

An Investigation into the Effectiveness of Latent Myofascial Trigger Point Dry Needling on Muscle Activation Patterns



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Abstract

Title: An Investigation into the Effectiveness of Latent Myofascial Trigger Point Dry Needling on Muscle Activation Patterns

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Background: Recent research on latent trigger points (LTrP) provides an opportunity to investigate the efficacy of trigger point (TrP) treatments without pain influencing results. One proposed negative outcome of LTrPs is an alteration in muscle activation patterns (MAPs), which is also associated with scapular dyskinesia and shoulder pathology. Using subjective (scapular dyskinesia tests) and objective (surface electromyography (sEMG) data) outcome measures to assess the efficacy of TrP treatment would be of benefit to both clinicians and researchers.

Aim: To investigate the effectiveness of dry needling versus manual release and placebo dry needling on LTrPs in periscapular muscles.

Methods: Inter- and intra-tester reliability and validity of the scapular dyskinesia test (SDT) and the scapular control test (SCT) were examined on 30 participants, prior to the inclusion of one in the main study. ICC analysis was used to assess reliability and t-tests were performed on sEMG data to assess validity. The effectiveness of LTrP dry needling, manual release, and placebo dry needling was then assessed on 60 participants to determine their effects on MAPs. Both subjective (SCT) and objective (sEMG data) outcome measures were utilised to assess the efficacy of the individual treatments. Wilcoxon signed rank and Kruskal-Wallis tests were carried out on the SCT data and a mixed between-within ANOVA was used for the sEMG data.

Results: The SDT and SCT demonstrated good to excellent inter- and intra-tester reliability, however they were not valid at determining the presence of altered MAPs. There were no statistically significant differences in MAPs after any of the interventions, as assessed using both the SCT and sEMG data.

Conclusion: The use of the SDT and the SCT to determine abnormalities in MAPs may be unjustified. LTrP dry needling and manual release treatments were ineffective at altering MAPs and further research is needed to assess their efficacy.

Declaration

My submission as a whole is not substantially the same as any that I have previously made or currently am making, whether in published or unpublished form for a degree, diploma, or similar qualification at any university or similar institution. I am the author of this thesis and declare that the work submitted is my own work, that any data presented is accurate, were collected and analysed by myself and that appropriate credit has been given where reference has been made to the work of others.

Signature: _____

Michael Donohoe

Date: _____

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List of Abbreviations

2-D	2-dimensional
3-D	3-dimensional
5-HT	5-hydroxytryptamine, serotonin
AC	Acromioclavicular
ACh	Acetylcholine
AChE	Acetylcholinesterase
AChR	Acetylcholine Receptors
ADP	Adenosine Diphosphate
ASIC	Acid-sensing Ion Channels
ATP	Adenosine Triphosphate
ATPase	Adenosine Triphosphatase
ATrPs	Active Myofascial Trigger Points
BK	Bradykinin
Ca ²⁺	Calcium Ions
CGRP	Calcitonin Gene-related Peptide
CI	Confidence Interval
cm	Centimetre
CNS	Central Nervous System
CPD	Continual Professional Development
EMG	Electromyography
GH	Glenohumeral
GIRD	Glenohumeral Internal Rotation Deficit
H ⁺	Hydrogen Protons
ICR	Instantaneous Centre of Rotation
IL-1	Interleukin-1
IL-6	Interleukin-6
κ	Kappa
K ⁺	Potassium
LBP	Low Back Pain

LTrPs	Latent Myofascial Trigger Points
MAPs	Muscle Activation Patterns
MEPP	Miniature Endplate Potential
min	Minutes
ml	Millilitre
mm	Millimetre
MPS	Myofascial Pain Syndrome
MVC	Maximal Voluntary Contraction
NDI	Neck Disability Index
NRS	Numerical Rating Scale
pH	Potential Hydrogen
PNF	Proprioceptive Neuromuscular Facilitation
SC	Sternoclavicular
SCT	Scapular Control Test
SDT	Scapular Dyskinesis Test
Sec	Seconds
sEMG	Surface Electromyography
SENIAM	Surface EMG for a Non-invasive Assessment of Muscles
SF-36	Short Form-36
SP	Substance P
ST	Scapulothoracic
TENS	Transcutaneous Electrical Nerve Stimulation
TNF- α	Tumor Necrosis Factor Alpha
TrPs	Myofascial Trigger Points
TRPV	Transient Receptor Potential Vanilloid Receptor
T-tubules	Transverse Tubules

Chapter 1: Introduction

1 Introduction

Muscle pain and tenderness is a common phenomenon recognised by the International Association for the Study of Pain. While this phenomenon of muscle pain and tenderness, in the absence of obvious disease, is well recognised, it is poorly understood (Quintner et al., 2015). Many theories on muscle pain exist but myofascial pain syndrome (MPS) is the most widely accepted model for this phenomena. At the centre of MPS lies the myofascial trigger point (TrP). TrPs are described as hyperirritable points located within a taut band of skeletal muscle or fascia, which cause referred pain, local tenderness and autonomic changes when compressed. TrPs are said to exist in many classifications but there are two basic types that are most frequently made reference to: active trigger points (ATrPs) and latent trigger points (LTrPs).

ATrPs produce spontaneous pain, tenderness in a taut band of muscle fibres, familiar pain to the patient, a local twitch response when stimulated manually or with a needle, and referred pain away from the stimulated TrP (Simons et al., 1999). In contrast, LTrPs are minor, subclinical neuromuscular lesions, which do not cause pain until stimulated manually or with a needle (Simons et al., 1999). Both ATrPs and LTrPs cause allodynia (pain due to a stimulus which does not normally cause pain) at the TrP site and hyperalgesia (abnormally increased sensitivity to pain) away from the TrP following applied pressure or needle insertion. In clinical practice, a TrP is considered active if the elicited pain is familiar to the patient.

While there has always been a general consensus that LTrPs precede ATrPs, research has only recently begun to focus on LTrPs in more detail and they have been shown to be significantly more complex in nature (Celik and Mutlu, 2013). They are now thought to possess clinical implications prior to their potential conversion to ATrPs, despite the absence of pain (Celik and Mutlu, 2013). These clinical implications include decreased range of motion, muscle weakness, and altered muscle activation patterns (MAPs) (Celik and Mutlu, 2013). While a healthy, pain-free person with LTrPs may not be conscious of any of these potential deficiencies, all are known injury risk factors (Clarsen et al., 2014). Thus, they are of interest to researchers and practitioners who strive to reduce injury risk factors in athletic populations. Based on the findings of this recent research, it is speculated that treating LTrPs in pain-free individuals may resolve the proposed

deficiencies, thereby improving the effected muscle's function and preventing TrPs from transforming into their painful active state. However, despite the extensive research that has been conducted on TrPs, their most limiting factor has been the lack of conclusive evidence for their underlying pathophysiology, and this has hindered their acceptance into mainstream medicine (Shah et al., 2015). Recently the hypothesis of TrPs as the source of muscle pain has been challenged, due to the lack of scientific evidence to support the empirical evidence (Quintner et al., 2015).

Despite the lack of a scientific basis, with the research that has been carried out over recent decades, there are a number of proposed treatment options for TrPs. Such treatments generally follow the principle of inactivation of the TrP to reduce pain, and correction of the factors that precipitated and perpetuated the formation of the TrP (Gerwin, 2010). Treatments can be essentially divided into invasive and non-invasive techniques (Huguenin, 2004). Such treatment options have included, TrP pressure release (manual release), spray (vapocoolant) and stretch, muscle energy techniques, transverse friction massage, thermotherapies, transcutaneous electrical nerve stimulation (TENS), laser therapy, ultrasound, exercise, dry needling (needling without an injectate), and wet needling (needling with an injectate) (de las Peñas et al., 2005).

Of the variety of treatment options that exist for TrPs, manual release and dry needling are the most commonly used (Cagnie et al., 2015). Manual release is performed by applying tolerably painful, persistent manual pressure, usually with the thumb or fingertip, against the tissue barrier of the TrP and is sustained until the tenderness experienced by the patient has subsided (Simons et al., 1999). A systematic review of manual therapies on TrPs concluded that there are only a few randomised controlled trials that analyse treatment of MPS using manual therapies and that further studies are needed (de las Peñas et al., 2005).

Although various dry needling approaches exist, the most common and best supported approach targets TrPs (Dommerholt, 2011). Dry needling is a procedure in which an acupuncture-like needle is inserted into the skin and muscle in the location of a TrP (Dommerholt et al., 2006b). Needles are removed once the TrP is deactivated and is typically followed by stretching exercises (Furlan et al., 2005). The mechanism by which dry needling acts to deactivate the TrP is undetermined (Cummings and Baldry, 2007).

A systematic review and meta-analysis on the effectiveness of dry needling for upper-quadrant myofascial pain recommended dry needling, compared to sham or placebo, for decreasing pain immediately after treatment and at four weeks in patients with MPS in the upper limb (Kietrys et al., 2013). However in a more recent review of the literature by Dunning et al. (2014), it was concluded that there is a paucity of high-quality evidence to underpin the use of TrP dry needling for the purpose of short and long-term pain and disability reduction in patients with musculoskeletal pain disorders.

As uncertainty continues to surround the proposed hypotheses for TrPs it is of paramount importance that the efficacy of their treatments are scrutinised, especially due to the lack of consistency observed in treatment studies. The pain-free nature of the LTrP offers the opportunity to investigate some specific clinical implications of TrPs, without pain potentially confounding results. One of the proposed clinical implications of LTrPs is the alteration of MAPs (Celik and Mutlu, 2013), and it has also been associated with an increased risk of shoulder pathology (Wadsworth and Bullock-Saxton, 1997).

Shoulder pathologies can be extremely debilitating both for the general population and athletes. The overall function of the shoulder complex is to allow for placement, function, and control of the hand in space, thus any pain associated with shoulder pathologies can potentially hinder simple everyday tasks (Roach et al., 1991). The scapula plays a key role in the function of the shoulder complex, acting as the stable base of support for the origin of many key muscles (Kibler et al., 2013). Scapular stability is thought to be needed for the force production of these muscles and a lack of it has been termed scapular dyskinesis, which is defined as an alteration of normal scapular kinematics (Kibler et al., 2013). In recent years the concept of scapular dyskinesis has gained a lot of attention as a potential cause of injury and thus a number of clinically based visual assessment tests, which aim to objectify the quality of the scapula's motion, have been developed (Ellenbecker et al., 2012; Kibler et al., 2002; McClure et al., 2009; O'Connor et al., 2015; Uhl et al., 2009). However, due to the complex movement of the scapula and the overlying soft tissues which obstruct a clear view, one of the limitations of these tests has been their inter- and intra-tester reliability.

In addition, it is unclear if scapular dyskinesis is a cause of shoulder pathology or if it is a result of it (Lucas et al., 2004). One of the many causes of scapular dyskinesis is thought

to be altered periscapular (around the scapula) MAPs (Kibler et al., 2013). However, research is equivocal with regard to whether the presence of pain causes altered MAPs or if altered MAPs occur first and cause biomechanical changes which led to shoulder pathologies (Lucas et al., 2004). If the recent research on LTrPs is considered, and specifically altered MAPs as one of their proposed implications, LTrPs may be an unidentified abnormality that potentially causes both scapular dyskinesia and shoulder pathologies.

Recent research on LTrPs and the list of their proposed clinical implications offers an opportunity to examine the efficacy of TrP treatment options without the presence of pain. The implication of altered MAPs is one of particular interest due to its involvement in scapular dyskinesia and shoulder pathologies. While one previous study has investigated how LTrPs cause altered MAPs and the effects of dry needling (Lucas et al., 2004), it did not examine dry needling in isolation, combining passive stretching in the intervention. Thus, research is needed to examine the efficacy of LTrP treatment options in isolation. The use of MAPs as an outcome measure, in particular within the shoulder complex is one that would be of benefit not only to the research community but also clinicians.

1.1 Aim and Objectives of the Research

Aim

The primary aim of this research is to establish the efficacy of TrP dry needling and manual release in treating LTrPs.

Objectives

1. To determine which of two scapular dyskinesia assessment tests are the most reliable and valid tests to assess scapular dyskinesia.
2. To determine whether any potential alterations in MAPs can be visually observed during a scapular dyskinesia assessment test following TrP dry needling, manual release, or placebo dry needling treatment interventions.
3. To determine whether any potential alterations in MAPs are observed following sEMG analysis of muscles that control shoulder kinematics during a standardised movement.

4. To determine whether any visual alterations observed during a scapular dyskinesis assessment test corresponded to alterations in sEMG.

1.2 Thesis Overview

Following the brief introduction to the research topic and an outline of the primary aim and objectives in the current chapter, a review of the relevant literature is subsequently presented in Chapter two. The first section of the literature review (section 2.1) focuses on the main topic of this thesis, TrPs and is further subdivided into five subsections. These subsections focus on the current knowledge of TrPs, their clinical characteristics, the underpinning pathophysiology, how they are identified and the reliability of these techniques, and the treatment options available. It also presents the most recent description of the evolving integrated TrP hypothesis and discusses the nature of its active and latent forms. This review also examines the opposing viewpoints on TrPs and the controversy that surrounds TrPs, MPS and their treatments. This section of the review discusses the failings of the TrPs hypothesis as the source of MPS and the alternative theories.

As this research has focused on how treating LTrPs with dry needling effects the MAPs of the shoulder complex, section 2.2 of the literature review details the anatomy, movements and control of the shoulder complex. This section first briefly details the anatomy of the shoulder complex followed by a review of the current understanding of the kinematics of the individual joints involved in movement of the upper limb and how to assess it. This section then discusses how the complex interaction of these joints are controlled by the timing of specific MAPs of the shoulder girdle muscles.

Chapter three describes a pilot study that was carried out to evaluate the reliability and validity of two scapular dyskinesis tests, in the assessment for scapular movement abnormalities. Chapter four then details the main study carried out to examine the effectiveness of dry needling to treat LTrPs compared to manual release and a placebo dry needling treatment. Chapter five details an overall conclusion of the research conducted and recommendations for future research.

Chapter 2: Literature Review

2 Literature Review

A review of the literature was conducted on two areas that were pertinent to this research, TrPs and the shoulder complex. The following sections have reviewed key aspects of the research in both areas to provide an understanding of what is currently known and how this research aims to add to it.

2.1 Myofascial Trigger Points

This first section focuses on TrPs and has been divided into five subdivisions. Firstly the scope of the problem and why LTrPs are a topic of interest are discussed. Secondly, the classifications of TrPs and specifically the differences between the active and latent forms are detailed. Thirdly, the literature is presented on how TrPs are identified clinically, and followed by the research behind the hypotheses for TrPs. Finally the efficacy of the various treatment options that are used in the treatment and management of TrPs are discussed.

