonable to posit that the interstitial concentrations of nociceptor activating chemicals affect the amount of motoneuron disfacilitation and that the

concentrations associated with LTrPs, can produce this phenomenon in the absence of spontaneous pain.

In summary, although no literature could be found specifically dealing with the motor effects of LTrPs, it has been a popular belief of clinicians that these neuromuscular lesions are indeed capable of motor effects (Hong, C. Z. 2004; Simons, D. G. 2005). The 'normalisation' of the MAP once LTrPs had been removed (clinical signs absent) greatly strengthens the conclusions stated in Chapter 4 (pages 160;174; 204), that LTrPs in the scapular rotator muscles affect the timing of muscle activation in this group and though this study was not intended to investigate the processes that may have produced these results, the presentation of ideas in the previous sections is meant to promote further discussion regarding the motor effects of LTrP-containing muscles and act as a spur for further debate and investigation.

5.1.4.4 Adaptive role of infraspinatus

Where LTrPs existed in the scapular rotator muscles, the only consistency in the MAP was that the Inf was activated first (occurred in 92% of trials). Infraspinatus plays an integral role in the early phase of elevation of the arm in the scapular plane by pulling on the greater tubercle of the humerus to cause slight external rotation, depression and posterior translation of the

humeral head in the glenoid fossa as the humerus begins to abduct, which along with the remaining rotator cuff muscles results in a compressive force to promote the dynamic stability of the glenohumeral joint (Neumann 2002; Payne et al. 1997; Reddy et al. 2000; Sharkey & Marder 1995). According to Kibler (1998), the muscles that connect the scapula to the vertebrae and ribs (all parts of the trapezius and rhomboids, serratus anterior, levator scapular and pectoralis minor), act to optimally position the scapula during upper extremity motion to best accommodate the rotating humeral head, contributing to a stable and functional glenohumeral joint. He argued that where the glenoid was not optimally positioned during arm movements, the muscles that positioned the humeral head may be predisposed to altered function in order to preserve the movement (Kibler, W. B. 1998b). He further suggested that where adaptation of function was ineffective or insufficient, the tissues attempting to compensate for the proximal dysfunction may be subjected to overload and injury. In the current study, except for the significantly earlier activation of the Inf, the MAP of the LTrP group was inconsistent with many different sequences of muscle activation occurring within the group, which might suggest that the early activation of the Inf was an attempt to adapt to sub-optimal glenoid positioning created by an inefficient MAP of the upward scapular rotator muscles, potentially exposing the compensating muscle to increased loads and ultimately injury.

5.1.5 Conclusions

Based on the results of this study, it can be concluded that:

1. The presence of LTrPs in the scapular rotator muscles is associated with changes in MAPs in the absence of pain, manifested as altered activation times and increased variability of muscle activation patterns.
2. Non-painful afferent input can influence neuromuscular activation patterns in support of the proposal of Sterling and colleagues (2001).
3. A pain-free neuromuscular dysfunction (LTrPs) in a proximal segment of the upper extremity kinetic chain can affect the function (MAPs) of structures in related segments in support of Kibler's, theory (Kibler 1998)
4. Such changes may predispose individuals to increased risk of subacromial impingement, overuse of the infraspinatus due to earlier activation and decreased efficiency of movement with resultant earlier onset of fatigue during scapular plane elevation.

5.2 The Effects of Removing LTrPs on MAPs during Loaded Scapular Plane Elevation

5.2.1 Introduction

Latent TrPs located in the scapular rotator muscles were found to alter MAPs during elevation of the arm in the scapular plane while holding hand-weights selected to represent loads that may be lifted during commonly performed tasks (males 4kg, females, 1.3kg). These findings and the related methodologies were reported in Chapter 4, Section 4.2 (page 162) and were used for comparison with the MAPs recorded for the LTrP subjects after the LTrPs had been removed, in the current section.

5.2.1.1 Question addressed:

Does the removal of LTrPs from the scapular rotator muscles change the MAP of these muscles or functionally related shoulder girdle muscles employed to elevate a load commonly encountered in daily activities during scapular plane elevation?

5.2.2 Methods

5.2.2.1 Subjects and Procedures

After the LTrP treatment interventions had been completed and the first sEMG investigations (unloaded state, section 5.1) had concluded, subjects repeated the 'Loaded' trials with the same hand-weights used in previous loaded trials (Section 4.2). Once again each subject completed three loaded arm elevations as described previously (section 4.2) with four seconds rest between each trial to allow the sEMG to return to a baseline level.

5.2.2.2 Statistical analysis

Paired t-tests were employed to compare the mean times of muscle activation of the LTrPs subjects pre and post their respective interventions. An independent t-test was used to compare the LTrP subjects who had their LTrPs removed to the control group. Again, F statistics were used to compare the variability of activation times for each muscle between the LTrPs subjects in their various states and the control group as appropriate. All significance levels were set at $p < 0.05$.

5.2.3 Results

As there were no significant difference between the MAPs of the LTrP subjects who went on to have treatment compared with those who had sham treatment prior to any interventions, the mean MAPs displayed through this Chapter for LTrP subjects prior to treatment were pooled (N=28) and each sub-group (treatment and sham treatment) were compared to the pooled data.

5.2.3.1 *LTrP subjects pre and post treatment (within group comparisons): loaded motion*

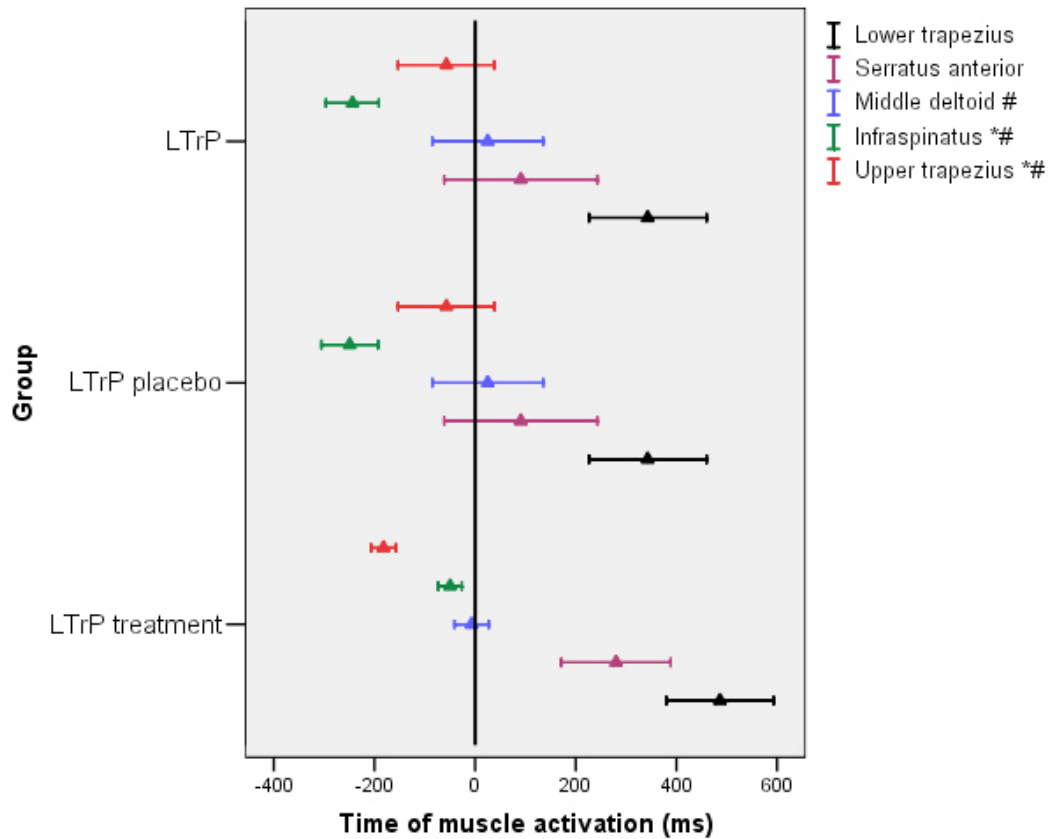
Table 5.2.1 and Figure 5.2.1 compare the mean activation times for the LTrP subjects prior to intervention with their post-intervention results. The LTrP group post-placebo showed no significant differences from the activation pattern prior to placebo intervention. In contrast, the paired t-test comparing the LTrP subjects prior to treatment with their “treated” LTrP group (LTrPs removed using SDN and PIR stretches), displayed a significant difference in mean activation times from the pre-intervention condition where the UT was activated significantly earlier and the Inf significantly later. In addition, the variability in activation times significantly decreased post treatment for the UT, Inf and MD.

Table 5.2.1: Mean muscle activation times for the LTrP subjects prior to and after placebo and treatment interventions in the loaded state.

	UT	Inf	MD	SA	LT
LTrP	-57	-244	25	91	343
LTrP placebo	-47	-264	19	86	371
LTrP treatment	-182*#	-50*#	-7#	280	486

significant difference in activation time between LTrPs present compared with LTrPs absent. # significant difference in the variability of activation times between LTrPs present compared with LTrPs absent ($p < 0.05$). UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius.

MAPs: LTrP Vs Pre and Post Interventions Loaded



*significant difference in activation times. # significant difference in the variability of activation times (p<0.05).

Figure 5.2.1: Mean muscle activation times for the LTrP subjects prior to and after placebo and treatment interventions in the loaded state (mean and SD displayed).

5.2.3.2 *Control Vs LTrPs removed (between group comparisons): loaded motion*

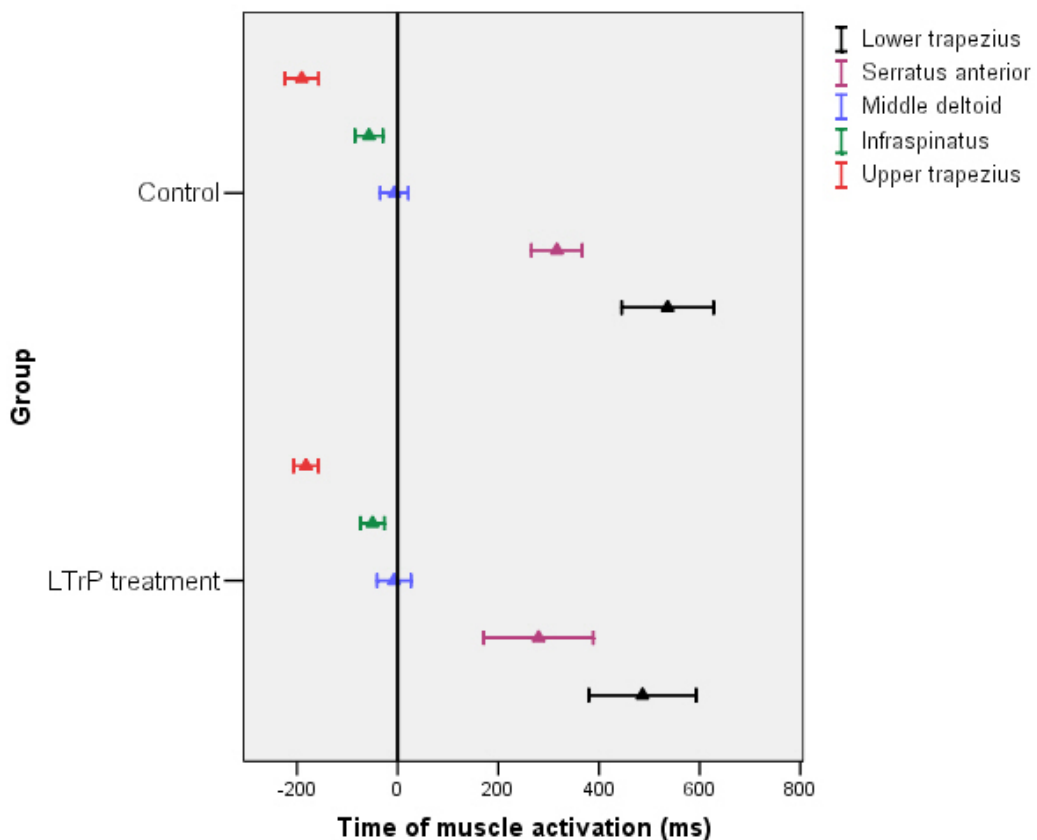
Table 5.2.2 and Figure 5.2.2 display the comparisons between the control group and the LTrP treatment group (LTrPs removed). There were no significant differences between groups (mean activation times, variability of activation times) for any muscle after LTrPs were treated.