2.1.1 The Scope of the Problem

MPS is a clinical problem that has generated interest and debate among clinicians for decades (Shah et al., 2015). While TrPs have been reported to be central to MPS, they may not necessarily be present in those with MPS (Simons et al., 1999). Though no large epidemiological studies reporting the prevalence of TrPs have been published, anecdotal evidence from experienced practitioners implies that pain caused by TrPs is a very common phenomenon (Huguenin, 2004; McCain, 1994; Simons et al., 1999), particularly after trauma or sustained muscular contractions.

In 2002, it was estimated that 10% of the population of the USA had one or multiple chronic musculoskeletal problems (Alvarez et al., 2002). A study performed in the Netherlands, suggested that the impact of unexplained musculoskeletal pain syndromes, on perceived general wellbeing, was a significant issue for patients and physicians producing considerable economic consequences (Boonen et al., 2005). In a study conducted by Skootsky et al. (1989), MPS was diagnosed in 21% of the patients in a general orthopaedic clinic and in 30% of patients in an internal medicine group practice, in a study conducted at the University of California, Los Angeles, Medical Ambulatory Care Center. In addition, MPS is said to be the leading cause of job-related

disability and the second leading cause of disability in the US, costing Americans more than \$50 billion each year (Martin et al., 2008).

2.1.2 Myofascial Trigger Points: Classifications and Definitions

TrPs are complex entities and the current lack of understanding and conflicting hypotheses of the underlying mechanisms provides a strong case for their investigation. TrPs are considered the hallmark characteristics of MPS and are proposed to feature motor, sensory, and autonomic components (Dommerholt et al., 2006a). 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 et al., 1999), as opposed to healthy muscle, which does not contain taut bands or TrPs (Shah et al., 2005). An individual contraction knot appears as a segment of a muscle fibre with severely contracted sarcomeres and an increased diameter (Figure 2.1).

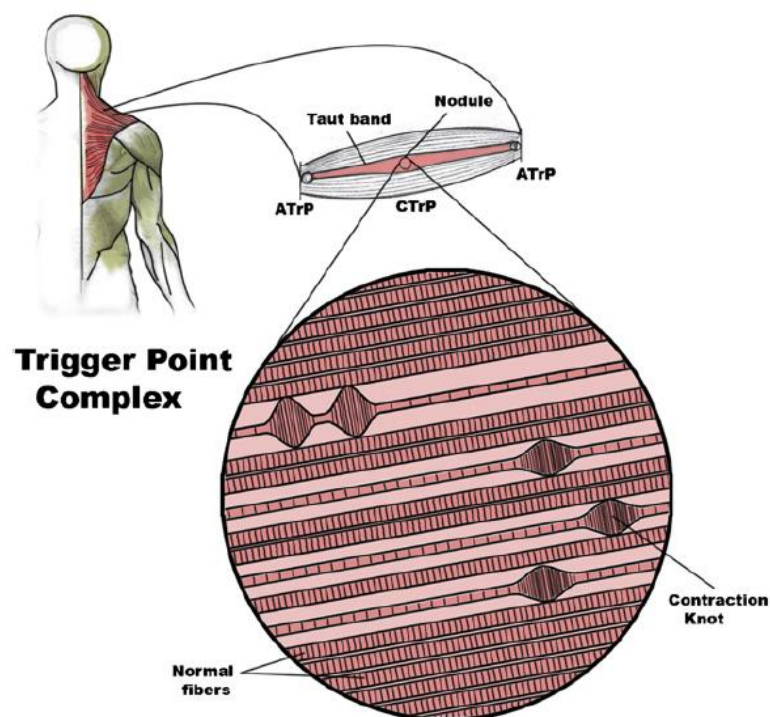


Figure 2.1 Schematic representation of a trigger point complex [reproduced from Shah & Gilliams (2008)]

TrPs are classified into active and latent forms. ATrPs cause spontaneous pain and are defined as those that cause a clinical pain complaint (Simons et al., 1999). In their most active state, ATrPs cause pain at rest and in a less severe state, pain with activity (Simons et al., 1999). ATrPs are always tender and have a number of unwanted effects on muscle,

such as, reduced tolerance of stretch and muscle weakness (Simons et al., 1999). In contrast, LTrPs are not spontaneously painful with use or at rest and are recognised by a taut band in the muscle, that does not reproduce pain until manually stimulated (i.e. during palpation) (Simons et al., 1999). Thus, the TrP is thought to be dynamic, changing in its degree of irritability or activity, and raises the question of what the minimum changes are that occur in a muscle when it is injured or stressed to form a TrP. The clinically evident progression from a non-tender taut band to a tender taut band suggests that the first change in muscle is the development of the contracted, taut group of muscle fibres that can become painful when sufficiently stressed (Simons et al., 1999). Table 2.1 shows the classifications of TrPs and their definitions as per the Travell and Simons' Trigger Point Manuals (Simons et al., 1999).

Table 2.1 Classifications & definitions of TrPs [modified from Simons et al. (1999)]

<p>Myofascial Trigger Point (TrP): A hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. The spot is painful on compression and can give rise to characteristic referred pain, referred tenderness, motor dysfunction, and autonomic phenomena.</p>
<p>Active Myofascial Trigger Point (ATrP): A myofascial trigger point that causes a clinical pain complaint. It is always tender, prevents full lengthening of the muscle, weakens the muscle, refers a patient-recognised pain on direct compression, mediates a local twitch response of muscle fibres when adequately stimulated, and, when compressed within the patient's pain tolerance, produces referred motor phenomena and often autonomic phenomena, generally in its pain reference zone, and causes tenderness in the pain reference zone.</p>
<p>Latent Myofascial Trigger Points (LTrPs): A myofascial trigger point that is clinically inactive with respect to spontaneous pain; it is painful only when palpated. A latent trigger point may have all the other clinical characteristics of an active trigger point and always has a taut band that increases muscle tension and restricts range of motion.</p>

2.1.3 Identifying Myofascial Trigger Points

A diagnosis of MPS relies upon the identification of ATrPs in specific muscles where their presence is known to account for a patient's particular symptoms. Due to the fact that 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.

2.1.3.1 Patient Subjective History

ATrP pain is typically described as a relatively 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, sharp, electricity like sensations (Baldry, 2005). According to Simons (2004), 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 working at a desk).
3. Repetitive activity, with pain increasing with increased exposure to the repetitive activity (e.g. using a screwdriver).

Patients may be aware of specific movements that are restricted due to the pain elicited by activating the muscle containing the TrP (Simons, 2004) but can often move through a large proportion of the full range of movement, 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. Patients may also report a loss of strength in affected muscles, in the absence of obvious atrophy. However, while the patient can perform tasks requiring strength, the effort needed is perceived as greater than before the onset of TrP symptoms (Simons, 2004). Furthermore the quality or coordination of movement may look or feel abnormal (Simons, 2004; Simons et al., 1999). Whether the decrease in strength and poor coordination experienced by patients is due to the ATrP or simply by the presence of pain has not been established.

Finally, Baldry (2005) noted 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 (arrector muscles of hairs) changes such as goose bumps, sweating, persistent lacrimation (secretion of tears) 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 should be

initiated to attempt to identify the clinical signs of TrPs as discussed in the following paragraphs.

2.1.3.2 Physical Examination

Baldry (2005) suggested that, locating TrPs through palpation is the most important part of the clinical examination, but also advocated the use of physical tests to identify, or confirm a patient's reported limited, painful, or uncoordinated movement. According to Simons et al. (1999) the best guide to the precise location of 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 fibres are still slack but the fibres of any taut bands are placed under additional tension, rendering them more easily distinguishable (Simons et al., 1999). Once the muscle being examined is positioned, "snapping palpation" (a cross-fibre plucking motion similar to plucking a guitar string) has been proposed to differentiate any taut bands from adjacent normal muscle fibres (Simons et al., 1999). It is important to note that the presence of a taut band of skeletal muscle is not considered in itself diagnostic of the presence of an ATrP and therefore to MPS, due to the fact that taut bands and LTrPs have been identified in subjects with no complaint of pain (Gerwin et al., 1997; Njoo and Van der Does, 1994; Wolfe et al., 1992).

Once a palpable taut band of skeletal muscle has been located, the identification of a tender nodule within it should be assessed, 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, 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, the TrP is said to be latent (LTrP) (Simons et al., 1999). In contrast, 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 et al., 1997). The presence of referred pain and the extent of the referred pain pattern, whether it is the partial or complete referred pain pattern associated with a particular TrP, has been considered by Simons et al. (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, a sudden twitch contraction that occurs either in the fibres of the taut band containing the TrP, a different taut band in the same muscle, or in a taut band in another muscle (Simons, 2004). The local twitch response can be elicited by either strong compression of, or needle insertion into, the suspected TrP (Chen et al., 2001) and is considered the most objective sign that a TrP has been identified or effectively treated (Gerwin et al., 1997; Hong, 1994). The local twitch response is thought to be a spinal cord reflex and has been recorded using electromyography (EMG), and palpated or observed by many authors (Audette et al., 2004; Baldry, 2002; Cummings and White, 2001; Gerwin et al., 1997; Hong, 1994).

In summary, current research considers a TrP 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 local twitch response in response to stimulation of the TrP with snapping palpation or needle insertion.

2.1.4 Aetiology, Pathogenesis and Hypothesis of Myofascial Trigger Points

Perhaps one of the most limiting aspects of TrPs that has challenged their acceptance into mainstream medicine has been the uncertainty surrounding their aetiology and pathophysiology (Shah et al., 2015). Numerous clinicians have encountered and described tender nodules in muscle and have attempted to explain their aetiology, tissue properties, and relationships to muscle related pain (Shah et al., 2015). A review by Dommerholt et al. (2006a) noted that as far back as the 16th century, de Baillou (1538-1616) described a condition very similar to MPS, as cited by Ruhmann (1940). Over the centuries TrP like lesions have been described countless times by innumerable investigators who have identified them by a multitude of names (Simons, 2004). However, despite various investigators identifying TrP like lesions, the aetiology and pathophysiology of TrPs and MPS are still not fully understood.

TrPs have two clinical attributes that still require further explanation to assist the understanding. The first is present in both ATrPs and LTrPs, and is described as a motor dysfunction of the muscle that is characterised by a constant, discrete hardness within

the muscle, the taut band (Gerwin et al., 2004). The other is described as a sensory abnormality that is characterised primarily by pain (Gerwin et al., 2004). To understand the proposed motor and sensory abnormalities associated with the development of TrPs the normal processes involved in muscle contraction will be briefly described.

2.1.4.1 Normal Skeletal Muscle Contraction

The contraction of skeletal muscle is triggered by nerve impulses or action potentials that travel from the brain or spinal cord (Katch et al., 2011). An action potential propagates down a motor neuron to the skeletal muscle fibre. The site where a motor neuron excites a skeletal muscle fibre is called the neuromuscular junction. The events at the neuromuscular junction occur in series of coordinated steps (Katch et al., 2011). Firstly, an action potential travels the length of the axon of a motor neuron to an axon terminal. This results in voltage gated calcium channels opening and calcium ions (Ca^{2+}) then diffuse into the terminal causing synaptic vesicles to release acetylcholine (ACh), which diffuses across the synaptic cleft and binds to acetylcholine receptors (AChR). AChR contain ligand-gated cation channels which open and allow sodium ions to enter the muscle fibre and potassium ions to exit the muscle fibre. The greater influx of sodium ions relative to the out flux of potassium ions causes the membrane potential to become less negative. Once the membrane potential reaches a threshold value an action potential propagates along the sarcolemma. Neural transmission to a muscle fibres ceases when ACh is removed from the synaptic cleft, through either diffusion away from the synapse or it is broken down by the enzyme acetylcholinesterase (AChE) to acetic acid and choline (Katch et al., 2011). Choline is then transported into the axon terminal for the resyntheses of ACh (Katch et al., 2011).

Typically a single motor neuron arising in the brain or spinal cord conducts action potentials that travel to hundreds of skeletal muscle fibres within a muscle. The sequence of events that converts action potentials in a muscle fibre to a contraction is known as excitation-contraction coupling (Katch et al., 2011). Action potentials travel across the entire sarcolemma of muscle fibres and are rapidly conducted into the interior of the muscle fibre by structures called transverse tubules (T-tubules) (Katch et al., 2011). At numerous junctions the T-tubules make contact with a calcium storing membranous network known as the sarcoplasmic reticulum (Katch et al., 2011). Where it touches the T-tubules, the sarcoplasmic reticulum forms sack like bulges called

terminal cisternae. The membrane of the T-tubules and the terminal cisternae are linked by a series of proteins that control calcium release (Katch et al., 2011). As an action potential travels down the T-tubule it causes a voltage-sensitive protein to change shape. This shape change opens a calcium release channel in the sarcoplasmic reticulum, allowing Ca^{2+} to flood the sarcoplasm. This rapid influx of Ca^{2+} triggers a contraction of the skeletal muscle fibre (Katch et al., 2011), in a process which is detailed below.

Contraction of skeletal muscle is triggered by a series of molecular events known as the cross bridge cycle. In a skeletal muscle fibre sarcomeres are the basic contractile unit. A sarcomere shortens when myosin heads and thick myofilaments form cross bridges with actin molecules and thin myofilaments. The formation of a cross bridge is initiated when Ca^{2+} released from the sarcoplasmic reticulum binds to troponin causing troponin to change shape. Tropomyosin then moves away from the myosin binding site on actin molecules, allowing the myosin head to bind to actin and form a cross bridge. However, before this cross bridge can occur myosin heads must be activated. This occurs when an adenosine triphosphate (ATP) molecule binds to the myosin head and is hydrolysed to adenosine diphosphate (ADP) and inorganic phosphate. The energy liberated from the hydrolysis of the ATP molecule activates the myosin head forcing it into the cocked position. The cross bridge cycle consists of four steps (Katch et al., 2011) which are depicted in Figure 2.2. Firstly, cross bridge formation occurs when the activated myosin head binds to actin forming a cross bridge. Once the cross bridge is formed inorganic phosphate is released from the myosin head and the bond between actin and myosin becomes stronger. Next the power stroke occurs, when ADP is released and the activated myosin head pivots, sliding the thin myofilament toward the centre of the sarcomere. Thirdly, the cross bridge detaches, when another ATP molecule attaches to the myosin head the link between actin and myosin weakens and the myosin head detaches. Finally the myosin head is reactivated, when ATP is hydrolysed into ADP and inorganic phosphate. The energy release from the hydrolysis reactivates the myosin head returning it to the cocked position.

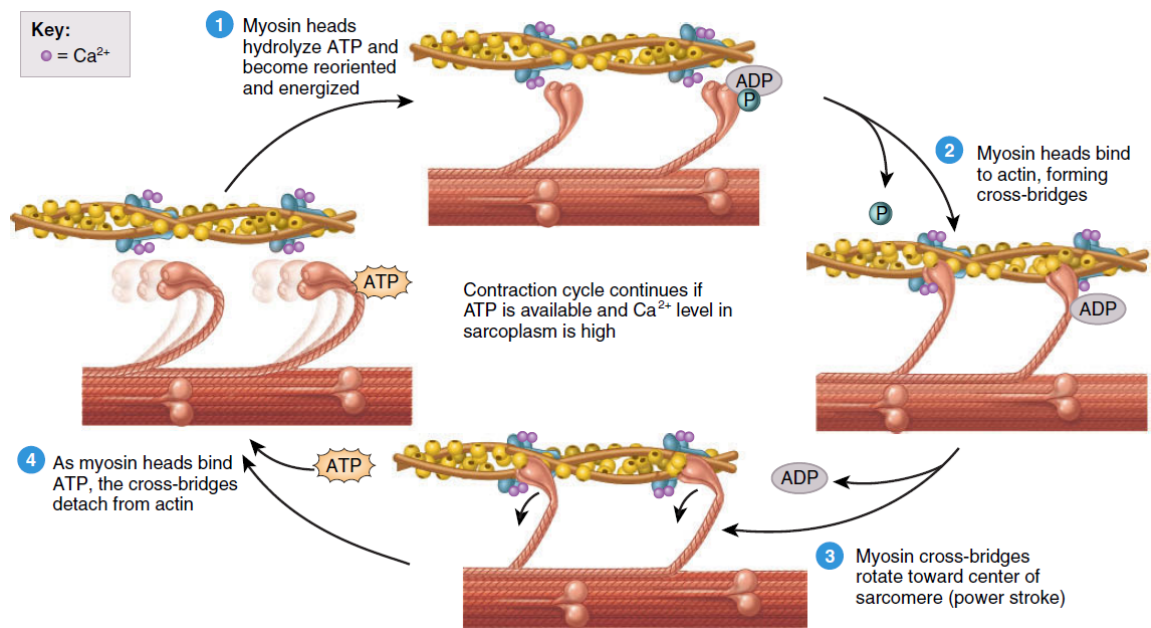


Figure 2.2 The cross bridge cycle [reproduced from Tortora and Derrickson (2014)]

As long as the binding sites on actin remain exposed and ATP is available the cross bridge cycle will repeat. As the cycle repeats the thin myofilaments are pulled toward each other and the sarcomere shortens. Cross bridge cycling ends when Ca^{2+} are actively transported back into the sarcoplasmic reticulum. Removal of Ca^{2+} returns troponin to its original shape, allowing tropomyosin to glide over and cover the myosin binding site on actin.

2.1.4.2 Aetiology and Pathogenesis of Myofascial Trigger Points

As previously discussed, there is general agreement that any kind of muscle overuse or direct trauma to the muscle can lead to the development of TrPs. Overuse is thought to occur from sustained or repetitive low-level contractions, eccentric muscle contractions, and maximal or submaximal concentric muscle contractions (Gerwin, 2010). Though muscle damage is not thought to be required for the development of TrPs, overuse mechanisms may cause disruption of the cell membrane. This can cause damage to the sarcoplasmic reticulum with a subsequent release of high amounts of Ca^{2+} , and disruption of cytoskeletal proteins, such as desmin, titin, and dystrophin (Bron and Dommerholt, 2012).

The role of sustained low-level contraction in the formation of TrPs is thought to be due to a reduction in available oxygen and energy source (Bron and Dommerholt, 2012). During muscle contractions there is a temporary obstruction to capillary blood flow

which is restored immediately with relaxation of the muscle. During dynamic rhythmic contractions, intramuscular blood flow is enhanced by this contraction-relaxation rhythm, also known as the muscular pump (Bron and Dommerholt, 2012). However, during sustained muscular contraction, muscle metabolism is highly dependent upon oxygen and glucose, which are in short supply due to the restricted blood flow (Bron and Dommerholt, 2012). The percentage of maximal voluntary contraction (MVC) of muscles necessary to induce sufficient intramuscular blood pressure varies depending on the architecture of the muscle (Bron and Dommerholt, 2012). Even contractions at 10% and 25% of MVC may produce intramuscular pressure high enough to significantly impair the intramuscular blood circulation (Bron and Dommerholt, 2012).

Since oxygen and glucose are required for the synthesis of ATP, which provides the energy needed for muscles contractions, sustained contractions may cause a local energy crisis due to the lack of oxygen. It is hypothesised that due to the lack of oxygen, the region of muscle switches to anaerobic glycolysis (Bron and Dommerholt, 2012), under which most of the pyruvic acid produced is converted into lactic acid, thus increasing the intramuscular acidity (pH) (Bron and Dommerholt, 2012). In normal circumstances most of the lactic acid diffuses out of the muscle into the bloodstream, however, during sustained low-level contraction, this process is thought to be impeded (Bron and Dommerholt, 2012).

Shah et al. (2005) and Gautam et al. (2010) reported that the environment of ATRPs may have a pH well below 5, which is sufficient to excite muscle nociceptors, including acid-sensing ion channels (e.g. ASIC 1 and 3), and the transient receptor potential vanilloid receptor TRPV1 (Gautam et al., 2010; Shah et al., 2005). Along with the decrease in pH, small increases of the hydrogen ion (H^+) concentration, as seen with inflammation, heavy muscle work, and ischemia, are sufficient to excite muscle group IV endings, contributing to mechanical hyperalgesia and central sensitisation (Dommerholt, 2011). Low pH levels will also downregulate AChE, increase the efficacy of ACh, and maintain the sarcomere contraction. The decreased pH also triggers the release of several nociceptive substances, such as calcitonin gene-related peptide (CGRP) (Dommerholt, 2011), which can enhance the release of ACh from the motor endplate and simultaneously decrease the effectiveness of AChE in the synaptic cleft (Bron and Dommerholt, 2012). CGRP also upregulates the AChR at the muscle and thereby creates

more docking stations for ACh (Bron and Dommerholt, 2012). Each of the above processes helps maintain the sarcomere contraction and creating hyperalgesia at the site.

Relaxation within muscle cells only occurs when the actin and myosin cross-bridges detach, which requires the presence of ATP to weaken the link between myosin and actin (Katch et al., 2011). Simultaneously, Ca^{2+} detach from the troponin molecule, allowing it to return to its original shape, which in turn allows tropomyosin cover the actin-myosin binding site, thus preventing further cross-bridging (Katch et al., 2011). Under these normal conditions large amounts of free Ca^{2+} will re-enter the sarcoplasmic reticulum by the Ca^{2+} pump, adenosine triphosphatase (ATPase), which places a high demand on ATP during relaxation (Katch et al., 2011). In cases of sustained low-level contraction, when severe energy depletion may occur, the sarcomere may stay contracted, until enough ATP is available to resolve the intracellular Ca^{2+} accumulation (Bron and Dommerholt, 2012). High levels of intracellular Ca^{2+} are associated with sustained sarcomere contraction and muscle damage, and this high Ca^{2+} concentration has been suggested to play a causative role in the development of muscle disorders and TrPs.

Sustained low-level contractions are common in the work place where prolonged postures are required. Such tasks typically require the use of small motor units which innervate type I, red coloured, slow oxidative, muscle fibres. Hägg (2000) suggested that the continuous activity of these motor units in sustained contractions causes overuse and muscle fibre damage, especially to type I fibres during low-level activities. It was because of the overuse of these muscles that Hägg coined the Cinderella hypothesis, which postulates that damage to muscles can occur when the type I muscle fibres, activated first and last to deactivate, are required to work the longest and may not obtain adequate amounts of time to recover (Hägg, 2000).

Maximal or submaximal contractions, along with eccentric contractions, have also been proposed as potential causes for the development of TrPs (Bron and Dommerholt, 2012). The development of TrPs during maximal or submaximal contraction is said to occur when the demands of exercise begin to exceed the ability of the muscle cells to carry out the necessary reactions quickly enough (Bron and Dommerholt, 2012). If the

demands on the muscle are maintained the muscle is said to run out of ATP and sustained sarcomere contractions may occur and thus the development of TrPs (Bron and Dommerholt, 2012). In relation to the development of TrPs from eccentric contractions, the mechanism is thought to be due to disruption of the cytoskeletal and myofibril structures (Bron and Dommerholt, 2012). The damage to these structures is said to increase the concentration of Ca^{2+} , probably due to the disruption of the sarcoplasmic reticulum. As mentioned previously high concentrations of Ca^{2+} keep myosin binding sites on actin exposed.

2.1.4.3 The Integrated Trigger Point Hypothesis

There are many hypotheses on the formation of TrPs, namely those by Hägg (2000) and Hocking (2010), but the most prominent and most accepted model is the integrated TrP hypothesis detailed by Simons et al. (1999), and later expanded by Gerwin et al. (2004). The integrated TrP hypothesis combines information from electrophysiological and histopathological sources as described above. It has evolved over the years, but its origins were based on an energy crisis concept that did not incorporate an electrophysiological mechanism (Figure 2.3). This energy crisis concept postulated an increase in the concentration of Ca^{2+} outside of the sarcoplasmic reticulum, possibly due to mechanical rupture of either the sarcoplasmic reticulum itself or the sarcolemma (Simons et al., 1999). A sufficient increase in Ca^{2+} would maximally activate actin and myosin contractile activity. However, this energy crisis concept could only continue if the damaged sarcoplasmic reticulum or sarcolemma was not repaired, and as the repair processes could be expected to rapidly respond to such a phenomenon an alternative theory was needed. The energy crisis concept was updated to incorporate an electrophysiological mechanism as the integrated TrP hypothesis (Figure 2.4) (Simons et al., 1999).

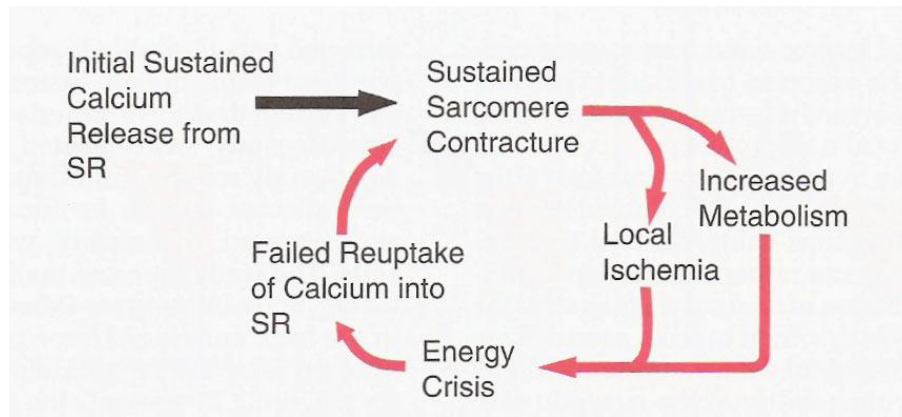


Figure 2.3 Energy crisis concept [reproduced from Simons et al. (1999)]

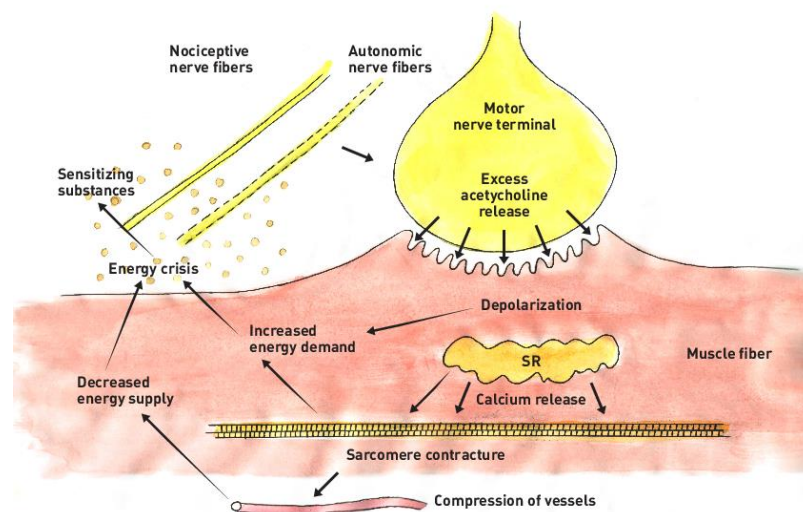


Figure 2.4 The Integrated Trigger Point Hypothesis [reproduced from www.dgs.eu.com]

It is now thought to be more likely that the sustained contractile activity is due to abnormal depolarisation of the post junction membrane that could continue indefinitely based on continuing excessive ACh release (Simons et al., 1999). Hubbard & Berkoff (1993) reported that there was a marked increase in the frequency of low-voltage (50-100 microvolts) electrical activity found at the point of maximal tenderness taut bands in humans. The site of this increased electrical activity has been localised to the neuromuscular junction endplate zone of the taut band, where it appears as an abnormally increased frequency of miniature endplate potentials (MEPP), in rabbit models (Hong and Yu, 1998) and in humans (Simons et al., 2002). The sustained contractile activity of the sarcomeres, due to the excessive ACh, cause markedly increased metabolic demand and squeeze shut capillaries that supply the nutritional and

oxygen needs of the region. The combination of these two events, increased Ca^{2+} release and abnormal depolarisation of the post junction membrane, are thought to produce a severe but local energy crisis (Simons et al., 1999).

The failure to reuptake Ca^{2+} to the sarcoplasmic reticulum is a key component in the continuation of the energy crisis. The Ca^{2+} pump that is responsible for this reuptake is dependent on an adequate supply of ATP. However due to the local ischemia caused by the sustained contracture sufficient ATP is not available for the Ca^{2+} pump to function normally, exposing the contractile elements to a further increase in calcium concentration and contractile activity, completing the vicious cycle (Simons et al., 1999). The severe local hypoxia and tissue energy crisis is also expected to stimulate the production of vasoreactive substances that could sensitise local nociceptors (Simons et al., 1999). This updated hypothesis is said to account for a number of key features relating to TrPs (Simons et al., 1999). Firstly, it accounts for the lack of a motor unit action potential, where the contraction of muscle fibres is due to the endogenous contracture of the contractile elements rather than a nerve-initiated contraction (Simons et al., 1999). Secondly, it is said to account for the frequency with which muscle overload activates TrPs (Simons et al., 1999). Thirdly, the updated hypothesis states that the release of substances, as a result of tissue distress caused by the energy crisis, could sensitise nociceptors in the region of the dysfunctional endplate of the TrP (Simons et al., 1999). Finally, it accounts for the effectiveness of essentially any technique that elongates the TrP portion of the muscle to its full stretch length, which is said to break the cycle that includes the energy-consuming contractile activity (Simons et al., 1999).

2.1.4.4 An Expansion of the Integrated Trigger Point Hypothesis

Gerwin et al. (2004) expanded on this hypothesis concluding that it is the alteration in the normal equilibrium between ACh, AChR, and AChE and the involvement of CGRP due to muscle injury which causes the formation of TrPs. The processes described below are illustrated in Figure 2.5.

Gerwin et al. (2004) hypothesise that the activating event in the development of TrPs is the performance of unaccustomed eccentric exercise, eccentric exercise in an unconditioned muscle, or maximal or submaximal concentric exercise that lead to muscle fibre damage and segmental hypercontraction within the muscle fibre.