Table 5.2.2: Mean muscle activation times for the Control group compared with the LTrP after treatment in the loaded state.

	UT	Inf	MD	SA	LT
Control	-191	-57	-6	316	536
LTrPs absent	-182	-50	-7	280	486

* significant difference in activation times. # significant difference in the variability of activation times ($p < 0.05$). UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius.

MAPs: Control Vs LTrP Post Treatment Loaded



*significant difference in activation times. # significant difference in the variability of activation times ($p < 0.05$).

Figure 5.2.2: Mean muscle activation times for the Control group compared with the LTrP after treatment in the loaded state (mean and SD displayed).

5.2.4 Discussion of Results

Chapter four reported on and discussed the results of an investigation into the effects of light loads on the MAPs of related shoulder girdle muscles during elevation in the scapular plane in healthy controls and subjects with LTrPs in the scapular rotator muscles. Under similar conditions (light loads) the protocols were repeated after LTrPs were removed from half the LTrP subjects thereby more accurately establishing the effects of LTrPs on MAPs under these conditions (loaded). The results virtually duplicated those found in the preceding Section (5.1, page 216), when scapular plane elevation was carried out without load in treated and untreated (sham) LTrP subjects. Hence it is logical to suggest that the same processes/mechanisms operated under each condition (unloaded and loaded) and are summarised as follows.

In the presence of LTrPs (pre-treatment condition), the only consistent aspect of the MAP was the early activation of the Inf. Placebo intervention, where LTrPs remained in the scapular rotator muscles, resulted in no change of the MAP from the pre-intervention state, but in contrast, where LTrPs were removed with treatment, the MAP 'normalised', showing no difference from that of the control group in the loaded state. The lack of change in MAPs after sham treatment in contrast to the "normalising" effect of the SDN treatment (same MAPs as controls under the same conditions), provides strong evidence that this treatment works and that LTrPs are indeed responsible for the altered MAPs in the scapular rotators and related muscles. In addition, the results add weight to the argument that LTrPs

should be treated because they do have what can be presumed as deleterious effects on MAPs in shoulder girdle muscles. It should be remembered that these data shed no light on the duration of the changes produced by treating LTrPs, however, while MAPs are “normal”, the risks of developing rotator cuff overuse or dysfunction, subacromial impingement or inefficient movement patterns is likely to be reduced. It is presumed that the same mechanisms underlying the way that LTrPs produce altered MAPs, continue to operate under load and may involve any of the combination of neural pathways (Type III and IV muscle afferents, fusimotor effects etc) discussed previously.

5.2.5 Conclusions

Based on the results of this study, it can be concluded that:

1. The addition of light external load does not reduce the capacity of the MAPs to normalise once LTrPs had been removed, implying that a positive outcome can be expected to hold for situations that require raising light weights overhead.
2. The findings of this study support both the views of Sterling and colleagues (2001) who suggested that non-painful afferent input could influence neuromuscular activation patterns and Kibler’s theory (1998), that dysfunction of a proximal kinetic chain segment affects the function of related segments.

3. Superficial dry needling followed by PIR stretching removes the clinical signs associated with the presence of LTrPs and the effects of the LTrPs themselves.

These outcomes may not hold in situations where repetitive arm elevations are required, an activity associated with the development or exacerbation of LTrPs (Simons, D., Travell & Simons 1999) and the subject of the following section of this chapter.

5.3 The Effects of Removing LTrPs on MAPs Post-fatiguing Movement during Scapular Plane Elevation

5.3.1 Introduction

In this final section of the experimental program, the effects of fatigue on the target muscles were examined following either SDN plus stretching or sham treatment of LTrPs. The protocols were the same as previously described: unloaded and loaded scapular plane elevation following fatigue induced by repetition of the same movement (Section 4.3, page 178) while holding hand-weights. Fatiguing activity was specifically “targeted” because of previous suggestions that fatigue is associated with the development or exacerbation of LTrPs (Simons, D., Travell & Simons 1999).

5.3.1.1 Question addressed:

1. Does the removal of LTrPs from the scapular rotator muscles change the MAP of this group or functionally related shoulder girdle muscles after fatiguing repetitive arm elevations during both unloaded and loaded scapular plane elevation?
2. Does fatigue alter the outcome of a LTrP treatment already shown to “normalize” MAPs in response to non-fatiguing exercise?

5.3.2 Methods

5.3.2.1 Subjects and Procedures

After the LTrP treatment interventions had been completed and the first two sEMG investigations (rested state, section 5.1 then loaded state, Section 5.2) had concluded, subjects were asked to again perform the fatiguing protocol as described in Chapter four (page 178), immediately followed by three trials of loaded scapular plane elevation, then three trials of the movement without load as outlined in Figure 5.3.1.

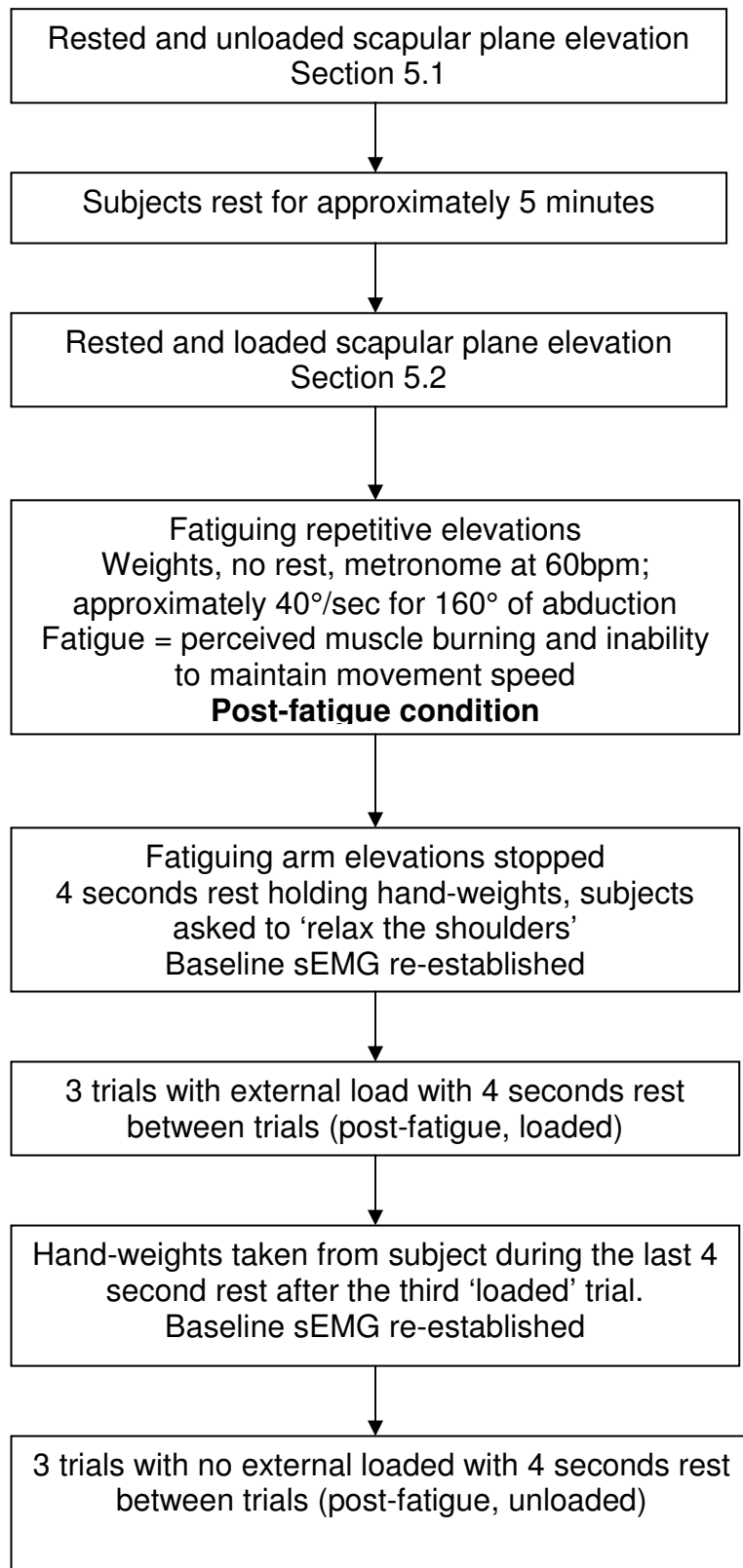


Figure 5.3.1: Experimental procedure to test the effects of fatigue on MAPs during scapular plane elevation

5.3.2.2 *Statistical analysis*

Paired t-tests were employed to compare the mean times of muscle activation of the LTrP subjects pre and post intervention (treatment or sham). An independent t-test was used to compare MAPs of the treated LTrP subjects with those of the control group under the same conditions. An additional independent t-test was employed to compare the mean activation times of the treated LTrP subjects post-fatigue with those of the control group in the rested state. Again, F statistics were used to compare the variability of activation times for each muscle between both LTrPs groups (treated and sham) and the control group as appropriate. Significance was again set at $p < 0.05$ for all tests.

5.3.3 **Results**

5.3.3.1 *LTrP subjects pre and post treatment post-fatigue (within group comparisons): unloaded motion*

Table 5.3.1 and Figure 5.3.2 compare the mean activation times for the LTrP subjects prior to intervention with their post-intervention post-fatigue and unloaded trials. The LTrP group post-placebo showed no significant differences from the activation pattern determined prior to sham intervention. In contrast, the LTrP group after LTrPs were removed using SDN and PIR stretching, displayed a significant difference ($p < 0.05$) in mean activation

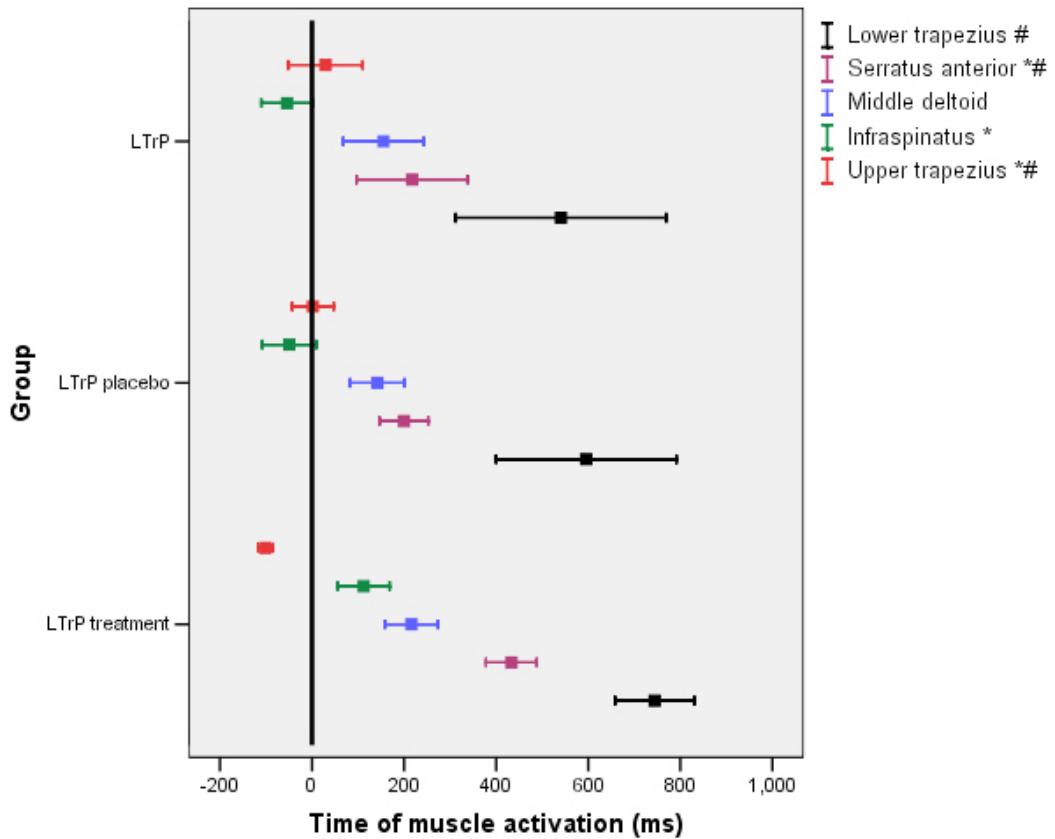
times from the pre-intervention condition for the UT (activated earlier) and Inf and SA (both activated later). The variability in activation times significantly ($p < 0.05$) decreased post treatment for the muscles that had formerly contained LTrPs (UT, SA and LT).

Table 5.3.1: Mean muscle activation times for the LTrP subjects prior to and after placebo and treatment interventions in the fatigued but unloaded state.

	UT	Inf	MD	SA	LT
LTrP	30	-54	155	218	541
LTrP placebo	2	-49	142	200	596
LTrP treatment	-101#	112*	216	433*#	745*#

* significant difference in activation time between LTrPs present compared with LTrPs absent. # significant difference in the variability of activation times between LTrPs present compared with LTrPs absent ($p < 0.05$). UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius.

MAPs: LTrP Pre Vs Post Interventions Fatigued



*significant difference in activation times. # significant difference in the variability of activation times (p<0.05).

Figure 5.3.2: MAPs for the LTrP subjects prior to and after placebo and treatment interventions post-fatigue and unloaded (mean and SD displayed).

5.3.3.2 Control Vs Treated LTrP group (between group comparisons): unloaded motion

Table 5.3.2 and Figure 5.3.3 display the comparisons between the control group and the LTrP treatment group (LTrPs removed) in the post-fatigue but unloaded condition. In treated subjects, all muscles except the UT, were

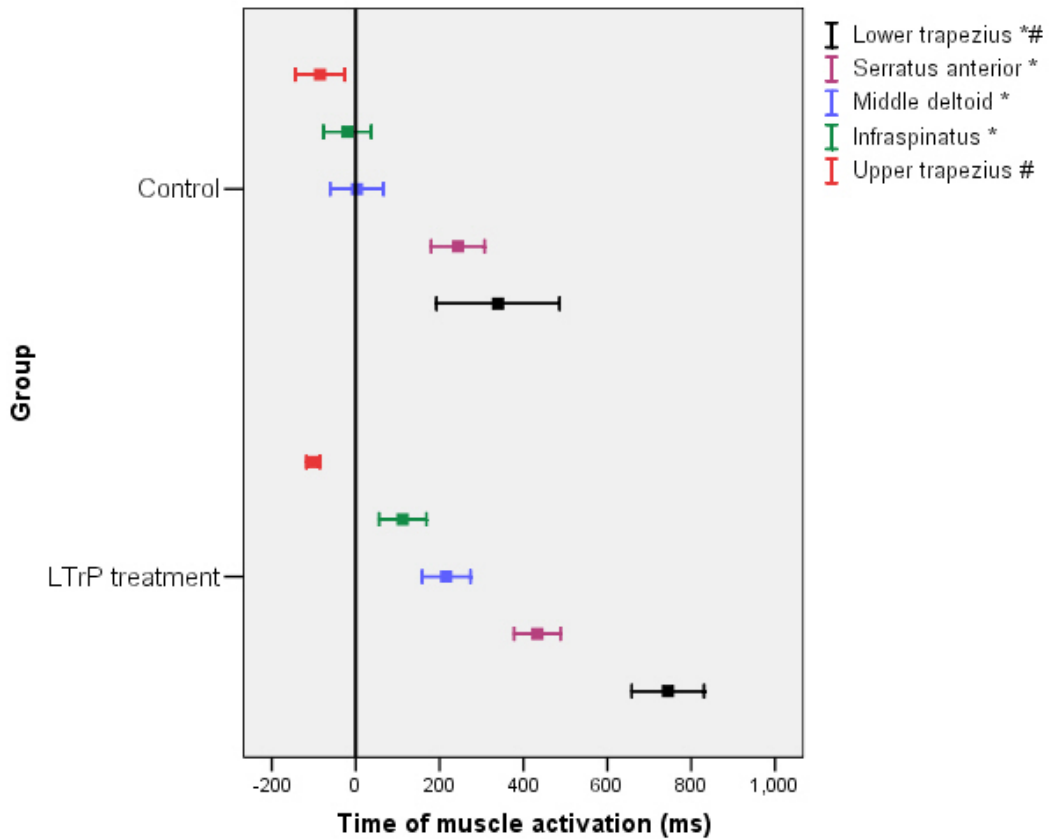
activated significantly later ($p < 0.05$) than in the controls. In addition, both the UT and LT muscles demonstrated significantly less variability of activation times versus controls after treatment.

Table 5.3.2: Mean muscle activation times for the Control group compared with the LTrP after treatment in the fatigued but unloaded state.

	UT	Inf	MD	SA	LT
Control	-85	-20	3	244	339
LTrPs absent	-112#	75*	202*	434*	776*#

* significant difference in activation times. # significant difference in the variability of activation times ($p < 0.05$). UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius.

MAPs: Control Vs LTrP Post Treatment Fatigued



*significant difference in activation times. # significant difference in the variability of activation times ($p < 0.05$).

Figure 5.3.3: MAPs comparing the control group to the LTrP subjects after the removal of LTrPs, post-fatigue and during unloaded scapular plane elevation (mean and SD displayed).

5.3.3.3 Treated LTrPs subjects post-fatigue Vs Control group rested (between group comparisons): unloaded motion

Table 5.3.3 and Figure 5.3.4 display the comparisons between the treated LTrP group (LTrPs removed) in the post-fatigue but unloaded condition and the control group in the rested and unloaded condition (i.e. controls before

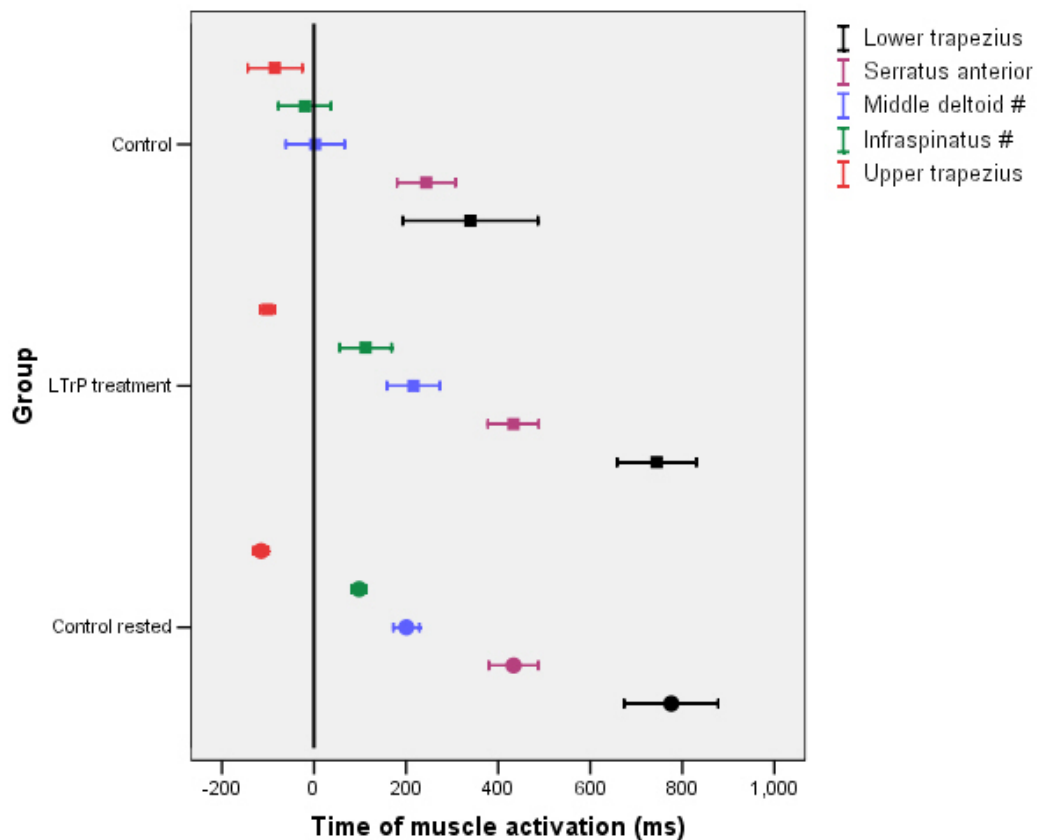
exposure to fatiguing exercise). No significant differences in mean activation times were found between the treated LTrP subjects in the fatigued and unloaded condition and the control group in the rested and unloaded condition. However, rested, unfatigued controls demonstrated significantly less variability in their activation times when performing scapular plane elevation without load (unloaded) and unfatigued.

Table 5.3.3: Mean muscle activation times comparing the control group (fatigued), the LTrP subjects once the LTrP had been removed (fatigued) and the control group (rested) during unloaded scapular plane elevation.

	UT	Inf	MD	SA	LT
Control fatigued	-85	-20	3	244	339
LTrPs absent	-110	71	196	428	765
Control rested	-115	75	201	434	776

* significant difference in activation time between control fatigued compared with LTrPs absent. # significant difference in the variability of activation times between control fatigued compared with LTrPs absent ($p < 0.05$). UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius.

MAPs: Control Fatigued Vs LTrP Post Treatment Fatigued Vs Control Rested



sig diff in variability of activation times control group rested Vs LTrP treated post-fatigued, all unloaded ($p < 0.05$).

Figure 5.3.4: MAPs comparing the control group (fatigued) the LTrP subjects after the removal of LTrPs (fatigued) and the control group (rested), during unloaded scapular plane elevation (mean and SD displayed). No significant differences in the timing of muscle activation exist between the LTrP subjects once the LTrP have been removed (fatigued) and the control group (rested), however the Inf and MD of the LTrP treatment group display greater variability in their activation times than does the control group in the rested state.