Hypoperfusion (decreased blood flow through an organ) caused by capillary constriction, as a result of muscle contraction, is said to add to the physical stress. Capillary constriction is also said to be increased by sympathetic nervous system adrenergic (nerve cells in which adrenaline, noradrenaline, or similar substances act as a neurotransmitter) activity (Gerwin et al., 2004). The resultant ischemia and hypoxia adds to the development of tissue injury and produces a local acidic pH with an excess of protons (Gerwin et al., 2004). The acidic pH levels result in inhibition of AChE activity, increased release of CGRP, and activation of ASIC on muscle nociceptors (Gerwin et al., 2004).

The presence of an acidic pH alone (in the absence of muscle damage) is sufficient to cause widespread changes in the pain matrix. However, the breakdown of muscle fibres results in the release of several pro-inflammatory mediators such as SP, CGRP, K⁺, 5-hydroxytryptamine (5-HT, serotonin), cytokines, and bradykinin (BK) that profoundly alter the activity of the motor endplate and activity/sensitivity of muscle nociceptors and wide dynamic-range neurons (Gerwin et al., 2004). Motor endplate activity is increased because of an apparent increase in ACh in the synaptic cleft. As mentioned, this increase is caused by several factors: increased CGRP, presynaptic motor terminal adrenergic receptor activity, AChE inhibition caused by CGRP, and the up-regulation of AChRs through the action of CGRP which creates more docking sites for ACh (Gerwin et al., 2004). MEPP frequency is increased as a result of greater ACh effect and in turn results in the development of the taut band. Release of pro-inflammatory mediators from injured muscle activates the muscle nociceptors, thereby causing tenderness and pain.

The presence of CGRP drives the system to become chronic, potentiating the motor endplate response and potentiating, with SP, activation of muscle nociceptors (Gerwin et al., 2004). The combination of acidic pH levels and pro-inflammatory mediators at the ATrP contribute to segmental spread of nociceptive input into the dorsal horn of the spinal cord and leads to the activation of multiple receptive fields (Gerwin et al., 2004). The continuous nociceptive input causes neuroplastic changes in the dorsal horn neurons, causing further activation of neighbouring and regional dorsal horn neurons that now have lower activation thresholds (Gerwin et al., 2004). This results in the

observed phenomena of hypersensitivity and allodynia, and referred pain that is characteristic of the ATrP.

The Gerwin et al. (2004) expansion of the integrated TrP hypothesis is based around the fact that there is normally an equilibrium between the release of ACh, the breakdown of ACh, and its removal from AChRs in the postsynaptic membrane by AChE. However, this is disrupted by muscle injury and the release of substances that activate muscle nociceptors causing pain. The muscle damage also facilitates ACh release, inhibition of ACh breakdown and removal from the AChR, and an up-regulation of AChRs, leading to the development of persistent muscle fibre contraction, as is characteristic of TrPs (Gerwin et al., 2004).

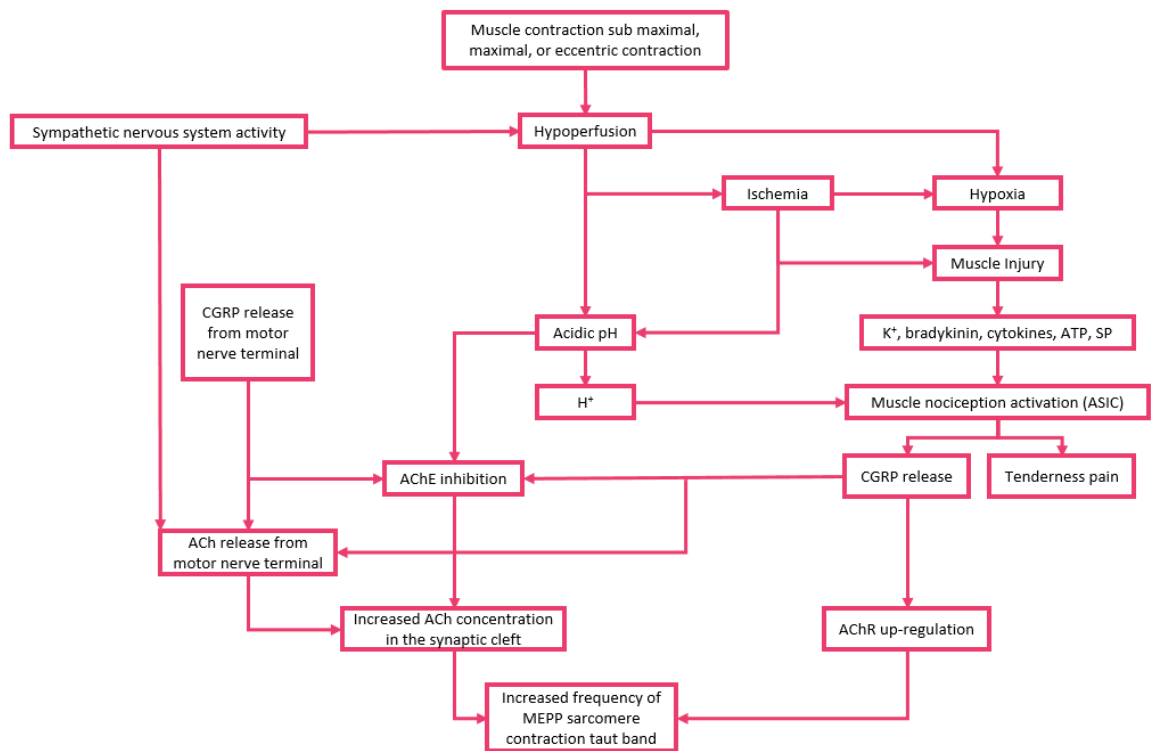


Figure 2.5 An Expansion of the Integrated Trigger Point Hypothesis [reproduced from Gerwin et al. (2004)]

2.1.4.5 Alternative Hypothesis

Hocking (2010) has postulated that eccentric loading does not provide a good model for the TrPs pathogenesis (Bron and Dommerholt, 2012). Hocking (2010) has suggested that sustained partial depolarisation or plateau depolarisation of an α -motor neuron due to an upregulation of voltage dependent calcium channels and $\alpha 1$ -adrenergic receptors. This along with a downregulation of calcium-activated potassium channels, would lead

to an increase in the motor terminal cytosolic Ca^{2+} concentration (Bron and Dommerholt, 2012). Hocking (2010) proposed that the increased Ca^{2+} concentration triggers the spontaneous release of ACh, thus suggesting this release in ACh would be the cause, not the result, of the energy crisis (Bron and Dommerholt, 2012). Therefore Hocking's theory is that centrally maintained α -motor neuron plateau depolarisation, rather than an intrinsic disorder of the motor endplate, is the fundamental pathophysiological mechanism which perpetuates the local muscle contracture associated with a TrP (Hocking, 2010).

2.1.4.6 The Trigger Point Debate

Due to the lack of evidence confirming the proposed TrP hypotheses there has been understandable debate around the topics of MPS, TrPs and their treatments. Quintner et al. (2014) questioned whether the development of the term TrP is based on sound science or rather of speculation and conjecture. In addition, Quintner et al. (2014) argued that the TrP theory and the associated concepts of MPS continue to be strongly held, despite the fact that it exemplifies circular reasoning, where TrPs are the cause of myofascial pain simply because painful muscles contain them (Quintner and Cohen, 1994).

In relation to clinical diagnosis, Quintner et al. (2014) stated that an extensive review, conducted by Tough et al. (2007), highlighted that at least 19 different sets of diagnostic criteria were used for the MPS/TrP syndrome. This review suggested that until reliable diagnostic criteria had been established there is a need for greater transparency in research papers on how a case of TrP pain syndrome is identified, and claims for effective interventions in treating the condition should be viewed with caution (Tough et al., 2007). A similar study, carried out by Lucas et al. (2009), found that the diagnosis of MPS from putative TrPs was based on a clinical test of unknown reliability and validity with no accepted reference standard (Lucas et al., 2009). Results from TrP reliability studies have shown varying outcomes, with early research demonstrating that physical examination could not be relied upon to diagnose the presence of TrPs (Hsieh et al., 2000; Lew et al., 1997; Wolfe et al., 1992). However, more recent studies have shown agreement that the phenomenon can be localised following sufficient training (Sciotti et al., 2001), assessments of an individual examiner are consistent from one test to another

(Al-Shenqiti and Oldham, 2005), and that more experience in assessment leads to better inter-tester agreement (Myburgh et al., 2011).

Shah et al. (2005 & 2008) reported that there is altered tissue biochemistry in tissue surrounding ATrPs compared to that of LTrPs and normal tissue. The studies state that elevated levels of CGRP, SP, norepinephrine, cytokines (tumour necrosis factor alpha (TNF- α), interleukin-1 and interleukin-6 (IL-1 and IL-6)), and low pH were found in all sampled regions of symptomatic patients, as well as uninvolved control muscle areas. These results from Shah et al. (2005 & 2008) are said to further supported the integrated TrP hypothesis (Simons, 2005). However, Quintner et al. (2014) stated that these reported alterations in biochemical milieu are consistent with inflammation due either to tissue damage or to altered peripheral nerve function, in contrast to pathology necessarily being in the tissue sampled (Chiu et al., 2012).

Studies that have completed EMG examination of TrPs have also reported equivocal results. An EMG study by Durette et al. (1991) failed to provide evidence of ongoing denervation or focal muscle spasm. Another study by Hubbard & Berkoff (1993) did however report spontaneous electrical activity in regions considered to be TrPs in patients with chronic tension headache and pericranial muscle tenderness. Simons (2001), one of the leading authorities on MPS and TrPs, addressed the question of whether endplate noise and spikes arise from normal endplates and in those diagnosed with fibromyalgia and LTrPs (Simons et al., 2002). However the authors included subjects with MPS associated with TrPs and the tender points of fibromyalgia. Simons et al. (2002) concluded that endplate noise is characteristic of, but not restricted to TrPs, and that the findings could not be considered a reliable diagnostic criteria for TrPs. Quintner et al. (2014) offered an alternative interpretation of these EMG finding, that spontaneous activity from single muscle fibres was generated by the activation of intramuscular nerve termini irritated by the needle being inserted.

Imaging studies that have used magnetic resonance elastography to identify a taut band (Chen et al., 2008, 2007) did not provide any diagnostic criteria or detail the relationship of a taut band to a TrP (Quintner et al., 2015). Attempts to visualise TrPs in abdominal muscles with the use of diagnostic ultrasound observed mixed echoic areas that became prominent on injection of local anaesthetic solution (Niraj et al., 2011). However, the

authors conceded that the finding could have been coincidental, as the image presented was consistent with the normal sonographic appearance of abdominal muscles (Gokhale, 2006).

Quintner et al. (2014) also took issue with the proposed integrated TrP hypothesis. They stated that although there is no experimental evidence in support of this hypothesis, others have accepted the motor endplate and the energy crisis theories of tonic muscle hyperactivity and TrP formation. Quintner et al. (2014) cites two studies that induced muscle pain in humans that have not provided evidence for a reflex increase in fusimotor drive and spindle discharge (Birznieks et al., 2008; Fazalbhoy et al., 2013). On the contrary, a study by Lund et al. (1991) found that persistent musculoskeletal pain was associated with decreased agonist muscle tone. Quintner et al. (2014) thus reported from these findings that digital pressure or other stimuli that evoke pain will decrease the tone of the muscle stimulated. Results from Lund et al. (1991) go against the integrated TrP hypothesis that correlates endplate activity or noise with pain arising from the TrP. Quintner et al. (2014) in their review, concluded that sufficient research has been performed to allow TrP theories to be discarded. They state that the scientific literature shows not only that diagnosis of the pathognomonic feature of MPS (the TrP) is unreliable, but also that treatment directed to the putative TrP elicits a response that is indistinguishable from the placebo effect.

It is vital that research continues to further the understanding of the pathogenesis of unexplained muscle pain. Whether the true source of the pathology is from TrPs is still currently unknown, in spite of decades of research carried out on these enigmatic lesions. While it is important to investigate TrPs as the potential cause of muscular pain, researchers and therapists should be open to new theories as pain is better understood.

2.1.5 Treatments

The increased research into TrPs in recent decades has resulted in a number of therapies being proposed for the treatment of TrPs. Commonly used methods are listed in Table 2.2 (Hong, 2004). The ongoing debate that surrounds TrPs gives further support for the need for additional research on TrP treatment interventions, to justify their use.

Table 2.2 Methods to treat TrPs [modified from Hong (2004)]

A. Manual Therapy Stretching – Intermittent Cold and Stretch Deep Pressure Soft Tissue Massage Trigger Point Pressure/Manual Release Ischemic Compression, Acupressure, Myotherapy, Shiatzu Chiropractic Therapy: Manipulation and Mobilisation Voluntary Contraction and Release Methods: Contract-Relax, Muscle Energy Technique, Reciprocal Inhibition, Post-Isometric Relaxation Others
B. Modality Therapy Thermotherapy Electro Therapy Laser Therapy Shockwave Therapy Others
C. Needling Traditional Acupuncture Dry Needling Myofascial trigger point injection
D. Others Therapeutic Exercise Medication Biofeedback
E. Combinations

It has been suggested that ice, heat, ultrasound and massage can be used in the treatment of TrPs (de las Peñas et al., 2005). These modalities are more focused towards ATrPs as they aim to achieve temporary relief of pain associated with ATrPs (de las Peñas et al., 2005). Treatments such as dry needling or injection with lidocaine or botulinum toxin type A, spray (with vapocoolant) and stretch, TENS and post-isometric relaxation have all been investigated for their effectiveness in resolving TrP pain (Graff-Radford et al., 1989; Hong, 1994; Jaeger and Reeves, 1986; Kamanli et al., 2005; Lewit and Simons, 1984). These studies typically measure changes in TrP pain with visual analogue scales and/or by the alterations in pressure pain thresholds by taking pre- and post-intervention measurements.

Although various treatment methods are in use and considered effective in the resolution of symptoms associated with TrPs, the mechanisms underlying the efficacy of these TrPs treatments are poorly understood. The current understanding of TrPs, as local muscle fibre contractures, has led the rationale of many treatment methods which aim to release the taut band (Hong, 2004). These therapies aim to release the

contracture therefore removing the peripheral mechanism of myofascial pain. Another mechanism by which therapies are proposed to treat TrPs is to modulate the central mechanism of myofascial pain. The mechanism by which these therapies are proposed to work is by causing a strong peripheral stimulus so that the sensitivity of the TrP is reduced, thus blocking any painful impulse into the higher centres of the brain (Hong, 2004).

In the subsequent review of various therapies every effort has been made to report specific statistical differences, however, this was not always achievable as a number of studies did not report them.