5.3.3.4 LTrP subjects (within group comparisons): loaded motion

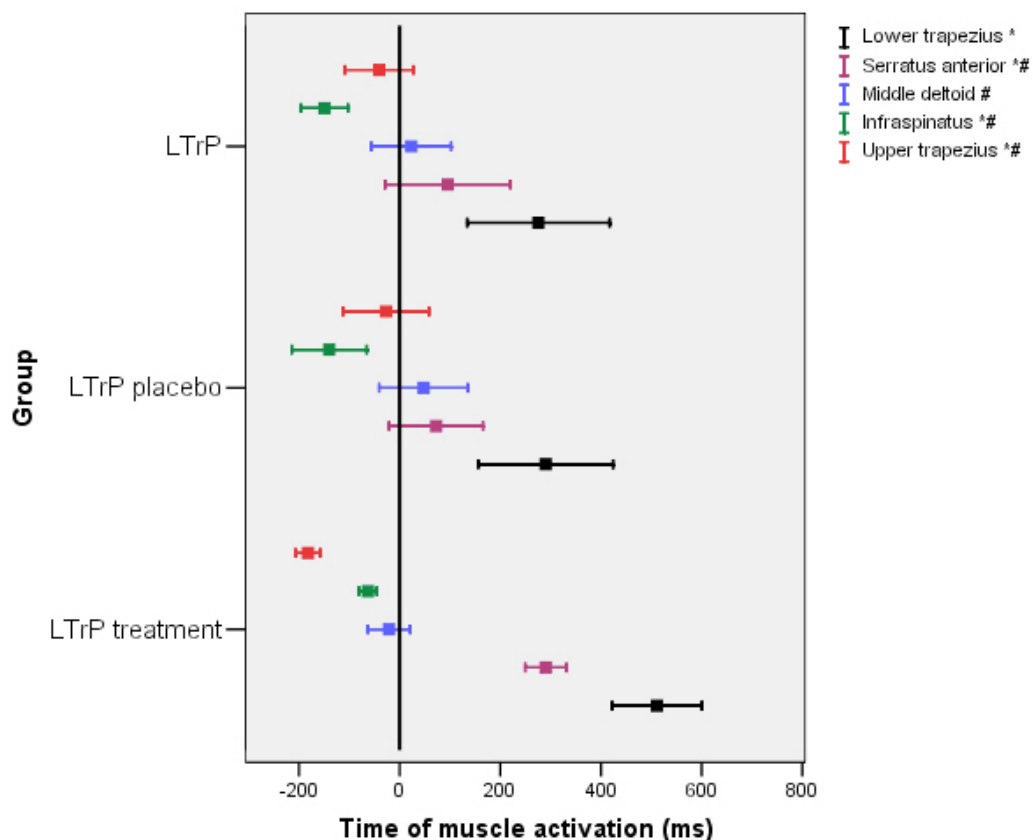
Table 5.3.4 and Figure 5.3.5 display the mean activation times for the LTrP subjects prior to intervention with their post-intervention states in the post-fatigue and loaded condition. The LTrP group post-placebo treatment, showed no significant differences from the activation pattern found prior to placebo intervention. In contrast, the treated LTrP group displayed significant differences in mean activation times after intervention: the UT was activated significantly earlier and the Inf, SA and LT all activated significantly later. Furthermore, their activation time variability significantly decreased post treatment in all muscles except the LT

Table 5.3.4: Mean muscle activation times for the LTrP subjects prior to and after placebo and treatment interventions in the fatigued and loaded state.

	UT	Inf	MD	SA	LT
LTrP	-41	-149	23	95	276
LTrP placebo	-27	-140	47	72	290
LTrP treatment	-182*#	-63*#	-21#	290*#	511*

* significant difference in activation time between LTrPs present compared with LTrPs absent. # significant difference in the variability of activation times between LTrPs present compared with LTrPs absent ($p < 0.05$). UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius.

MAPs: LTrP Pre Vs Post Interventions Fatigued and Loaded



*significant difference in activation times. # significant difference in the variability of activation times (p<0.05).

Figure 5.3.5: MAPs for the LTrP subjects prior to and after placebo and treatment interventions post-fatigue and loaded (mean and SD displayed).

5.3.3.5 Control Vs treated LTrPs (between group comparisons): loaded motion

Table 5.3.5 and Figure 5.3.6 display the comparisons between the control group and the treated LTrP subjects (LTrPs removed) in the fatigued and loaded condition. The UT was activated significantly earlier and the SA and

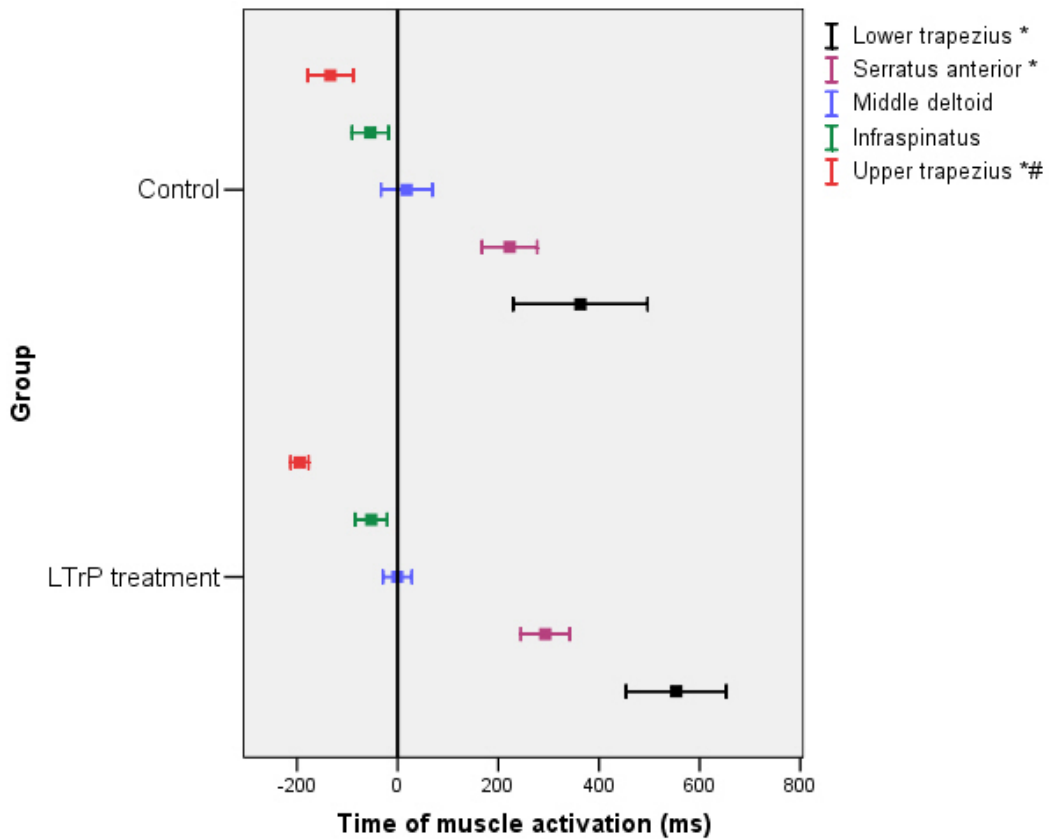
LT significantly later after LTrPs had been treated. In addition, the UT was significantly less variable in its activation time after LTrPs had been removed.

Table 5.3.5: Mean muscle activation times for the control group compared with the LTrP after treatment in the fatigued and loaded state.

	UT	Inf	MD	SA	LT
Control	-134	-54	-20	223	363
LTrPs absent	-195*#	-53	-1	294*	553*

* significant difference in activation times. # significant difference in the variability of activation times ($p < 0.05$). UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius.

MAPs: Control Vs LTrP Post Treatment Fatigued and Loaded



*significant difference in activation times. # significant difference in the variability of activation times ($p < 0.05$).

Figure 5.3.6: MAPs comparing the control group to the LTrP subjects after the removal of LTrPs, post-fatigue and during loaded scapular plane elevation (mean and SD displayed).

5.3.3.6 Treated LTrPs subjects post-fatigue Vs Control group rested (between group comparisons): loaded motion

Table 5.3.6 and Figure 5.3.7 display the comparisons between the LTrP treated group (LTrPs removed) in the post-fatigue and loaded condition and the control group in the rested and loaded condition (i.e. controls performing loaded scapular plane elevation prior to undergoing fatiguing exercise). No

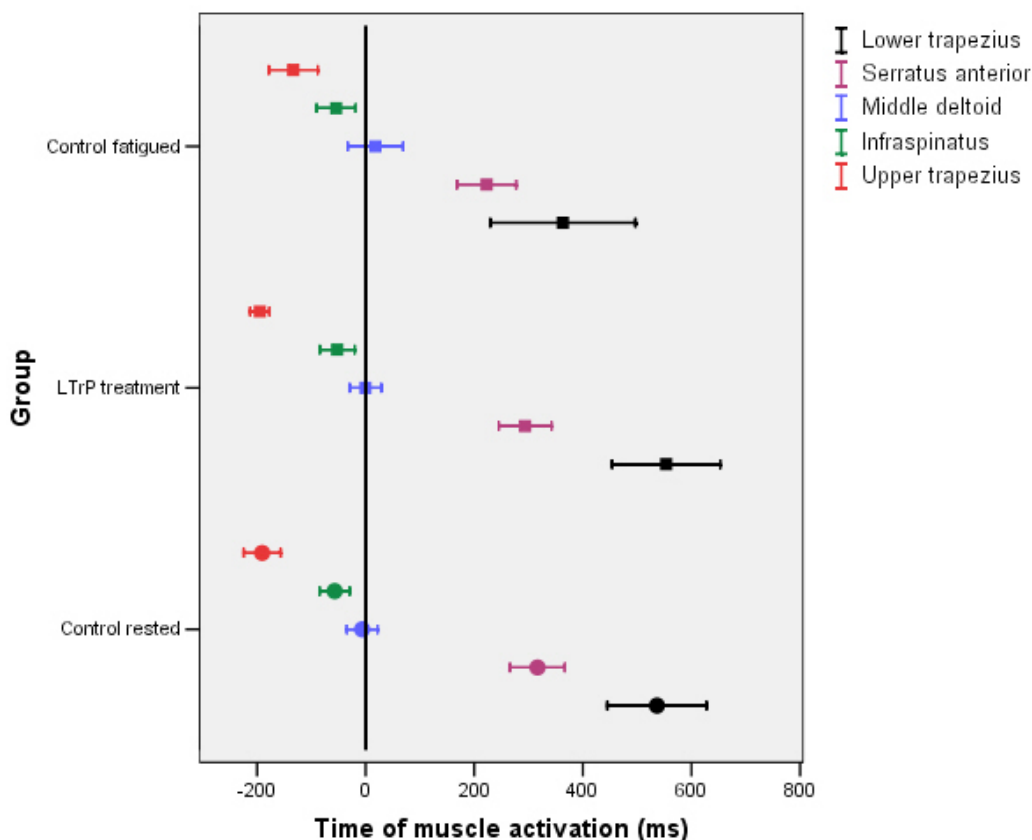
significant differences in mean activation times were found between the treated LTrP subjects (fatigued and loaded) compared with the control group performing loaded scapular plane elevation in the rested condition. However, controls (rested and loaded) had significantly less variability in activation times compared with the treated LTrPs subjects (fatigued and loaded condition).

Table 5.3.6: Mean muscle activation times comparing the control group (fatigued), the LTrP subjects once the LTrP had been removed (fatigued) and the control group (rested) during loaded scapular plane elevation.

	UT	Inf	MD	SA	LT
Control fatigued	-134	-54	-20	223	363
LTrPs absent	-195	-53	-1	294	553
Control rested	-191	-57	-6	316	536

* significant difference in activation time between control fatigued compared with LTrPs absent. # significant difference in the variability of activation times between control fatigued compared with LTrPs absent ($p < 0.05$). UT=upper trapezius, Inf=infraspinatus, MD=middle deltoid, SA=serratus anterior, LT=lower trapezius.

MAPs: Control Fatigued Vs LTrP Post Treatment Fatigued Vs Control Rested all Loaded



No significant differences found in the MAPs between the LTrP treated post-fatigue Vs Control rested ($p < 0.05$).

Figure 5.3.7: MAPs comparing the control group (fatigued) the LTrP subjects after the removal of LTrPs (fatigued) and the control group (rested), during loaded scapular plane elevation (mean and SD displayed). No significant differences in the timing of muscle activation exist between the LTrP subjects once the LTrP have been removed (fatigued) and the control group (rested).

5.3.4 Discussion of Results

Instead of discussing mean muscle activation times of specific muscles in various experimental states, this discussion will focus on the overall pattern that was exhibited in both the unloaded and loaded MAPs of subjects when LTrPs had been treated and removed. The fact that placebo treatment intervention, where LTrPs remained in the scapular rotator muscles, did not change the MAP of these subjects post-fatigue from the pre-intervention pattern, confirms the efficacy of the SDN plus stretching intervention by ruling out any placebo effect. The most significant result of this study was the finding that removing LTrPs from the scapular rotator muscles 'normalised' the MAP - they were most similar to those recorded from controls in the rested state (loaded and unloaded). Surprisingly, the treated LTrP results, post-fatigue were not the same as those produced by the controls under the same conditions (fatigued loaded and unloaded). It could be speculated that perhaps the treated LTrP subjects were not adequately fatigued, though they met the set criteria (inability to maintain the 400/sec cadence and 1600 range of shoulder abduction in both eccentric and concentric phases accompanied by "muscle burning" sensations). Assuming that they were on the basis of these criteria, an alternate explanation may be that they recovered from the fatiguing exercise more rapidly than did the control subjects, who were LTrP-free but had not received SDN or PIR stretching. In the current study, subjects had four seconds to relax their muscles after the fatiguing protocol which had the purpose of re-establishing a baseline sEMG signal before the test trials for loaded motion began. These three trials took eight seconds

each, with four seconds rest between trials, and were immediately followed by the three trials for unloaded elevations post-fatigue. This amounted to 36 seconds between the last repetition of the fatiguing protocol and the conclusion of the loaded motion trials, with the unloaded motion trials beginning 40 seconds and concluding 72 seconds after the last fatigue protocol repetition. It is possible that treating LTrPs rendered these muscles more able to disperse the metabolic products produced during the fatiguing exercise, leading to a decreased likelihood of producing sub-optimal MAPs secondary to changes to motoneuron excitability moderated by group III and IV afferent discharge. However, recall that the treatment for LTrPs included both stretching and light range of motion exercises (see page 212) which could increase blood flow. Furthermore, there is some evidence that SDN itself increases blood flow (see later). Since the control group received no treatment, a discussion of the mechanisms by which the employed treatment may affect neuromuscular function may help clarify whether treating LTrPs facilitates recovery from repetitive movements.

5.3.4.1 LTrP treatment interventions

Perhaps another approach to understanding how LTrPs affect MAPs post-fatigue (or indeed, any of their effects) is by determining the mechanisms by which the treatments work. An insight is provided through recent work by Langevin (Langevin, Bouffard et al. 2006; Langevin et al. 2005; Langevin et al. 2007; Langevin, Churchill & Cipolla 2001; Langevin, Storch et al. 2006),

whose results suggest there may be a mechanism of mechanotransduction occurring in the connective tissue secondary to needle movement. Though these investigations have been performed to investigate the mechanisms that underpin acupuncture, with further studies, the results may eventually help explain the effectiveness of SDN. Baldry (2005) has published a theoretical explanation of the mechanism by which SDN works, described briefly as follows. Superficial dry needling stimulates A-delta fiber mechanoreceptive nociceptors (fast conducting pain pathway), which project to the superficial zone (Lamina I) of the dorsal horn in the spinal cord. Between lamina I and II, 'stalked' cells receive direct input from A-delta fibers and have the effect of releasing the inhibitory opioid peptide enkephalin, which in turn inhibits activity in the substantia gelatinosa cells to which small, unmyelinated sensory afferents, including group IV nociceptors, project. This action can act to block noxious information conducted by group IV afferents, possibly stimulated by LTRPs. In addition, activity in the serotonergic descending inhibitory system and in the descending noradrenergic system, including the associated inhibitory effect of wide dynamic range transmission neurons, blocks the intra-dorsal horn passage of noxious information, all actions that can be initiated by needle stimulation of A-delta receptors in the skin (Baldry, PE. 2005). Confusingly, subjects receiving SDN often do not report feeling the 'needle prick', something expected if needling stimulates A-delta skin receptors. This phenomenon suggests that other factors may be in play. Assuming that Baldry's theory is valid, it provides an explanation for the increased PPTs following treatment that were observed in the current investigation, though it fails to explain the disappearance of the taut band.

This was the reason PIR was employed after SDN to encourage the muscle fibers containing contracted sarcomeres to fully lengthen, removing the taut band (Lewit & Simons 1984). Although PIR has been demonstrated to increase range of motion of TrP-affected muscles, the mechanism(s) underpinning the change in stretch perception or tolerance are not known, although alterations in muscle spindle afferent output (Rothwell 1994) and pain modulation have been proposed (Sharman, Cresswell & Riek 2006). Because of the limited information available to explain the mechanisms via which this treatment intervention removed the clinical signs of LTrPs, it may be speculated that the stimulation of A-delta mechanoreceptive nociceptors produced responses that inhibit the processing of nociceptive information at the dorsal horn, creating an opportunity to “re-educate” the muscle fibers to an increased length by PIR stretching (lengthening the contracted sarcomeres via some mechanism), resulting in an improved local circulation and the removal of metabolites that had previously stimulated the group III and IV nociceptors. With this noxious afferent activity discontinued or reduced, its effects on spinal cord reflexes and at supraspinal sites potentially resulting in sub-optimal descending commands would be reversed. Thus, removal of the clinical signs of LTrPs (principally pain on external pressure of a tender point within a taut band) normalised and possibly led to improved ability of the muscle to cope with fatigue-related noxious stimuli as well as affecting intrinsic muscle properties (e.g. reducing the heterogeneity of sarcomere length and improving cross bridge mechanics).

5.3.5 Conclusions

On the basis of the results, it can be concluded that:

1. Superficial dry needling followed by PIR stretching removes the clinical signs of LTrPs from the scapular rotator muscles and normalises the MAP of LTrP subjects post-fatigue with respect to the control group MAP in a rested state.
2. Treating LTrPs in proximal muscle groups affects the recruitment of functionally related muscles placed more distally in the kinetic chain of the upper extremity, especially where daily activities involve repetitive overhead tasks.

CHAPTER 6

CONCLUSIONS, LIMITATIONS AND FUTURE DIRECTIONS

6.1 Thesis Summary and Conclusions

With regard to LTrPs, despite a paucity of experimental evidence, current clinical opinion holds that though these neuromuscular entities allow pain-free movement, they are primarily associated with motor effects and occur commonly in 'healthy' muscles. In contrast, evidence exists to support the fact that ATrPs are prevalent and a common cause of pain in patients with musculoskeletal pain and have significant effects, including augmentation or inhibition of sensation and because of pain, movement adaptations. The primary aim of this study was to investigate the effects of LTrPs on the MAPs of key shoulder girdle muscles during scapular plane elevation of the arm, the results of which were presented in Chapters four and five. In connection with the main aim, a preliminary study was carried out to examine the frequency with which LTrPs occur in the scapular positioning muscles in a group of normal subjects, which was presented in Chapter three and summarised in the following paragraph.

The objective of the study presented in Chapter three was to determine how commonly LTrPs occur within the scapular positioning muscles of asymptomatic adults by examining healthy pain-free individuals represented by a sample of university staff and students. One hundred and fifty four healthy subjects volunteered to undergo a physical examination for the presence of LTrPs in the trapezius, rhomboids, levator scapulae, serratus anterior and the pectoralis minor muscles bilaterally. Of these subjects, 89.8% had at least one LTrP in the scapular positioning muscles

(mean=10.65 ± 6.8, range=1-27), with serratus anterior and upper trapezius harbouring the most LTrPs on average (2.46 ± 1.8 and 2.36 ± 1.3 respectively). Consistent with clinical opinion, this study found a high occurrence of LTrPs in the scapular positioning muscles. Having established that the presence of LTrPs in the scapular positioning muscles occurred commonly; the clinical significance of their presence and the question of whether they affect muscle activation patterns when raising the arm under a number of commonly occurring situations were investigated, the results of which were presented in Chapters four and five.

Chapter four presents the results of a comparative study that used sEMG to investigate MAPs in functionally related shoulder girdle muscles during elevation in the scapular plane under a number of commonly occurring conditions, including unresisted movement (unloaded), carrying a light hand-weight (loaded) and after fatiguing repetitive arm elevations both with and without external load (fatigued). The resultant MAPs of a sample that had LTrPs in the scapular rotator muscles, but not the Inf or MD, were compared with a control group who were LTrP-free. Irrespective of the experimental condition, there were a number of MAP features of the respective groups that were common and are described as follows:

The control group had a relatively stable and sequential order of muscle activation that consisted of the UT, Inf, MD, SA than LT. The time at which the arm began moving with respect to the times at which muscles were activated differed for different experimental conditions. For example, when

external load was added, the entire MAP shifted (earlier) so that UT, Inf and MD were all activated prior to movement of the arm (as opposed to only the UT in the unloaded state), a phenomenon thought possibly designed to allow extra motor units to be recruited to generate the extra force needed to cope with the increased external load. After fatigue, LTrP-free individuals activated the muscles in the same order but the MAP was 'condensed', (the muscles were generally activated in a shorter time period) and with increased variability. These findings indicate that when fatigued, although the order of activation is maintained, LTrP-free individuals display a more variable, less consistent MAP than when rested, again consistent with fatigue-related movement performance in sports or repetitive work tasks.

For healthy subjects, the neural pathways that are associated with fatigue (as well as the accompanying accumulation of metabolites, decreased pH and their resultant effects on muscle activation and movement) have been quite extensively investigated and are summarised briefly as follows as they are hypothesised to pertain to the current work:

Group III and IV nociceptors are activated by fatigue-induced substances leading to:

- Sub-optimal descending signals to the motoneuron pool, potentially inhibiting motoneurons directly or through recurrent inhibition via Renshaw cells, resulting in decreased excitability of motoneurons and muscle activation.
- Increased fusimotor drive, facilitating motoneurons and muscle activation.

- Pre-synaptic inhibition of the Ia spindle fiber, depressing the monosynaptic excitatory synapse with motoneurons and inhibiting muscle activation.
- Changes in motoneuron intrinsic properties, such as changing their activation thresholds through hyperpolarisation and changing ionic conductance secondary to changes induced by fatigue in neurotransmitters, neuromodulators and neuropeptides.

The most significant finding for the LTrP group in all experimental conditions was that the only consistent aspect of their MAP was that Inf was activated first in most trials and conditions. Beyond this finding, the order of muscle activation was not consistent within or between LTrP-affected subjects. Based on evidence describing the role of the Inf during elevation of the arm in the scapular plane, it was hypothesised that early activation in this movement was aimed at compensating for sub-optimal positioning of the scapula by LTrP-affected muscles in order to minimise compression of subacromial structures. In addition to an inconsistent order of muscle activation, generally speaking, the variability of activations times, most commonly of the UT, SA and LT (LTrP-containing muscles), but also of the functionally related muscles (Inf and MD) in some conditions, was increased in the LTrP group. These findings were not significantly worsened either by adding light external loads or performing repetitive arm elevations in the scapular plane, suggesting that the mechanisms via which LTrPs affect MAPs were not exacerbated by these variables. The findings stimulated a

discussion of the mechanisms that may underpin changes in muscle activation in LTrP-affected muscles (UT, SA and LT in the present study).

Briefly, these included:

- Changes to motoneuron excitability secondary to group III and IV afferent discharge secondary to nociceptor activation based on the fact that SP and CGRP have been identified in increased concentrations at LTrP sites in UT muscles previously.
- Decreased fusimotor drive to motoneurons secondary to unloading of muscle spindles positioned on sections of muscle fibers where sarcomeres are contracted (LTrP contraction knot). Conversely, where spindles were positioned on sections of muscle fibers where sarcomeres lengthen consequent to the development of a LTrP contraction knot, increased spindle discharge might be expected given their ability to respond to very small changes in fiber length. In combination, mis-matched spindle feedback from both within a LTrP affected muscle fiber and between spindles in the taut band region as opposed to 'normal' fibers may contribute to changes in activation of affected muscles and functionally related muscles via CNS pathways.
- Altered intrinsic properties of LTrP-affected muscle fibers including, mechanical failure due to loss of overlap of actin and myosin in lengthened sarcomeres, active insufficiency of sarcomeres under contracture, (both affecting contractile capacity) and alterations to cross bridge attachment/detachment mechanics affecting contractile speeds.