2.1.5.1 Manual Therapies

Manual therapy is an important category of TrP treatment and has been described in detail by Travell and Simons (Simons et al., 1999; Travell and Simons, 1992, 1983). One of the first manual therapy methods described by Travell and Simons was the “Spray and Stretch” technique (Travell and Simons, 1992, 1983). This technique involved the use of either vapocoolant or ice on the skin, which act as a surface anaesthesia. As TrPs are often painful to stretch, vapocoolant or ice is applied before or concurrently with stretching of the affected muscle. The rationale for this method is to cause alarming impulses to be sent to the spinal cord due to the sudden drop in skin temperature resulting in an inhibitory effect on locally generated pain (Simons et al., 1999). This analgesic effect is said to keep the muscle relaxed as a stretch is applied, when otherwise the stretching sensation may cause discomfort and initiate an involuntary protective contraction of the muscle (Simons et al., 1999). In a systematic review of manual therapies, de las Peñas et al. (2005) found that spray and stretch had a positive effect on visual analogue scale scores and pressure pain thresholds after treatment (Jaeger and Reeves, 1986), however, the review concluded that spray and stretch was not more effective than deep pressure soft tissue massage (Hong et al., 1993).

Another common method of TrP release is the utilisation of a number of different voluntary contraction and release methods (Hong, 2004; Simons et al., 1999). This treatment method has a number of different forms such as: post-isometric relaxation, reciprocal inhibition, contract relax, hold relax, and muscle energy techniques. These forms of voluntary contraction and release have slight variations as to how they are

carried out but all employ some degree of active contraction followed by relaxation (Simons et al., 1999). The principle behind this method is a reduction in muscle tension following the contraction which provides an increase in range of motion during the period of relaxation (Simons et al., 1999). The rationale of this approach relates to the contracture of the muscle fibre whereby gentle intermittent muscular contractions may be very effective at normalising sarcomere lengths of the involved muscle fibre (Simons et al., 1999). Simons et al. (1999) stated that the action potentials resulting from the voluntary effort cause contraction of the elongated sarcomeres on both sides of the contracture. In addition, the added tension that this creates tends to pull open the contracted sarcomeres in the contracted knot. This process is said to separate the actin and myosin molecules in the contracted sarcomeres, thus relieving the energy crisis (Simons et al., 1999). This treatment method tends to be used in combination with others, thus there is a paucity of studies that have investigated its effectiveness in isolation. Yeganeh Lari et al. (2016) investigated the effects of a muscle energy technique compared to dry needling and a combination of both treatments on LTrPs in the upper trapezius muscles of females. All groups showed improvements in the outcome measures, visual analogue scale, pressure pain thresholds, and range of motion ($p < 0.001$), with the combined treatment group showing more significant improvements compared to the other two groups in all outcome measures ($p < 0.05$) (Yeganeh Lari et al., 2016). However this study did not include a control group, which is a common limitation of studies in this area.

Perhaps one of the most frequently used treatments of TrPs is the use of manual pressure. When applying the TrP pressure release treatment, the clinician lengthens the involved muscle to the point of increasing resistance within the comfort of the patient and then applies gentle, gradually increasing pressure to the TrP until the finger encounters a definite increase in tissue resistance (Simons et al., 1999). At this point it is normal for the patient to feel a degree of discomfort, but pain should not be experienced. Pressure is maintained until the clinician senses relief of tension under the palpating finger (Simons et al., 1999). Then, pressure is increased enough to take up the tissue slack and to encounter a new barrier. This process can then be repeated on each taut band within the muscle.

2.1.5.2 Modality Therapies

Therapeutic ultrasound and low-level laser therapy have been widely used in clinical settings for the treatment of TrPs. They have achieved popularity and recognition among physicians and therapists as non-invasive treatment options (Beckerman et al., 1992; Draper et al., 2010). As with other treatment options, ultrasound and low-level laser therapy are often used in conjunction with other treatments and therefore there is a dearth of controlled trials investigating the effectiveness of ultrasound and low-level laser therapy as stand-alone treatments of TrPs.

Manca et al. (2014) recently completed a well-designed randomised, double-blind, placebo-controlled study that investigated the effectiveness of ultrasound and low-level laser therapy on sixty participants with at least one ATrP. Participants were randomised into one of five groups: active ultrasound, placebo ultrasound, active low-level laser therapy, placebo low-level laser therapy, or no therapy (control). Data was collected at baseline, post-intervention and 12 weeks follow-up on three outcome measures, pressure pain thresholds, pressure related pain assessed with a numerical rating scale (NRS) and active cervical lateral flexion measured by goniometry. After two weeks of intervention, all groups showed significant improvements in all outcome measures, with decreases in pressure pain thresholds and NRS scores and an increase in cervical lateral flexion ($p < 0.05$). These changes were also significant in all groups at the 12 week follow-up ($p < 0.05$). With regard to pressure pain thresholds, when comparing between treatments, at post treatment there was no statistically significant differences detected between active therapies vs placebo therapies, whereas the control group scored significantly less than both the active and placebo groups ($p < 0.05$; $d > 0.8$). No differences were found among the intervention groups at 12 week follow-up, while controls kept on scoring less than any group ($p < 0.05$, $d > 0.05$). While this was a well-designed study, the poor reporting of specific p- and d-values is worth noting. Regarding NRS, active low-level laser therapy scored significantly better than active ultrasound ($p = 0.04$), placebo ultrasound ($p = 0.03$) and control ($p = 0.002$) following 2 weeks of treatment, but not better than placebo low-level laser therapy ($p = 0.21$). Both active and placebo therapies had a significantly higher score than no therapy ($p < 0.05$, $d > 0.8$). At 12 weeks follow-up no significant differences were detected among groups with the exception of active ultrasound, which scored significantly higher than the control ($p =$

0.03) but not than any other intervention group. Regarding active cervical lateral flexion, no significant differences were found when comparing active with placebo therapies following two weeks. The control group scored significantly less than active therapies, active ultrasound ($p = 0.02$, $d = 0.8$) and active low-level laser therapy ($p = 0.02$, $d = 0.8$), but not than placebos. At 12 weeks follow-up, groups were not significantly different with the exception of active ultrasound which scored significantly better than control ($p = 0.02$, $d = 0.8$). The results from this study show that ultrasound and low-level laser therapy, delivered as stand-alone treatments, although being superior to no therapy, do not provide any additional benefit beyond that of their placebos.

Electrotherapy is another commonly used modality treatment for TrPs. Transcutaneous electric nerve stimulation (TENS) is one such electrotherapy that is commonly utilised (Pal et al., 2014). TENS involves the application of electrodes connected to a small battery powered unit along a painful muscle. The aim of the treatment is to act as an analgesia and relieve muscle tension (Pal et al., 2014). Rodríguez-Fernández et al. (2011) compared the effects of TENS to sham TENS over the upper trapezius muscle and observed a significant improvement in referred pressure pain thresholds ($p < 0.001$) and cervical rotation ($p = 0.01$) in favour of the TENS group. Between group differences for referred pressure pain thresholds were small at 1 minute ($0.3\text{kg}/\text{cm}^2$; 95% Confidence Intervals (CI): 0.1, 0.4) and at 5 minutes ($0.6\text{kg}/\text{cm}^2$; 95% CI: 0.3, 0.8) post-treatment. Similarly, between group differences for cervical rotation were also small at 1 minute (2.0° ; 95% CI: 1.0, 2.8) and at 5 minutes (2.7° ; 95% CI: 1.7, 3.8) post-treatment (Rodríguez-Fernández et al., 2011). Thus the results from this study reveal that TENS can cause a mild hypoalgesic effect for mechanical stimuli, by increasing referred pressure pain thresholds, and small changes in cervical range of motion, however due to the magnitude of these changes they may have limited clinical relevance.

2.1.5.3 Needling Therapies

The insertion of needles into the body for therapeutic purposes is a long established practice, primarily in China since approximately the 7th century A.D. There are a number of different methods of needling used, but they can be grouped into one of two groups, dry needling and wet needling. Dry needling refers to treatments that do not involve delivery of an injectable substance whereas wet needling does. Dry needling was historically developed from Dr Janet Travell's injection techniques (wet needling) for the

treatment of TrPs with the use of a hypodermic needle. It was speculated that the effects from injection of the needle were related to the physical action of the needle and the evocation of a local twitch response rather than the effects of the injectable. It was because of the lack of an injectable that the term “dry needling” was coined.

A variety of injectable substances have been used in the treatment of TrPs using the wet needling technique. In a systematic review of needling, Cummings & White (2001) identified that the following substances have been utilised as an injectable: bupivacaine, saline, naloxone, mepivacaine, lidocaine, lignocaine, botulinum toxin type A, prednisolone, diclofenac, lignocaine, methyl prednisolone acetate, and etidocaine. Cummings & White (2001) also reported that when treating TrP pain with injection, the nature of the injected substance makes no difference to the outcome, and that wet needling is not therapeutically superior to dry needling (Cummings and White, 2001).

Dry needling is now typically performed with acupuncture type needles, where the filament is solid and incapable of delivering an injectable substance. Although various dry needling approaches exist, the most common approach targets TrPs (Dommerholt, 2011). The advantages of TrP dry needling are increasingly documented (Dommerholt et al., 2006b) and the treatment falls within the scope of physical therapy practice in many countries. A systematic review and meta-analysis was carried out by Kietrys et al. (2013) on the effectiveness of ATrP dry needling for the upper-quarter. Twelve studies met the inclusion criteria and these studies examined the effects of dry needling immediately post treatment and at approximately 4 weeks follow-up. The included studies compared dry needling to a number of treatments, including sham treatment and control. Four studies compared the immediate effects of dry needling to sham or control on pain (Hsieh et al., 2007; Irnich et al., 2002; Tekin et al., 2013; Tsai et al., 2010). Kietrys et al. (2013) reported that the overall effect size for the four studies (Hsieh et al., 2007; Irnich et al., 2002; Tekin et al., 2013; Tsai et al., 2010) was 1.06 (95% CI: 0.05, 2.06), which suggested a large effect favouring dry needling over sham or control. However, there was a large 95% CI range observed, making it difficult to come to a definitive conclusion on the effectiveness of dry needling.

Hsieh et al. (2007) conducted their study on fourteen patients with bilateral shoulder pain where ATrPs in their bilateral infraspinatus muscles were involved. An ATrP in the

infraspinatus muscle on a randomly selected side was treated with dry needling and the ATrP on the contralateral side was not, acting as a control. Shoulder pain intensity, range of motion of shoulder internal rotation, and pressure pain thresholds of the ATrPs in the infraspinatus, anterior deltoid, and extensor carpi radialis longus were investigated before and immediately after dry needling. Results from this study demonstrated that both active and passive range of motion of shoulder internal rotation and the pressure pain thresholds of ATrPs on the treated side significantly increased ($p < 0.01$), and the pain intensity of the treated shoulder was significantly reduced ($p < 0.001$) after dry needling. There were no significant changes demonstrated in any of the outcome measures in the control side. Interestingly this study demonstrated that dry needling treatment for ATrPs in the infraspinatus muscle not only showed a reduction in pressure pain thresholds of the treated muscle but also the anterior deltoid and extensor carpi radialis longus muscles, which are perceived to be located in the referral zone of TrPs in the infraspinatus. While this study by Hsieh et al. (2007) did include a control it did not compare dry needling to a placebo treatment. Again, similar to the Manca et al. (2014) study on ultrasound and low-level laser, this study did not report exact p-values.

Irnich et al. (2002) conducted a study on thirty six patients with chronic neck pain and limited cervical spine mobility. Every patient was treated once with needle acupuncture at a distant point, dry needling of local TrPs and sham laser acupuncture. The outcome measures for this study were motion-related pain intensity measured using 0-100 millimetre (mm) visual analogue scale, and range of motion. In addition to these, patients scored changes of general complaints using an 11-point verbal rating scale. All outcome measures were recorded immediately before and after each treatment by a blinded investigator. To eliminate carry-over treatment effects, a 1 week wash-out period between treatments was employed. Results from this study demonstrated that for motion related pain, the use of acupuncture at non-local points reduced pain scores by about a third, 11.2 mm ($p < 0.0001$; 95% CI: 5.7, 16.7) compared to dry needling which had a reduction of 1.0 mm ($p = 0.7$; 95% CI: 4.5, 6.5), suggesting that non-local acupuncture is effective and dry needling ineffective compared to sham. Range of motion scores demonstrated slight improvement immediately following dry needling and non-local acupuncture compared to sham treatment. Range of motion following dry needling improved by 1.7° ($p = 0.032$; 95% CI: 0.2, 3.2) compared to sham, while range

of motion improved by an additional 1.9° ($p = 0.016$; 95% CI: 0.3, 3.4) following non-local acupuncture. For patient assessment of change, non-local acupuncture was significantly superior both to sham treatment, 1.7 points ($p = 0.0001$; 95% CI: 1.0, 2.5), and dry needling, 1.5 points ($p = 0.008$; 95% CI: 0.4, 2.6). Similar results were found when differences between groups for change assessment were analysed by non-parametric methods: non-local acupuncture, $p = 0.0003$; dry needling, $p = 0.007$; and sham, $p = 0.6$. The results of this study demonstrated that non-local acupuncture was a more effective treatment option than dry needling of local TrPs when comparing them to a sham treatment.

Tekin et al. (2013) conducted their study on thirty nine subjects with established TrPs and examined dry needling and sham dry needling as a treatment of MPS. Dry needling was applied using acupuncture needles and sham dry needling was applied using a blunted needle. The blunted needle caused a pricking sensation without penetrating the skin. Treatment composed of six sessions which were performed in 4 weeks. The outcome measures used in this study were the 10 cm visual analogue scale and Short Form-36 (SF-36) used to evaluate quality of life. Participants were evaluated three times as follows: initially prior to treatment for visual analogue scale and SF-36, after the first treatment session, only for visual analogue scale, and after the 4 weeks, using the visual analogue scale and SF-36. Results from this study demonstrated that visual analogue scale scores of the dry needling group were significantly lower at both recording sessions compared to before treatment, 2.6 cm improvement following the first session ($p < 0.001$) and a 4.4 cm improvement following the last session ($p < 0.001$). The visual analogue scale scores of the sham dry needling group were also significantly lower at both recording sessions compared to before treatment, 1.0 cm improvement following the first session ($p = 0.001$) and 1.1 cm improvement following the last session ($p = 0.17$). When visual analogue scale scores were compared between the dry needling and sham dry needling groups the before treatment scores were found to be similar, but the scores after the first treatment and last treatment were found to be significantly lower in the dry needling group ($p = 0.034$ and < 0.001 , respectively). When the SF-36 scores of the groups were compared, both the physical and mental component scores were found to be significantly increased in the dry needling group (all $p < 0.05$), whereas only those of vitality scores were found to be increased significantly in the sham dry needling group

($p < 0.05$). The results from this study demonstrated that when compared with sham dry needling, patients who were treated with dry needling showed better improvement with regard to pain and quality of life assessment.