Given that MAPs of LTrP subjects were found to be different from those who were LTrP-free, the next step was to remove the LTrPs from half the LTrP subjects by treating them with SDN and PIR stretching to test the affects of treating LTrPs on MAPs. These data were presented in Chapter five as described next.

Chapter five involved randomly assigning half the LTrP subjects to treatment consisting of SDN and PIR stretching and half to a sham treatment (representing placebo intervention) that consisted of sham ultrasound therapy. After treatment (true or sham), half the subjects had no LTrPs and the remaining half did. All 'treated' subjects then repeated all sEMG investigations performed in Chapter four. The significant findings are outlined as follows:

- Sham ultrasound treatment produced no difference from the pre-interventions MAPs.
- SDN and PIR stretching resulted in MAPs that were not significantly different from the control group in the unloaded and loaded experimental conditions, confirming the high likelihood that the presence of LTrPs in the upward scapular rotators produced the different (compared with healthy subjects) MAPs.
- In the unloaded and loaded conditions, treating LTrPs 'normalised' and presumably optimized MAPs at the time of treatment, however it is not known how long this affect remains since no follow up testing was performed.

- When fatigued, treated LTrP subjects had the same order of muscle activation as the control group post-fatigue, however some differences in mean activation times and variability of these activation times remained, suggesting there are differences in the way these populations responded to fatigue-inducing repetitive movements.
- Treating LTrPs results in activation patterns post-fatigue no different to those produced by healthy subjects in a rested state, implying that treatment of LTrPs may result in an increased ability to maintain an optimal MAP when exposed to fatigue.

In conclusion, based on the results of the current work, LTrPs commonly occur in scapular positioning muscles and do have deleterious effects on MAPs and thus affect motor control mechanisms. Treating LTrPs with SDN and PIR stretching increases PPTs and removes associated taut bands and “normalises” the MAP during scapular plane elevation in commonly occurring conditions, at least transiently.

6.2 Limitations

The results of the three closely linked studies must be considered in light of a number of limitations.

6.2.1 The Prevalence of Latent Trigger Points (LTrPs) in the Scapular Rotator Muscles in Healthy Subjects

- (i) The 154 subjects were all volunteers from one environment (university campus) as opposed to randomly selected from a larger population.
- (ii) For this study to be considered an epidemiological study, the sample size was small and thus would limit the external validity of the results.
- (iii) Data collections stopped once 14 LTrP-free subjects were identified that could act as the control group in the remainder of the experimental program. Therefore, the sample size of this study was not pre-determined but was determined by the duration of time required to identify 14 LTrP-free subjects.

6.2.2 The effects of LTrPs on muscle activation patterns during scapular plane elevation (Chapter 4) and The effects of treating LTrPs on MAPs during scapular plane elevation (Chapter 5)

- (i) No blinding was employed in this study, that is, the same investigator performed the LTrP examination, the sEMG

evaluations, the LTrP treatment interventions, the re-examination fro LTrPs and the repeated sEMG evaluations post-treatment. The aspect of the experimental procedures where no blinding had the greatest potential to affect the results was at the second LTrP examination after the respective interventions (treatment and sham treatment). The fact that an algometer was used to measure PPTs prior to and post-treatment interventions made the LTrP examinations more objective and helped to minimise the lack of examiner blinding.

- (ii) A power analysis resulted in the number of each final group being calculated as 14. The LTrP groups (N=14 treatment group; N=14 placebo treatment group) filled first, however it took 154 examinations to find 14 volunteers to form the control group. In itself, this elucidates how uncommon it is to be LTrP-free in the scapular positioning muscles.

- (iii) Spectral analysis of the sEMG signals was not carried out to confirm the presence of fatigue, nor was the duration of muscle contraction extracted from the recordings. Though both of these analyses may have added additional information regarding muscle activation patterns during fatigue, they most likely do not weaken the findings of the analysis that was performed.

6.3 Future Directions

The results of Chapters four and five demonstrated that LTrPs affect MAPs during elevation of the arm in the scapular plane in various commonly occurring conditions. The principle recommendation for future investigations to arise from this work are to investigate the mechanisms via which LTrPs may exert their effects on MAPs would provide a greater understanding of the effects of these “sub-clinical” entities and might lead to the development of interventions by which painful overload conditions that may develop secondary to LTrP-mediated sub-optimised motor control, could be prevented or better managed. Further microdialysis studies in the vein of Shah and colleagues (2005) to further illuminate the chemical compounds at TrP sites would be useful and may help clarify which pharmaceuticals might be a useful adjunct in treating or managing MPS. With a clear understanding of the biochemicals involved, studies investigating the neural pathways, presumably predominantly by group III and IV afferent fibers due to chemonociceptor stimulation, but also investigation into which other afferent fibers may fire in association with the presence of LTrPs. Studies, perhaps using similar methodologies to those performed by Gandevia and Taylor’s group on the neural mechanisms underpinning fatigue processes, might be useful to elucidate the sites within the CNS where LTrP-related input is being processed. Of particular interest are spinal cord reflexes, Renshaw cells and the fusimotor system and muscle spindle receptors and how these systems or cells may be affected by LTrP-related inputs and ultimately affect motoneuron firing.

Another area of focus may be the muscle fibers forming the LTrP taut band. Aspects that might help shed light on LTrP effects on motor control are intersarcomere dynamics, cross bridge attachment/detachment mechanics and the effects of the presence of a taut band on the rest of the muscle (possibly via the motor unit configurations) and how these elements affect the muscle's ability to react to neural commands and generate force, especially in cooperation with other muscles and joints.

Furthermore, since all the testing for the current study was performed in one day, follow up sEMG evaluations to test the duration of the effects of treatment of LTrPs on MAPs would provide further information regarding the nature of the LTrP effects on MAPs and provide insights into the efficacy of management programs. In addition, spectral analysis of sEMG would provide objective evidence of the degree of fatigue associated with LTrPs and is recommended in future studies.

APPENDICES

APPENDIX A

Plain Language Statement Given to Participants



RESEARCH PROJECT INVOLVING HUMAN

1 / 12 /99

Dear Participant,

RE: PARTICIPATION IN AN RMIT RESEARCH PROJECT

Thanks for your interest in this project. My name is Karen Lucas and I am undertaking a PhD degree in the Department of Chiropractic, Osteopathy and Complementary Medicine at RMIT University. My research project is titled *The effect of latent myofascial trigger points in the scapular rotator muscles on the recruitment pattern in the lower trapezius, serratus anterior and infraspinatus muscles during elevation of the arm in the scapular plane* and aims to investigate muscle recruitment patterns of the shoulder girdle of individuals with and without knots in specific muscles to see if there are any differences.

As a subject in this study, you will be invited to come to the Chiropractic research lab (room 201.5.28). You will be assessed for latent trigger points, or knots in your muscles around your shoulder blades by feeling them for any painful spots. Immediately after this assessment, you will have 16 surface electrodes (adhesive patches) taped to your skin on your back and asked to perform some simple movements with your arms. These electrodes are attached to fully isolated amplifiers which will protect you from any possible power surge. This test will enable us to measure the electrical activity in your muscles.

Next you will be given 20 minutes of muscle therapy, depending on what was found in your initial assessment. This therapy will be either myofascial dry needling, in which a very thin disposable acupuncture needle will be placed in your muscle for about 5 minutes. Most people experience no discomfort during or after this treatment, but it is possible to feel a mild, dull ache for 5 minutes up to 24 hours post treatment. All infection control procedures will be strictly adhered to. The other muscle therapy is gentle massage of the back and neck. You will not be able to choose which therapy you receive.

Depending on what has been found in your muscles, you may have to have your muscle activity re-tested using the electrodes taped to your back. It would be expected that neither of these therapies should effect your health adversely.

The entire process should take between 60 and 90 minutes, depending on what is found in the initial assessment.

The data collected in this project may give us new knowledge on how the shoulder muscles function when raising the arm. It may also provide insight into how to prevent chronic shoulder pain.

Participation in this project is totally voluntary and you can withdraw, without prejudice, at any stage, including the withdrawal of any previously supplied unprocessed data. As a participant, you are invited to, and should, ask for clarification regarding any aspect of your participation that may be concerning you. Data from this project may be used in a presentation or a published article at a later date, but your personal information and any data collected from you will not be personally identified and your confidentiality will be protected at all times.

Thank you for your interest in this project.

Karen Lucas
BAppSc(Human Movement)(Hons.)
AdDip (Myotherapy)
Ph: 9925 7596

Dr Barbara Polus
ApSci; MSc; PhD
Ph: 9925 7714

Dr Peter Rich
Dip PE; BSc; MSc; PhD

Any queries or complaints about your participation in this project may be directed to the Secretary, RMIT Human Research Ethics Committee, RMIT, GPO Box 2476V, Melbourne, 3001. The telephone number is (03) 9925 1745.

2.

- (c) The project is for the purpose of research and/or teaching and not for treatment;
- (d) I have been informed that the confidentiality of the information I provide will be safeguarded.

Signature: _____ Date: _____
(Participant)

Signature: _____ Date: _____
(Witness to signature)

Participants should be given a photocopy of this consent form after it has been signed.

Any queries or complaints about your participation in this project may be directed to the Secretary, RMIT Human Research Ethics Committee, RMIT, GPO Box 2476V, Melbourne, 3001. The telephone number is (03) 9925 1745.

APPENDIX C

INTRA-EXAMINER RELIABILITY IN LATENT MYOFASCIAL TRIGGER POINT EXAMINATION

C.1 Introduction

The aim of this study was to investigate the intra-examiner reliability of identifying latent trigger points (LTrPs) in the scapular rotator muscles of healthy adult subjects. Eight subjects (4 male, 4 female, aged 22-55 years, mean 38.2 years), who experienced no pain in their upper back, neck, shoulders or arms, had their scapular rotator muscles examined for the clinical characteristics of LTrPs. The muscles examined for LTrPs were the pectoralis minor, levator scapulae, serratus anterior and all parts of the trapezius, and rhomboids.

Gerwin and colleagues (1997), in their interrater reliability of trigger point (TrP) examination study defined the clinical characteristics of TrPs as follows: taut bands (TB), a tender point (TE) within the TB, pain reproduction (Rep P), referred pain (Ref P) and local twitch response (LTR). Those definitions were also used in the present study except for the pain reproduction, as LTrPs do not elicit pain at rest. The term pressure-pain threshold (PPT) was used instead and defined as the number of kilograms per square centimetre that had to be exerted in a direction perpendicular to the skin surface before the sensation of pressure became the sensation of pain. The PPT was measured with an algometer (Activator Methods, Phoenix, AZ) using the procedure validated by Fischer (1986). A LTrP was defined as a TE within a TB that may or may not elicit a LTR or Ref P in response to snapping palpation or direct compression respectively and had a PPT of less than expected in normal muscle tissue. In this study, the palpation pressure used to elicit a pain response was standardized subjectively and defined as pressure that would not usually cause a pain response in normal muscle tissue, as judged by experienced examiners. In contrast, Hong and co-workers (1996) found that even when normal muscle tissue near a LTrP was compressed, 23% of

subjects reported a referred pain response if there was no limit placed on how much pressure could be employed. This finding suggests that it may be helpful to further objectify the amount of pressure used to identify a LTrP, in order to decrease the likelihood of false-positives.

In earlier work, Fischer (1987) used pressure algometry to measure the pressure-pain threshold (PPT) of normal back and shoulder girdle muscles. A mathematical algorithm was used to calculate the PPT below which a muscle could be considered abnormal. Fischer noted that males and females had different PPT's for the same muscles and that PPT's decreased in a cephalad direction. On the basis of the cited studies, the definition used to identify a LTrP in this study became:

A tender point within a palpable taut band of skeletal muscle that had a PPT of less than that expected in normal muscle tissue (see Fischer's values, 1987 and table 1), with or without referred pain or a LTR.

Table C.1: Lowest PPT (kg/cm²) at which a muscle can be considered 'normal' Fischer, 1987).

	Males (kg/cm²)	Females (kg/cm²)
Upper trapezius	2.9	2.0
Scapular muscles	3.9	2.8

C.2 Methods

C.2.1 Subjects

After gaining approval from the RMIT University Human Research Ethics Committee, 8 pain-free subjects (4 female, 4 male; mean age 38 ± 9.1 years)

who volunteered for the study had their scapular rotator muscles examined for the clinical characteristics of LTrPs. The muscles examined for LTrPs were the pectoralis minor and serratus anterior (examined lying supine), all parts of the trapezius and rhomboids and the levator scapulae (examined lying prone). Subjects were excluded if they reported any pain in the back, neck or either upper limb any time in the previous week.

A therapist who had been trained and was experienced in LTrP examination used the identification procedures outlined by Simons et al (1999, p. 116-7) briefly described as follows. The subject was lying on a table in a warm and relaxed state with the upper body disrobed. 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 normal muscle fibers are still slack but the fibers of any taut bands are placed under additional tension, which renders them most easily distinguishable from the normal fibers. Next, cross-fibre palpation was used to identify any taut bands (fig. 1). Fiber examination occurred via flat palpation for all muscles except the upper trapezius, 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 (fig. 2) using the procedure validated by Fisher (1987). If the PPT was less than that of 'normal' muscle tissue (table 1), the tender point was defined as a LTrP and its position was documented on an enlarged body diagram. The subject was also asked if the pain referred elsewhere and the TE was stimulated with snapping palpation to attempt to elicit a LTR. Pressure-pain threshold 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 60 seconds) due to the fact that LTrPs can be inactivated by sustained pressure (Hong, 1999).



Figure C.1: Palpation perpendicular to the direction of the muscle fibers to identify the taut band.



Figure C.2: Using the algometer to measure the PPT of a subjectively painful nodule.

Subjects were examined for LTrPs three times. The second examination was 30 minutes after the first and the third examination was 24 hours after the first. All the clinical findings for each LTrP were recorded and the locations were described using anatomical landmarks and drawn on a body outline by the examiner for later analysis. The third examination 24 hours later served to decrease the examiner bias that may occur when the examiner remembers the area in which a LTrP was located previously. The time between examinations could not be too long as it is possible that the LTrP may be affected by the examination process itself or the activities in which the subject participated between examinations. The order of muscles assessment was random for each subject.

C.2.2 Statistical analysis

To assess the agreement between the findings of each examination the kappa statistic, which reports pairwise judge agreement corrected for chance agreement (Cohen, 1960) was used. The kappa statistic is dependent upon the presence of two or more choices so when there is 100% agreement between examinations, the kappa value will be low (Hobart et al, 1996). When this occurs, the percentage agreement will be reported in the results.

To assess the reliability between algometer scores for each trigger point in each examination, intraclass correlations (ICC) were performed. The ICC is a reliability coefficient that is calculated using variance estimates obtained through an analysis of variance, reflecting both the degree of correspondence and agreement among scores (Portney and Watkins, 1993).

C.3 Results

Kappa scores are displayed in Table 2 then classified in Table 3. Mean PPTs for each muscle are reported in Table 4 and the ICCs for the PPTs between examinations are reported in Table 5.

Table C.2: Kappa Scores for each combination of examinations.

Muscle	Exam 1 v 2	Exam 2 v 3	Exam 1 v 3
Pectoralis minor	0.71	1	0.71
Serratus anterior	1	0.6	0.6
Upper trapezius	1	1	1
Middle trapezius	1	0.5	0.5
Lower trapezius	0.75	0.33	0.33
Rhomboids (together)	1	1	1
Levator scapulae	1	0.35	0.38

Portney and Walkins (1993) reported that Landis and Koch suggested the following levels of agreement for kappa statistics:

- > 0.8 = excellent agreement
- 0.6-0.8 = substantial agreement
- 0.4-0.6 = moderate agreement
- < 0.4 = poor to fair agreement

Table C.3: The extent of agreement between examination findings for each muscle according to Portney and Walkins (1993) definitions for kappa statistics.

Muscle	Exam 1 v 2	Exam 2 v 3	Exam 1 v 3
Pectoralis minor	substantial	excellent	substantial
Serratus anterior	excellent	substantial	substantial
Upper trapezius	excellent	excellent	excellent
Middle trapezius	excellent	moderate	moderate
Lower trapezius	substantial	poor	poor
Rhomboids (together)	excellent	excellent	excellent
Levator scapulae	excellent	poor	poor

Table C.4: Mean algometer scores where LTrPs had been identified (PPT) for each muscle and examination.

Muscle	Exam 1	Exam 2	Exam 3
Pectoralis minor	3.3	3.1	2.4
Serratus anterior	3.6	2.4	2.8
Upper trapezius	2.8	3.0	2.7
Middle trapezius	3.4	3.6	2.4
Lower trapezius	3.6	3.4	3.0
Rhomboids (together)	3.9	3.6	2.9
Levator scapulae	3.5	3.7	2.2

Table C.5: ICCs for PPTs measured with the algometer (kg/cm²) between examinations for all muscles.

<i>ICC for PPTs</i>	Exam 1 v 2	Exam 2 v 3	Exam 1 v 3
	0.92	0.33	0.28

C.4. Discussion and Conclusions

For each muscle examined, the agreement was either “substantial” or “excellent” between examinations 1 and 2. That is, the point on the body identified as a LTrP in examination 1 was highly likely to also be identified as a LTrP in examination 2 by the same examiner. Additionally, if there was no LTrP identified in a muscle in examination 1, there was likely to be no LTrP found in that muscle in examination 2. With regard to PPTs, there was no significant difference in PPT scores between groups where LTrPs had been identified. There were however significant differences in PPTs between where there was no LTrP identified and where there was. In other words, the PPT is significantly higher in muscle where there are no clinical characteristics of LTrPs (taut band, tender point) when compared to the PPT of muscle where these signs have been identified. In addition, the ICC for the mean PPT for each muscle between examinations one and two was large (ICC of 0.5 or higher = “large”; ICC for examinations one and two=0.92). However, where ICCs were performed between examinations that occurred on different days, the ICCs did not produce good agreement, being classified as either moderate (ICC of 0.3-0.5) or small (ICC of 0.1-0.3).

Based on these results, it was concluded that:

1. Examinations performed on the same day for LTrPs in scapular positioning muscles, performed by the same examiner using the process described above was reliable.
2. Where repeat examinations were performed more than 24 hours apart, PPTs decreased and the agreement (based on kappa scores) decreased to moderate or poor for the middle and lower trapezius and levator scapulae for this examiner.

APPENDIX D
LTrP Group Characteristics (Chapters 4 and 5)

SUBJECT	Tx. GROUP	AGE	OCC	DOM HAND	SEX	PM D	SAD	UT D	MT D	LT D	RH D	LS D	TOTAL SRM
1	Tx	20	Sdt	R	M	1	2	1	1	2	2	2	20
2	Tx	20	Sdt	R	M	0	3	3	0	1	2	2	16
3	Tx	21	Sdt	R	F	2	3	2	1	1	2	1	20
4	Tx	22	Sdt	R	F	1	1	1	0	0	0	1	7
5	Tx	27	Sdt	R	F	0	0	1	0	0	1	0	6
6	Tx	29	Sdt	R	F	1	1	1	0	1	0	0	12
7	Tx	30	Sta	R	F	1	0	1	0	1	0	0	3
8	Tx	31	Sta	R	M	1	2	2	1	2	2	2	19
9	Tx	33	Sdt	R	F	1	2	1	0	1	0	1	16
10	Tx	35	Sta	R	M	0	2	1	0	0	0	0	9
11	Tx	37	Sta	R	F	2	1	0	0	1	0	0	6
12	Tx	40	Sta	R	F	1	2	2	0	2	2	1	14
13	Tx	46	Sta	R	M	0	2	2	1	0	1	0	13
14	Tx	53	Sta	R	M	2	2	2	2	2	2	2	21
15	PI	19	Sdt	R	F	0	0	2	0	1	3	0	11
16	PI	23	Sdt	R	M	0	2	1	0	1	1	0	12

SUBJECT	INT. GROUP	AGE	OCC	DOM HAND	SEX	PM D	SAD	UT D	MT D	LT D	RH D	LS D	TOTAL SRM
17	PI	24	Sdt	R	M	0	0	1	1	1	2	1	12
18	PI	24	Sdt	R	F	1	3	1	0	1	1	0	14
19	PI	26	Sdt	R	F	1	2	0	0	1	1	0	8
20	PI	26	Sdt	L	M	0	3	1	0	1	2	0	12
21	PI	30	Sta	R	M	2	2	2	0	0	0	1	13
22	PI	34	Sdt	R	M	1	2	2	1	1	1	1	14
23	PI	45	Sta	R	M	0	2	1	1	1	2	1	14
24	PI	46	Sta	R	M	0	1	1	0	0	0	1	14
25	PI	47	Sta	R	M	2	3	1	1	2	2	2	21
26	PI	49	Sta	L	M	1	3	2	0	2	0	0	14
27	PI	53	Sta	R	F	1	0	2	0	0	0	0	8
28	PI	58	Sta	R	M	2	3	2	0	2	2	1	22
Mean		33.86				0.86	1.75	1.39	0.36	1.00	1.11	0.71	13.50
SD		11.4				0.76	1.04	0.69	0.56	0.72	0.96	0.76	4.99

ITx. group=treatment LTrP subject was randomly assigned to; Tx=LTrP treatment group; PI=LTrP placebo group; Occ=occupation; Std=student; Sta=staff; Dom hand=hand dominance; M=male; F=female; PM D=pectoralis minor dominant side; SA D=serratus anterior dominant side; UT D=upper trapezius dominant side; MT D=middle trapezius dominant side; LT D=lower trapezius dominant side; Rh D=rhomboids major and minor combined dominant side; LS D=levator scapulae dominant side, Total SRM=total number of LTrPs in the scapular rotators as a group.

APPENDIX E

Myofascial TrP Differential Diagnoses

Myofascial TrPs can result from various mechanical and systemic causes that must be identified and treated specifically (Gerwin, R. 2004). For example, Simons and colleagues (Simons, D., Travell & Simons 1999) identified two conditions that often underpin MPS but are commonly overlooked: fibromyalgia and joint-mediated pain or dysfunction. They pointed out that these conditions often interact but require different diagnostic examination techniques and significantly different treatment approaches. More specifically, TrP pain can mimic specific pain conditions (eg radiculopathy, angina) from which they must be differentiated in order that the correct treatment be implemented. For a comprehensive list, readers are referred to table 2.5 in (Simons, D., Travell & Simons 1999).

In a review on the differential diagnosis of TrPs, Gerwin (2004) outlined the following conditions that might require investigation and specific treatment when TrPs have been identified (Gerwin, R. 2004):

1. *Delayed onset muscle soreness (DOMS)* which occurs secondary to unaccustomed exercise, usually involving eccentric contractions, and is the result of local muscle damage, inflammatory changes and nociceptor sensitisation (Proske & Morgan 2001).
2. *Hypermobility syndromes* which produce multiple mechanical stresses secondary to ligamentous laxity causing poor joint stabilisation and resultant muscle overload.
3. *Forward head posture* and the resulting muscular overload often associated with posterior displacement of the mandible, temporomandibular joint pain, headache and upper airway obstruction.
4. *Pelvic torsion-related pain* caused by chronic anterior pelvic tilt. Pain arises from the muscular overload required to adjust to the pseudo-leg-length inequality or pseudoscoliosis.
5. *Sacroiliac joint dysfunction or hypomobility* can cause pelvic and spine dysfunction that results in painful widespread axial muscle TrPs.