Tsai et al. (2010) conducted their study on thirty five patients with ATrPs in the upper trapezius muscles. Participants were divided into a control group, which received sham dry needling, and a dry needling group; both groups received dry needling in the extensor carpi radialis longus muscle. Dry needling in this study used a 5 ml syringe connected with a 25-hypodermic needle (0.5 mm in diameter), 1 ½ inches in length as opposed to acupuncture needles used in other studies. The sham dry needling that was utilised in this study involved the needle penetrating the skin into the subcutaneous layer over the suspected TrP region. The needle was then moved in the same manner and same speed as the dry needling technique but of a different depth so that the needle tip was maintained in the subcutaneous tissue without further penetration into the muscle tissue. Though this was referred to as sham dry needling in this study this technique is commonly referred to as superficial dry needling. The outcome measures used in the study were subjective pain intensity, rated from 0-10, pressure pain thresholds, and range of motion of the cervical spine. Unfortunately specific p-values were not reported in this study. The study reported a significant decrease in the mean pain intensity after treatment in the dry needling group ($p < 0.05$), but not in the sham dry needling group ($p > 0.05$). After normalisation of the data into percentage of improvement, the degree of improvement in the subjective pain relief was significantly higher ($p < 0.05$) in the dry needling group than the sham dry needling group. Regarding pressure pain thresholds, the mean pressure pain thresholds were significantly increased after treatment in the dry needling group ($p < 0.05$), but not in the sham dry needling group ($p > 0.05$). There was also a significantly higher degree of improvement in the pressure pain thresholds in the dry needling group compared to the sham dry needling group ($p < 0.05$). Finally regarding range of motion, there was a significant increase in the mean range of motion after treatment in the dry needling group ($p < 0.05$), but not in the sham dry needling group ($p > 0.05$). The degree of improvement in the range of motion of neck side bending was significantly higher in the dry needling group ($p < 0.05$). However, the results of this study must be viewed with caution as the

sham dry needling technique used in this study replicated superficial dry needling and therefore was not a true sham treatment.

Kietrys et al. (2013) also reviewed three studies which compared the effects of dry needling to sham or control at 4 weeks post-treatment (Ilbuldu et al., 2004; Itoh et al., 2007; Tekin et al., 2013). The overall effect size for the three studies reported (Ilbuldu et al., 2004; Itoh et al., 2007; Tekin et al., 2013) was 1.07 (95% CI: -0.21, 2.35) suggesting a large effect favouring dry needling. Kietrys et al. (2013) did however note that the 95% CI crossed the line of no difference, suggesting that caution should be used when making conclusions based on overall effect size. Two of the three studies (Itoh et al., 2007; Tekin et al., 2013) favoured dry needling over the sham or control at 4 weeks, and both had large effect sizes (1.95 and 1.5, respectively).

Ilbuldu et al. (2004) investigated the effects of laser, dry needling and placebo laser on ATrPs on sixty patients at three time points, before treatment, after 4 weeks of treatment, and a follow up at 6 months. They observed a significant decrease in pain at rest ($p < 0.05$), at activity ($p < 0.001$), and increase in pain threshold ($p < 0.001$) in the laser group compared to other groups after 4 weeks of treatment (Ilbuldu et al., 2004). However, there was no difference in any of the parameters at the 6 month follow up. A weakness of this study was the disparity in dosage of each treatment group, with patients in both laser groups receiving treatment three times a week and the dry needling group only once.

Itoh et al. (2007) conducted their study on forty patients with non-radiating chronic neck pain. Participants were randomised into one of four groups; the acupuncture group, which received treatment at traditional acupoints for neck pain; the TrP group, which received dry needling treatment; the non-TrP group, which received treatment at non-tender points 50 mm away from TrPs; and a sham acupuncture group, which were treated with stainless steel needles (0.2 mm x 50 mm), but the tips had been cut off and smoothed to prevent the needle penetrating the skin. Each group received two phases of treatment with an interval phase between them, all phases lasted 3 weeks and the total experiment period was 13 weeks. Participants received a total of 6 treatments, one per week, each lasting for 30 min. The outcome measures used in this study were pain intensity, measured with a 0-100 mm visual analogue scale, and a disease specific quality

of life questionnaire, the neck disability index (NDI), which has a 60-point scale. Results from this study, relative to visual analogue scale scores, demonstrated that the mean score tended to decrease in all groups. In the TrP group, statistically significant differences were seen when comparing the visual analogue scale scores pre-treatment (67.0 ± 13.2 mm) with 3 weeks later (18.6 ± 18.05 mm, $p < 0.01$). This improvement was also present following the interval period (26.1 ± 22.3 mm, $p < 0.05$). There were no significant differences between pre-treatment scores and later scores for the acupuncture group, non-TrP group or sham acupuncture groups. By the end of the second treatment period (9 weeks after the start of treatment), the TrP group reported relatively lower pain intensity than the other groups, however the differences were only significant in the TrP group ($p < 0.01$). In relation to NDI scores, the scores tended to decrease at 3 weeks after the first treatment. In the TrP group, a statistically significant difference was observed comparing pre-treatment scores (13.0 ± 6.3) with 3 weeks later (3.9 ± 3.4 , $p < 0.01$), but there was no significant reduction seen in any of the other groups. By the end of the second course of treatment, only the TrP group reported a statistically significant difference ($p < 0.01$). The results from this study suggest that TrP dry needling treatment may be more effective than other acupuncture treatments for chronic neck pain.

Kietrys et al. (2013) concluded that based on the studies published at the time of its analysis, dry needling was recommended as a grade A treatment, compared to sham or placebo, for immediate reduction of pain in patients with upper quarter MPS. They also, cautiously, recommended dry needling as a grade A treatment compared to sham or placebo for reduction in pain at 4 weeks. However, Harvie et al. (2014) critiqued this systematic review and meta-analysis, suggesting that the quality of papers included in the review, the interpretation of the results, and the subsequent recommendations may be questionable. The questioning of the Kietrys et al. (2013) is certainly warranted due to the inconsistent results observed, and particularly due to the wide 95% CI reported for the effect sizes of the studies. The issues raised by Harvie et al. (2014) highlights the need for high quality research in this area.

2.2 The Shoulder Complex

This second section focuses on the shoulder complex, the region of the body in which this research investigated the effectiveness of LTrP dry needling to change MAPs, and has been divided into seven subsections. Firstly a brief overview of the anatomy of the shoulder complex is provided prior to detailing its intricate kinematics. This is followed by a review of scapular dyskinesis and its proposed role in shoulder pathology. The assessment methods that have been developed for the scapula and scapular dyskinesis are then discussed followed by the current understanding of the role of MAPs and how they are measured. Finally, the shoulder complex section concludes with the discussion of whether scapular dyskinesis is a topic of concern or whether it is a result of natural variation.

2.2.1 Anatomy of the Shoulder Complex

The shoulder is a complex system of a number of joints and muscles working together to allow for a large range of upper limb mobility. This complex interaction between passive and active structures facilitates the stability of the upper limb during the variety of orientations available (Voight and Thomson, 2000). The shoulder complex is formed by three joint articulations and a quasi-joint (Figure 2.6): the sternoclavicular (SC) joint, the acromioclavicular (AC) joint, the glenohumeral (GH) joint, the scapulothoracic (ST) joint. The collective actions of these joints make the shoulder complex the most mobile joint complex in the body, however, this mobility comes at the price of stability (Osar, 2012).

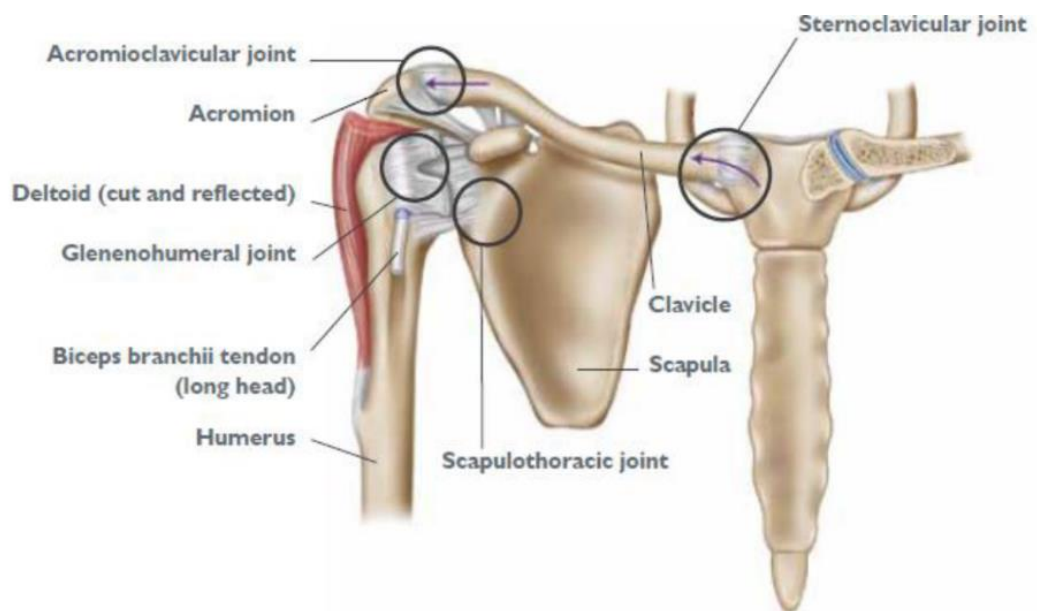


Figure 2.6 The shoulder complex and its four articulations [reproduced from Osar (2012)]

The synovial SC joint is the only bony attachment of the shoulder complex and upper limb (appendicular skeleton) to the spine (axial skeleton) (Osar, 2012). It is a saddle joint consisting of the medial end of the clavicle and the manubrium of the sternum, however, it functions as a ball and socket joint (Palastanga and Soames, 2012). The SC joint is stabilised passively by the sternoclavicular, interclavicular and costoclavicular ligaments and actively by the sternocleidomastoid, pectoralis major and the subclavius muscles.

The AC joint aids in the optimal positioning of the scapula for overhead motion (Osar, 2012). It is classified as a diarthrodial joint and is formed by the distal end of the clavicle and the medial aspect of the acromion process of the scapula (Di Giacomo et al., 2008). The AC joint is stabilised by both static and dynamic stabilisers. The static stabilisers include the AC ligaments (superior, inferior, anterior and posterior), the coracoclavicular ligaments (trapezoid and conoid) and the coracoacromial ligament (Di Giacomo et al., 2008). The dynamic stabilisers include the deltoid, trapezius and subclavius muscles (Di Giacomo et al., 2008; Osar, 2012).

The GH joint is an enarthrodial, or ball and socket, joint with the head of the humerus forming the ball and the glenoid fossa of the scapula forming the socket. The GH joint has the greatest freedom of movement of any joint, however, this is at the expense of stability (Osar, 2012). This instability is due to the rather shallow glenoid fossa into which the rounded head of the humerus sits (Kibler and Sciascia, 2015). The articulation

between these two surfaces account for very little, if any, stability of the joint. The GH joint is instead stabilised by both passive and active mechanisms. The glenoid labrum, joint capsule and the capsular ligaments act as the passive supports while the deltoid and rotator cuff muscles act as the active supports.

The ST joint is formed by a pseudo-articulation of the scapula on the thorax. The ST joint has been characterised as a physiological joint between the anterior aspect of the scapula and the posterolateral aspect of the chest wall (Mottram, 1997). It is not a true joint as it lacks ligamentous support, a joint capsule, a synovial membrane, and synovial fluid, but its movement is vital to shoulder complex function. Movements of the ST joint serve to increase the range of movement of the GH joint, by changing the relative position of the glenoid fossa with respect to the thoracic spine (Palastanga and Soames, 2012). In doing this it optimises the alignment of functional supports of the GH joint. Di Giacomo et al. (2008) have stated that there are four roles of the ST joint, all of which play a vital role in the function of the shoulder complex. Firstly, to maintain the integrity of the GH joint the scapula must move in a coordinated manner with the moving humerus, so that the instant centre of rotation is constrained within a physiological pattern throughout full range of shoulder motion. Secondly, the scapula must provide motion along the thoracic wall. This motion creates a stable platform for the upper limb during a variety of tasks, such as reaching, pushing or pulling. Thirdly, to avoid impingement and coracoacromial arch compression the scapula must elevate the acromion. This is required during throwing or elevation of the arm. Finally, the scapula must act as a link between the proximal and distal parts of the body in order to transfer large forces and high energy from the legs, back and trunk to the delivery points, such as the arm and hand.

In order for these roles to be achieved the scapula has a number of movements at its disposal. The scapula is commonly described as having six degrees of freedom; adduction and abduction, retraction and protraction, depression and elevation, downward and upward rotation, internal and external rotation, and anterior and posterior tilting. The precise kinematics of the ST joint, along with the other joints, is detailed in section 2.2.2. Due to the shoulders limited bony, capsular and ligamentous constraints the muscles that interact with it play a vital role in both its stability and optimising its function. This is due to the curvature of the humeral head being larger

than the curvature of the glenoid, minimising joint stability, and the fact that the supporting ligaments are only taut at the end ranges of GH motion (Kibler and Sciascia, 2015). It is therefore vital the positioning muscles of the scapula function adequately, to optimise the position the glenoid (Kibler and Sciascia, 2015).

Of the seventeen muscles that attach to the scapula there are three that have the greatest effect on its positioning during overhead movements: the serratus anterior, the trapezius, and the pectoralis minor muscles. The serratus anterior muscle acts together with the trapezius muscle to provide a very strong and mobile base of support which is designed to optimise the position of the glenoid fossa. This optimising of the glenoid fossa's position allows for optimal effective use of the rotator cuff muscles which control the glenohumeral joint (Kibler and Sciascia, 2015). The serratus anterior muscle is composed of three functional portions. These portions arise from a series of slips from the lateral aspect of the upper eight ribs and their corresponding fascia and insert into the anterior medial aspect of the scapula (Figure 2.7). The upper slip passes horizontally backwards to the superior angle of the scapula. This slip accounts for a large portion of the serratus anterior muscle mass. It acts as the main axis of rotation at the superior medial border of the scapula, effectively anchoring it and allowing the rotation required to lift the arm over the head. It works in conjunction with the trapezius muscle on the dorsal aspect where it attaches to the base of the spine of the scapula. The second to fourth slips insert into the medial border and these fibres draw the scapula forward around the thoracic cage (Di Giacomo et al., 2008). The lower four slips pass obliquely upwards and backwards converging to the inferior angle of the scapula.