6. *Somatic dysfunction or muscle-joint dysfunction*, a painful limitation of range of motion caused by muscular restriction of joint motion, seen commonly where a vertebral rotation or lateral displacement is sustained by persistent TrPs in paraspinal muscles.
7. *Static overload* which occurs when mechanically stressful positions are held for prolonged periods of time causing overload and fatigue of the active muscles. This situation results from many common workplace and daily tasks which need to be specifically addressed.
8. *Nerve root compression* can present with TrPs, treatment of which may bring transient relief of the muscle pain, but the TrPs will recur until the nerve root is decompressed.
9. *Muscle imbalance* resulting from muscle weakness (from any cause) can produce a musculoskeletal imbalance leading to mechanical asymmetries and muscular overload of the compensating muscles.

The following *systemic illnesses* have been associated with the presence of TrPs, although in most cases, causal relationships have not been confirmed:

1. *Autoimmune disorders* including lupus, Sjogren's and polymyalgia rheumatica.
2. *Infectious diseases* such as Lyme disease or post-Lyme disease syndrome, mycoplasma pneumonia, Chlamydia pneumonia and parasitic disease.
3. *Allergies*, when left untreated, may cause widespread myalgia that resolves when the allergies are treated.
4. *Viscero-somatic pain syndromes*, in which internal organ dysfunction is associated with somatic segmental referred pain syndromes. Examples include endometriosis causing abdominal myofascial pain, interstitial cystitis and irritable bowel syndrome both associated with chronic pelvic pain syndromes and liver disease that causes local abdominal or referred shoulder regional pain.
5. *Brain tumour and base of skull pain* may be caused by primary or secondary posterior fossa tumours with associated suboccipital or upper cervical TrPs that can be transiently de-activated.

APPENDIX F

Myofascial TrP Injections and Other TrP Therapies

F.1 Injection Therapy for TrPs

As more is known about the biochemical milieu and pathoneurophysiology of MPS, clinicians and researchers are trialling new pharmacological substances in injection therapy to treat myofascial TrPs. As this aspect of TrP therapy falls outside the professional scope of this author, only a basic review of this literature has been performed.

F.1.1 Botulinum Toxin A

More trials of various natures are appearing in the medical literature testing the efficacy of injecting botulinum toxin A (BTX A) into myofascial TrPs, though no systematic review has been performed as yet. Investigators have had mixed results ranging from one prospective, randomized, double-blind, placebo-controlled, 12-week, multicentre study finding an efficacious affect beyond placebo (Gobel et al. 2006; Wheeler, Goolkasian & Gretz 1998) and one study finding a positive within-subject effect for BXT A injections but no improvement over saline. Conversely, more trials have found no difference to saline (Ferrante et al. 2005; Ojala, Arokoski & Partanen 2006; Porta & Maggioni 2004; Qerama et al. 2006). Although one trial did not find any benefit of BXT A above placebo, the authors suggested that due to many

subjects who received two BXT A injections becoming asymptomatic, that further investigations were warranted (Wheeler, Goolkasian & Gretz 1998).

Graboski and co-workers (Graboski, Gray & Burnham 2005) found no difference in a trial comparing BTX A with bupivacaine in duration or magnitude of pain relief, function, satisfaction or cost of care cost of injectate excluded. They suggested that given the high cost of BTX A, bupivacaine would be a more cost-effective injectate for TrPs.

In a trial comparing TrP injection with BTX A to dry needling and lidocaine injection, the authors considered lidocaine injection more practical due to the fact it caused fewer disturbances than dry needling and was more cost effective than BTX A injection. An additional recommendation was to use BTX A in TrP patients who were resistant to conventional treatments (Kamanli et al. 2005).

F.1.2 Botulinum Toxin B

A small uncontrolled, single-center, outpatient, open-label study (Lang 2004) to evaluate the clinical safety and efficacy of botulinum toxin type B (MYOBLOC) in reducing myofascial pain associated with piriformis syndrome suggested the possibility that botulinum toxin type B may be of potential benefit in the treatment of pain attributed to piriformis syndrome.

In summary, regarding the injection of botulinum toxin into TrPs, it is the opinion of well respected researchers and clinicians that there are many other treatment modalities that are at least as effective as these injections (Simons, D. G. & Dommerholt 2006).

F.1.3 Anti-inflammatory injectates

In earlier times, injection of corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDs) into TrPs were more routinely performed, but once it was established there was no advantage over pain-killing substances such as lidocaine, these anti-inflammatory substances fell from favour. Recently, new injectable medications are being trialled that more directly target specific components of the pathophysiology of the TrP. As an example, one such study is outlined below.

Muller and Stratz (Muller & Stratz 2004) performed a pilot study comparing the effect of 5-HT₃ receptor antagonist (tropisetron) injections to analgesic (prilocaine) injections on visual analogue scales (VAS). They found a significant decrease in pain at three hours and subsequently at seven days on the VAS and a higher percentage of subjects that categorised themselves as “improved” at eight weeks in the tropisetron group compared with the prilocaine group. The authors indicated that the analgesic action of the 5-HT₃ receptor antagonist tropisetron manifested rapidly and lasted over a long duration (eight weeks) and probably had an anti-inflammatory effect which could be attributed to the inhibited release of substance P and other

neuropeptides from the nociceptors and the blocked release of inflammatory substances from macrophages, and monocytes.

F.1.4 Vitamin B₁₂

Although vitamin B₁₂ deficiency is recognised as a condition that may promote or perpetuate MPS and that this deficiency must be rectified as part of the treatment program (Gerwin, R. D. 2005), some clinicians have employed injection of vitamin B₁₂ directly into the TrPs itself. No RCTs on the injection of vitamin B₁₂ into TrPs was found in the literature.

F.2 Other Therapies Used For Treating TrPs

A very brief overview of 'other therapies' for treating TrPs is presented.

F.2.1 Laser Therapy

Three types of lasers, Ga-As (Altan et al. 2005; Gur et al. 2004; Hakguder et al. 2003); He-Ne (Ilbuldu et al. 2004; Snyder-Mackler et al. 1989) and infrared diode (Ceccherelli et al. 1989) have been used to treat TrP pain. All but one study (Altan et al. 2005), showed at least short-term success in treating TrP pain. In two studies, a significant positive effect persisted at three months (Ceccherelli et al. 1989; Gur et al. 2004), but at six months no difference was noted when compared to the control group who received

placebo laser (Ilbuldu et al. 2004). It should be noted that Altan and co-workers (2005) showed no advantage over placebo (Altan et al. 2005), however both the treatment and placebo groups participated in a concurrent program of isometric exercises and stretching, which the authors suggested may have confounded the conclusions.

F.2.2 Electrotherapies

Six types of electrotherapies have been reported in the MPS literature including transcutaneous electrical nerve stimulation (TENS), electrical muscle stimulation (EMS), high voltage galvanic stimulation (HVGS), frequency modulated neural stimulation (FREMS) and interferential current (IFC) (McMakin 2004; Rickards 2006).

In a study judged as having poor internal validity (Rickards 2006), HVGS reduced pain scores at 15 days post-treatment but did not decrease analgesic use (Tanrikut et al. 2003). Interestingly, in a case series study using frequency-specific microcurrent to treat chronic low back myofascial pain (McMakin 2004), the author reported a statistically significant 3.8-fold improvement in pain reduction, suggesting a more thorough investigation was warranted on the basis of the results. Similarly, TENS has been shown to reduce TrP pain more effectively than EMS in two studies (Ardic, Sarhus & Topuz 2002; Hsueh et al. 1997) and has also been found to be superior to ultrasound in significantly reducing pain intensity (Hou, C. R. et al. 2002). The same study also revealed that when used in combination with other physical

therapy modalities or manual techniques both TENS and IFC produced a reduction in pain intensity. A frequency dependent effect was identified by Graff-Radford and colleagues (1989) who found superior pain reducing effect when TENS was used at 100 hertz (Hz), 250ms stimulation compared with 2Hz, 250ms (Graff-Radford et al. 1989). In all these studies the findings were based upon immediate post-treatment effects, so that the medium or long-term effects are not known. Related work by Smarnia and co-workers (2005) shed some light on the duration of the effects of TENS, finding an immediate pain relieving effect, however the effect did not persist at the one month follow up examination (Smania et al. 2005). Perhaps more positive were the results of the work performed by Farina and co-workers (2004), in which they evaluated TENS and Frequency Modulated Neural Stimulation (FREMS) for their ability to reduce TrP pain levels and alleviate the other clinical characteristics of TrPs. Both showed improvement at one month, but only the FREMS group maintained their improvement at 3 months (Farina, S. et al. 2004).

F.2.3 Ultrasound Therapy

In a systematic review on non-invasive TrP treatments, Rickards (2006) reported that “standard” ultrasound applications had no effect on TrP pain beyond placebo in two studies (Gam et al. 1998; Majlesi & Unalan 2004). Similarly, Lee and colleagues (2002) demonstrated no significant difference between ultrasound and placebo ultrasound in TrP treatment (Lee, J. C., Lin & Hong 2002). In contrast, a trial that received a low validity score in

Rickard's systematic review (Rickards 2006), high power pain threshold ultrasound (HPPT-US) significantly reduced TrP pain intensity compared with conventional ultrasound (Majlesi & Unalan 2004), though any adverse effects of this practice have not been fully investigated.

F.2.4 Magnetic Therapy

In recent investigations that show promise, in a small study (n=9 in each group), Smania and co-writers (2003) reported that repetitive magnetic stimulation (rMS) produced significantly better results than placebo in reducing TrP pain in the upper trapezius (Smania et al. 2003), while in a follow up and larger study (n=53 assigned to 3 groups) (Smania et al. 2005), the same authors found rMS produced significant positive changes in treatment outcomes up to three months following treatment when compared to TENS and placebo ultrasound.

APPENDIX G

Electrode Positioning Procedure to Minimise Cross-Talk for the Infraspinatus, Lower trapezius and Serratus Anterior Muscles

G.1 Aim

To ensure the surface electromyographical (sEMG) signal recorded was valid for the infraspinatus (no cross-talk from posterior deltoid), lower trapezius (no cross-talk from latissimus dorsi) and the serratus anterior (no cross-talk from pectoralis major).

G.2 Methods

G.2.2 Subject Preparation

Pre-gelled, silver-silver-chloride surface electrodes (Red Dot Paediatric surface electrodes, Melbourne) were placed over the following muscles: infraspinatus, posterior deltoid, teres minor, lower trapezius, latissimus dorsi, serratus anterior and pectoralis major. Prior to electrode application, the subjects' skin was shaved, abraded and wiped with alcohol in order to reduce skin impedance. All electrodes were attached in the positions described in the table below with an inter-electrode distance of 20mm. The dominant-hand side was tested.

Table G.1: Testing position, electrode position and test action for all muscles with potential for cross-talk.

MUSCLE	ELECTRODE POSITION	TESTING POSITION	TEST ACTION
Infraspinatus	4cm below the scapular spine, on the lateral aspect of the infraspinous fossa, 2cm apart and parallel to the scapular spine	90° elbow flexion, no shoulder flexion. Possible slight abduction of arm.	Resisted external rotation. Some arm abduction may also occur
Posterior deltoid	2cm below the lateral aspect of the scapular spine, on an oblique angle in line with the fibres.	Arm at side, elbow flexed to 90°.	Resisted extension of the arm.
Lower trapezius	Two finger-breadths lateral to the spinous processes at the level of the inferior angle of the scapula, on an oblique angle in line with the fibres.	Arm abducted to 140°	Hold against gravity.
Latissimus dorsi	Three finger-breadths distal to and along the posterior axillary fold, parallel to the lateral border of the scapula	Elbow extended, arm abducted 30° in the coronal plane and internally rotated.	Resisted extension and internal rotation
Serratus anterior	Below the axilla, anterior to the latissimus, placed vertically over ribs 4-6.	Elbow flexed 45°, shoulder abducted 75° and internally rotated 45°.	Resisted scapular protraction
Pectoralis major	Horizontal placement 4 finger-breadths below the clavicle, medial to the anterior axillary border.	Elbow flexed 90°, shoulder abducted 75°.	Horizontal adduction (press palms together)

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