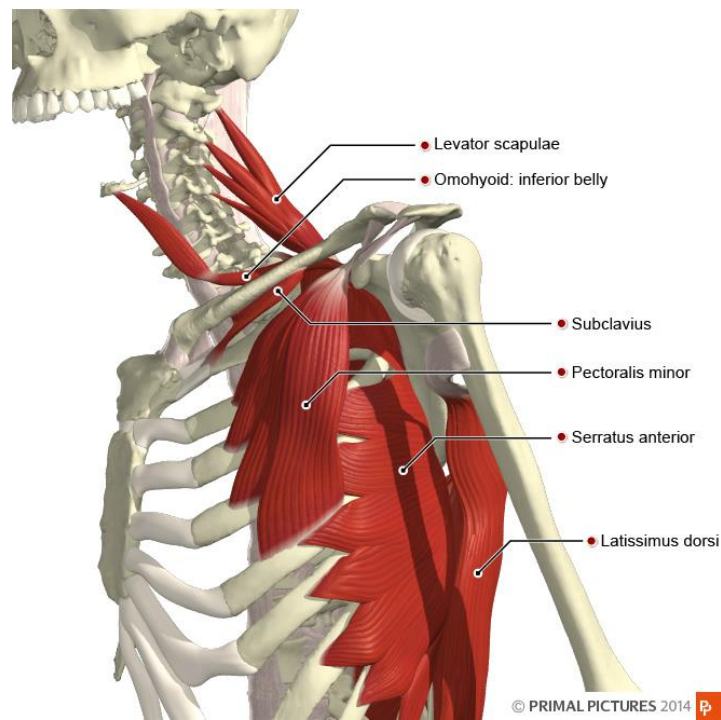


Figure 2.7 Deep muscles of the shoulder [reproduced from www.anatomy.tv]

Due to the inherent instability of the GH joint, the ability to position and control movements of the scapula is essential for optimal upper limb function (Mottram, 1997). Knowledge of the origin and insertions of these muscles which stabilise and move the scapula assist in the understanding of their role in optimal shoulder complex function and whether they are involved when it is lacking. Stabilisation of the scapula on the thorax involves the combined coupling of the upper and lower fibres of the trapezius muscle with the serratus anterior and the pectoralis minor muscles (Speer and Garrett, 1994). Elevation of the scapula involves a slight alteration with the activation and coupling of the serratus anterior and the lower trapezius with the upper trapezius and pectoralis minor muscles (Bagg and Forrest, 1986; Speer and Garrett, 1994).

2.2.2 Kinematics of the Shoulder Complex

Understanding the 3-dimensional (3-D) motion of the shoulder complex is essential in order to understand motion-related abnormalities. In clinical settings the use of visual evaluation tools are often used to determine abnormal motion from what is considered normal. It is not known whether any altered motion is causative or compensatory in the painful shoulder, but recognising and treating motion abnormalities will be hindered unless what is considered normal motion is well understood.

During shoulder elevation substantial motion occurs at all four joints, the SC, AC, ST and GH. Motion at each of these joints contributes to the freedom of motion of each other. Therefore, any abnormal or restricted motion at one joint could be expected to affect the other joints. To understand the interplay between these four joints of the shoulder complex the specific kinematics that occurs at each joint during shoulder flexion (coronal plane) and abduction (frontal plane) in symptomatic and asymptomatic groups will be detailed. The kinematic data from this section was taken from two cross-sectional studies by Lawrence et al. (2014a and 2014b) who used simultaneous tracking of bone-fixed sensors to allow for highly accurate assessment of motion and the biomechanical relationships between the joints of the shoulder complex. Lawrence et al. (2014a and 2014b) compared differences in SC, AC, ST and GH motion between symptomatic and asymptomatic individuals during shoulder motion performed in three planes of humerothoracic elevation. In keeping with the planes of motion that have been investigated in this thesis only flexion and abduction will be discussed.

2.2.2.1 Kinematics of the Sternoclavicular Joint

Angular motions that occur at the SC joint are described as clavicular movement relative to the sternum. These motions are elevation/depression, anterior/posterior rotation, and protraction/retraction. Lawrence et al. (2014a) demonstrated that during both flexion and abduction elevation of the shoulder symptomatic and asymptomatic groups demonstrated similar patterns of SC motion. As humeral elevation increased, both groups demonstrated consistent progression in all SC motions (Figure 2.8). Both groups demonstrated consistent SC elevation during humerothoracic elevation. The symptomatic group started with less SC elevation however, they achieved the same values at end of range. The pectoralis minor has the greatest effect on SC elevation and increased activity of it could have been a confounding factor in the measures of SC elevation, causing the reduced humerothoracic elevation in the symptomatic group.

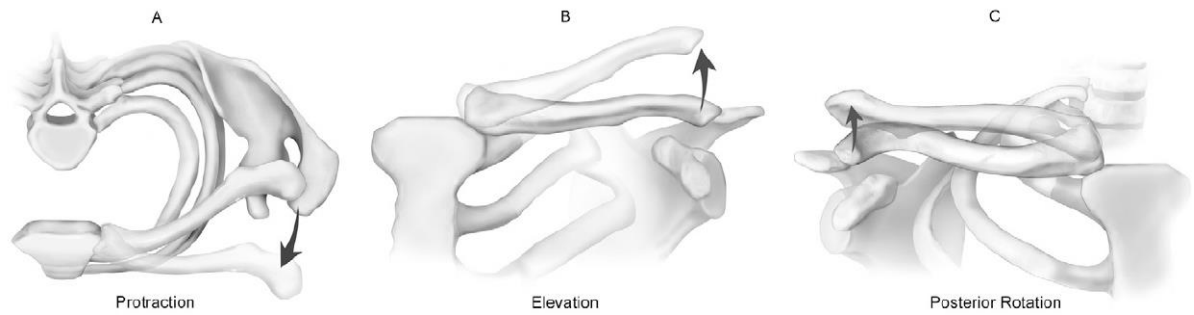


Figure 2.8 Motions of the clavicle (A) protraction-retraction (as seen from the superior view of a right shoulder), (B) elevation-depression (as seen in the anterior view of a right shoulder, and (C) anterior-posterior rotation (as seen in the lateral view of a right shoulder) [reproduced from Ludewig et al. (2009)]

Both groups showed consistent SC posterior rotation with humerothoracic elevation however, the symptomatic group had on average 5.9° less during flexion ($F(1, 19) = 12.15, p = .003$) and 5.2° less during abduction ($F(1, 19) = 8.55, p = 0.009$). Interestingly, SC posterior rotation was consistently decreased in the symptomatic group regardless of angle, phase, or plane of humerothoracic elevation. No muscle has been shown to directly produce sternoclavicular axial rotation therefore this motion is believed to occur as the result of scapular motion, specifically upward rotation of the scapula, which in turn produces joint motion through tension in the acromioclavicular and coracoclavicular ligaments (Lawrence et al., 2014a). Again, the pectoralis minor muscle may have had a role to play in this reduced SC posterior rotation. Reduced pectoralis minor passive lengthening is said to be one of the limiting factors to optimal scapular upward rotation, external rotation, and posterior tilting (Borstad and Ludewig, 2005). Borstad & Ludewig (2005) demonstrated in their study on fifty subjects that those classified as having a short pectoralis minor muscle, consistently had significantly reduced scapular posterior tilting and external rotation. For example, in the coronal plane Borstad & Ludewig (2005) demonstrated that the short pectoralis minor group had 10.2° less external rotation at 30° arm elevation, 10.5° at 60°, and 10.5° at 90°, ($F(3, 141) = 5.19; p < 0.005$). The short pectoralis minor group also showed significantly reduced posterior tilting, with 7.1° less at 90° arm elevation and 9.1° at 120° ($F(3, 141) = 12.20; p < 0.00001$). In relation to the effects of a short pectoralis minor muscle on upward rotation Borstad & Ludewig (2005) stated that because upward rotation has more total motion compared to posterior tilting and external rotation, the effects seen in the short pectoralis minor group may have been too small to be detected.

Lawrence et al. (2014a) also demonstrated that SC retraction was the consistent motion in both groups during humerothoracic elevation. The asymptomatic group tended to start with greater retraction and maintained it throughout humerothoracic elevation, however the differences between groups were not of significance. Interestingly, at early stages of humerothoracic elevation, there is typically a larger difference between groups from approximately 30 to 60 degrees of elevation after which their values merge.

2.2.2.2 Kinematics of the Acromioclavicular Joint

Angular motions that occur at the AC joint are described as scapular (acromion) movement with respect to the clavicle. These angular motions include internal/external rotation, upward/downward rotation, and anterior/posterior tilting. Lawrence et al. (2014a) demonstrated that during both humerothoracic flexion and abduction both groups demonstrated progressive AC joint internal rotation, upward rotation, and posterior tilting, without any significant differences seen between the groups. The progressive internal rotation of the AC joint was due to the motion of the scapula around the thoracic cage causing the acromion to internally rotate relative to the clavicle. Similarly, upward rotation of the AC joint was caused as a consequence of upward rotation of the scapula during humerothoracic elevation. Finally, posterior tilting was caused as the lower fibres of the trapezius muscle cause posterior tilting of the scapula, thus causing a similar motion to occur at the AC joint.

The AC joint plays an important role as it transfers forces from the ST motion to the SC joint. Posterior rotation of the SC joint is thought to occur through tension in the acromioclavicular and coracoclavicular ligaments (Lawrence et al., 2014a). In line with this transfer of forces from the ST to the SC joints, it is clear that the AC joint has an important role to play in the relationship between these joints. This relationship between the SC, AC, and ST joints has been coined as coupling (Teece et al., 2008). The coupling theory proposes that abnormal SC or AC joint motion may lead to and/or result from abnormal ST motion (Lawrence et al., 2014a). This coupling theory is crucial to develop biomechanical theories for explaining pathology and potential causative or compensatory movement patterns, however, the mechanisms of these interactions are not well understood (Lawrence et al., 2014a).

2.2.2.3 Kinematics of the Scapulothoracic Joint

Angular motions that occur at the ST joint are described as movement of the scapula with respect to the thorax. These angular motions include upward/downward rotation, anterior/posterior tilt, and internal/external rotation. Lawrence et al. (2014a) demonstrated that during both flexion and abduction of the shoulder both asymptomatic and symptomatic groups had progressive ST joint upward rotation with increasing angles of humerothoracic elevation (Lawrence et al., 2014a). In humerothoracic abduction, differences were found at lower angles of abduction ($F(1, 58) = 3.10, p = 0.034$). The symptomatic group demonstrated 6.5° less upward rotation at 60° of arm raising ($F(1, 58) = 7.97, p = 0.007$) and 6.3° less upward rotation at 30° of arm lowering ($F(1, 58) = 7.60, p = 0.008$). The reduced scapular upward rotation in the symptomatic group during the early stages of humerothoracic elevation may imply that the symptomatic group caught up with the asymptomatic group at end of range humerothoracic elevation. Therefore, potentially it is a timing issue rather than a true lack of scapular upward with the symptomatic group.

For ST joint tilt, both groups demonstrated progressive posterior tilt during arm raising (Lawrence et al., 2014a). Differences were observed between groups towards the end of range elevation and as participants began to descend from this position, however, no significant differences were found between the groups. While this finding was not statistically significant, there may be a practical implication. Symptomatic patients typically have pain at end of range humerothoracic elevation. The inability of the symptomatic group to achieve the same level of ST posterior tilting as the asymptomatic group is likely to increase the impingement of subacromial structures. The muscles that may be implicated are those that account for posterior tilting of the scapula, in particular the lower fibres of the trapezius muscle and the pectoralis minor, acting as its antagonist.

Lawrence et al. (2014a) also found that the extent to which the scapula internally or externally rotated on the thorax during flexion and abduction was highly variable between participants. It is likely due to this high variability between participants that there was no significant differences seen between groups.

2.2.2.4 Kinematics of the Glenohumeral Joint

Angular motions of the GH joint are described as humeral motion with respect to the scapula (glenoid). These angular motions are elevation/depression, anterior/posterior, and internal/external rotation. Lawrence et al. (2014b) demonstrated that during humerothoracic elevation in both flexion and abduction both symptomatic and asymptomatic participants showed consistent humeral elevation relative to the glenoid (glenohumeral elevation).

Anterior/posterior angular motions refer to the position of the humerus relative to the plane of the scapula. Glenohumeral elevation varies depending on the plane of humerothoracic motion in which participants move. The plane of glenohumeral elevation is in reference to the scapula plane. As would be anticipated the plane of glenohumeral elevation was anterior during shoulder flexion and posterior during shoulder abduction. During shoulder flexion group differences depended on the angle of humerothoracic elevation ($F(3, 57) = 4.55, p = 0.006$). The differences between groups increased as humerothoracic elevation increased, with the humerus of symptomatic participants tending to be less anterior to the plane of the scapula compared to the asymptomatic group. However this difference was not found to be significantly different after follow-up tests ($p = 0.066$) (Lawrence et al., 2014b).

During shoulder flexion the humerus consistently externally rotated relative to the glenoid with increasing angles of humerothoracic elevation (Lawrence et al., 2014b). However during shoulder abduction, the humerus showed a pattern of increasing external rotation until approximately 55° of humerothoracic elevation, followed by decreasing external rotation, or relative internal rotation, for the remainder of the motion (Lawrence et al., 2014b). This was likely due to the progressive upward rotation of the scapula causing a change in the glenoid's orientation with increasing angles of humerothoracic elevation.

Linear translations also occur at the GH joint. These linear translations are described as anterior/posterior and superior/inferior translations of the humeral head relative to the glenoid. Unlike the angular motions of the joint, Lawrence et al. (2014b) described linear translations during interval of humerothoracic elevation: 30° - 60° , 60° - 90° , and 90° - 120° . During shoulder flexion, Lawrence et al. (2014b) demonstrated that both groups

demonstrated patterns of slight posterior GH translation during the 30°-60° interval, followed by anterior translation until 120° of humerothoracic elevation (Figure 2.9 A). During shoulder abduction however, the humeral head was consistently in an anteriorly translated position relative to the glenoid in both the asymptomatic and symptomatic groups (Figure 2.10 A) (Lawrence et al., 2014b). Group differences in GH anterior translation during shoulder flexion were only significant during the interval of 90°-120° of humerothoracic elevation, when the symptomatic group showed 1.4 mm more anterior GH translation ($F(2, 37) = 8.41, p = 0.001$) (Figure 2.9 A). During shoulder abduction Lawrence et al. (2014b) revealed that differences between groups in anterior GH translation depended on the interval of motion (Figure 2.10 A). However, the mean differences between groups did not reach statistical significance.

In relation to patterns of superior/inferior GH translation it varied between planes of humerothoracic elevation. During shoulder abduction, Lawrence et al. (2014b) demonstrated that the humeral head translated inferiorly relative to the glenoid in both asymptomatic and symptomatic groups (Figure 2.10). There was an average difference of 1.0 mm more inferior translation across all ranges of motion in the symptomatic group ($F(1,20) = 6.18, p = 0.022$) (Lawrence et al., 2014b). Similarly, during flexion the humeral head translated inferiorly however, there was no significant difference found between groups at any range of motion (Figure 2.9 B).

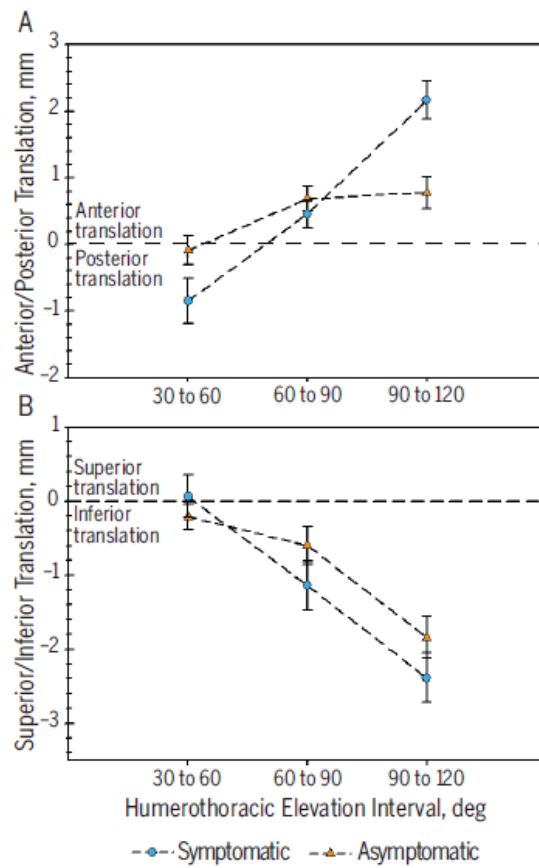


Figure 2.9 Translations of the humeral head relative to the glenoid during shoulder flexion: (A) anterior/posterior, (B) superior/inferior [reproduced from Lawrence et al. (2014b)]

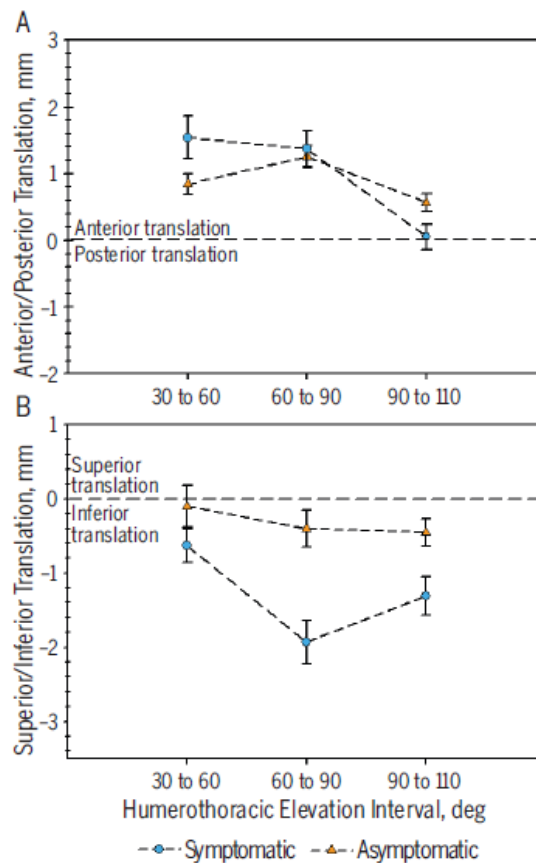


Figure 2.10 Translations of the humeral head relative to the glenoid during shoulder abduction: (A) anterior/posterior, (B) superior/inferior [reproduced from Lawrence et al. (2014b)]

The data presented above from the Lawrence et al. (2014a & 2014b) articles are key to furthering the understanding of the shoulder complex’s kinematics between symptomatic and asymptomatic populations. It is noteworthy that symptomatic participants who had reduced ST upward rotation at 30° and 60° humerothoracic demonstrated increased GH elevation at the same angles of humerothoracic elevation. This could imply that the reduction in ST upward rotation puts increased demand on the GH joint during the early stages of humerothroacic elevation.

2.2.3 Scapular Dyskinesis

Scapular dyskinesia is the term given to altered scapular motion and position (Kibler et al., 2009). The definition of dyskinesia is the alteration of normal scapular kinematics (Kibler et al., 2009). “Dys”, meaning alteration of and “kinesis”, meaning motion, reflects the loss of normal control of the scapular motion. As observed in the above sections detailing kinematics of the shoulder complex, abnormal motion can occur at all four joints of the shoulder. Visually observing these altered motions in a clinical setting is

challenging and motion of the ST joint is the most easily observed with the naked eye. This is due to the large amount of motion that occurs as the scapula goes through upward/downward rotation. This has led to the development of the term scapular dyskinesis, which specifically relates to altered scapular motion. Because of the broad number of conditions that may cause scapular dyskinesis, it by itself is not an injury or musculoskeletal diagnosis but it can be a contributing factor to one (Wright et al., 2012).

The multiple factors that may cause dyskinesis vary from bony causes to alteration of MAPs (Kibler et al., 2013). All possible causes are summarised in Table 2.3. In relation to soft tissue causes, inflexibility and stiffness of the pectoralis minor and biceps brachii short head can create anterior tilt and protraction of the scapula, due to their pull on the coracoid (Borstad and Ludewig, 2005). Soft tissue inflexibility in the posterior shoulder can lead to glenohumeral internal rotation deficit (GIRD), which creates a “wind up” of the scapula on the thorax, with reduced humeral internal rotation and horizontal abduction.

Table 2.3 Factors that may cause dyskinesis

Bony Causes	Increased thoracic kyphosis Clavicle fracture non-union or shortened mal-union
Joint Causes	High grade AC joint instability AC joint arthrosis Instability of GH joint Internal derangement of GH joint
Neurological Causes	Cervical radiculopathy Long thoracic nerve palsy Spinal accessory nerve palsy
Soft Tissue Causes	Inflexibility or intrinsic muscle problems

Alterations in periscapular muscle’s activation are related to scapular dyskinesis (Kibler et al., 2013). Serratus anterior activation and strength is decreased in patients with impingement and shoulder pain, contributing to the loss of posterior tilt and upward rotation causing dyskinesis (Cools et al., 2007). Alterations to normal activation of the upper and lower trapezius are also seen with scapular dyskinesis, with delayed onset of activation in the lower trapezius which alters scapular upward rotation and posterior tilt (Kibler et al., 2013). These alterations in MAPs cause a change in optimal scapular positioning and motion, resulting in a decrease in linear measures of the subacromial space (Atalar et al., 2009; Seitz et al., 2012; Silva et al., 2010), increased impingement

symptoms (Lukasiewicz et al., 1999), decreased rotator cuff strength (Kibler et al., 2006; Smith et al., 2002; Tate et al., 2008), increased strain on the anterior GH ligaments (Weiser et al., 1999), and increased risk of internal impingement (Mihata et al., 2010). Whether scapular dyskinesis is causative or compensatory to these conditions is unknown.

Despite the lack of understanding as to whether scapular dyskinesis is a cause or an effect of shoulder pain pathologies, its presence has the potential to impair normal function of the shoulder complex. The presence of scapular dyskinesis may make the system less efficient at carrying out its roles. Furthering the understanding of the role scapular dyskinesis plays in shoulder pathologies and the development of reliable and valid methods of assessing it are key to the effective treatment of such pathologies.

2.2.4 Clinical Assessment of the Scapula

Motion of the scapula, although objectively measurable in a laboratory setting with complex instrumentation such as 3-D tracking, is typically evaluated by clinicians using visual observation (Ellenbecker et al., 2012). Visual observation is primarily used by clinicians as it is time efficient, cheap, and easy to complete. The goal of scapular assessment is to identify abnormal scapular motion (dyskinesis), determine any relationship between altered motion and symptoms and identify the underlying causative factors of the movement dysfunction (Kibler and Sciascia, 2010; Kibler et al., 2009). Clinical assessment of scapular dyskinesis is inherently challenging due to the 3-D nature of scapular movement and soft tissue surrounding the scapula obscuring direct measurement of bony positioning (McClure et al., 2012). Due to these difficulties in reliably assessing the scapula there have been a number of proposed assessment methods.

There are two distinct types of clinical assessment of the scapula, static assessment which focus on the position of the stationary scapula, and dynamic assessments which attempt to classify movement abnormalities. Dynamic assessment methods also include evaluation of the scapula's position prior to movement occurring. Within these two types of clinical assessments there are varying methods to objectify the scapula's position and motion.

Static observation is the simplest method of observing the scapula and involves the assessment of the scapula's orientation on the thoracic spine in a stationary position. This observation can be performed in the frontal and the sagittal view with the arms held in predetermined positions. Observations of the scapula's positioning are often performed at rest, with both hands on the ipsilateral hips, and at 90° of shoulder abduction (Figure 2.11) (Struyf et al., 2014).

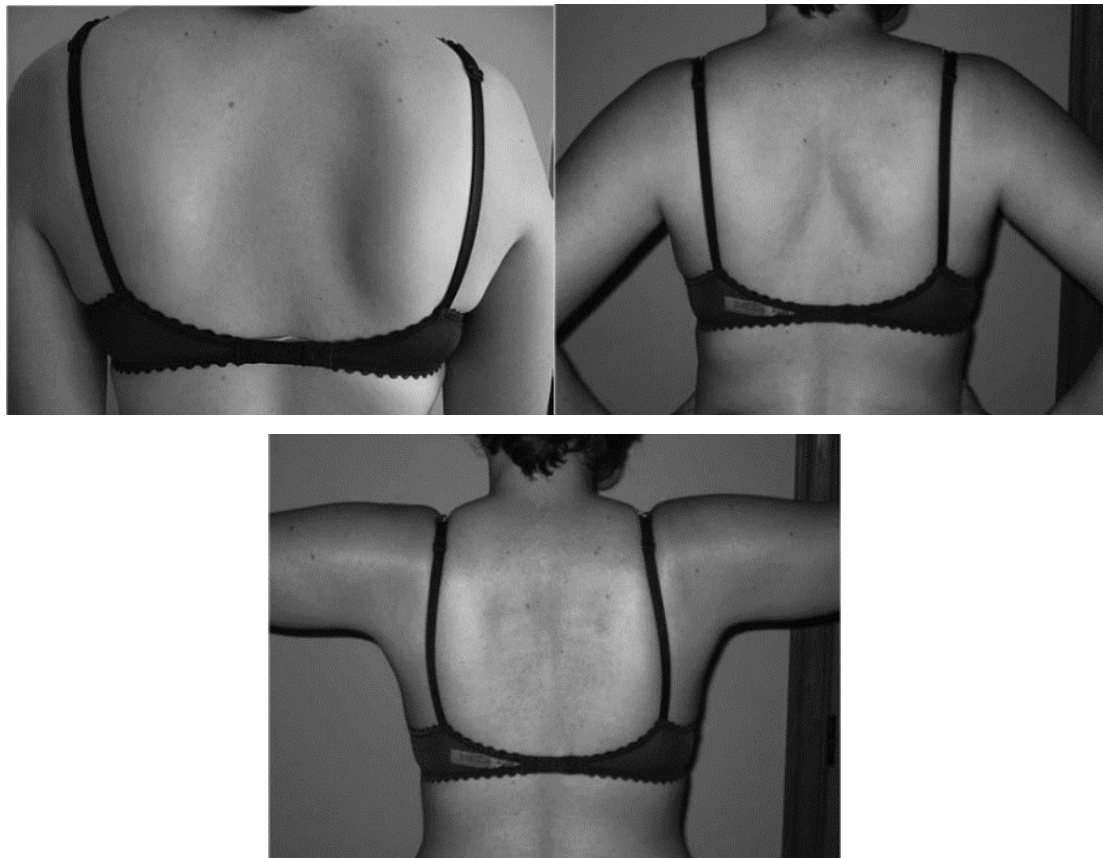


Figure 2.11 Observation of static scapular positioning at three positions [reproduced from Struyf et al. (2014)]

Struyf et al. (2014) suggest, that the appropriate scapular position at rest is with an approximately horizontal scapular spine, within +5° and -5° of scapular upward/downward rotation (Struyf et al., 2011), with the glenoid facing relatively downward in younger subjects and more upward in older subjects (Talkhani and Kelly, 2001). Struyf et al. (2014) also stated that the medial border of the scapula should be positioned parallel to the thoracic midline (Sobush et al., 1993), with the superior angle level with the spinous processes of T3 or T4, and the inferior angle with T7, T8, T9 or even T10 (Mottram, 1997).

There are a number of protocols used to assess the position of the scapula statically. Struyf et al. (2014) noted the reliability of four protocols, using the kappa coefficient (κ) (Table 2.4). Each of the listed protocols have a slight variation on how they rate tilting and/or winging of the scapula, which are two criteria typically used in clinical assessment of the scapula. Tilting refers to when the scapula has increased anterior tilting, which causes the inferior angle of the scapula to become more prominent. Winging refers to an increased internal rotation of the scapula, which causes the medial border of the scapula to become more prominent. These abnormalities may occur simultaneously or in isolation.

Table 2.4 Overview of reliability data of clinical observation of scapular positioning [reproduced from Struyf et al. (2014)]

Test	Kibler et al. (2002)	Struyf et al. (2009)	McClure et al. (2009)	Uhl et al. (2009)
Type I, II, III, IV (see Table 2.5)	$\kappa = 0.40$ (videotaped)			$\kappa = 0.44$ (direct observation)
Tilting (at rest versus during movement)		$\kappa = 0.48-0.52$		
Winging (at rest versus during movement)		$\kappa = 0.42-0.78$		
Yes (type I, II, III)/no (type IV) method				$\kappa = 0.41$
Dyskinesia (winging and/or dysrhythmia)			$\kappa = 0.48-0.61$	

κ = kappa coefficient

The main limitation to static scapular assessments are their inability to evaluate the scapula when it is in motion. It is possible to have no abnormalities present statically but they may occur during dynamic motion and vice versa. Therefore a key benefit of dynamic scapular assessment is its ability to assess abnormalities of the scapula during motion.

Dynamic scapular motion can be assessed in a variety of ways under two sub categories. These two sub categories include those that use objective scales to measure how much the scapula moves relative to another landmark, and those that aim to objectify the motion of the scapula, highlighting abnormal movement patterns. Commonly used assessment tests that use scales to measure movement on the scapula include the slide test (Odom et al., 2001) and measurement of scapular upward rotation (Watson et al.,

2005). However, these 2-dimensional (2-D) tests, similar to static tests, fail to fully assess the dynamic 3-D motion which occurs at the scapula. This limitation saw the development of visual dynamic assessment tests (Kibler et al., 2002; McClure et al., 2009; O'Connor et al., 2015; Uhl et al., 2009).

Visual dynamic assessment tests aim to classify the presence of scapular dyskinesis during shoulder motion. These methods are considered more functional and inclusive with the ability to judge scapular movement in 3-D patterns. Dynamic assessment tests use elevation of the humerus through full range of motion to assess scapular dyskinesis. The main difference between tests is the plane of motion used during the test. One or more of three planes of motion are commonly used: forward flexion in the sagittal plane, abduction in the coronal plane, or abduction in the scapular plane (scaption).

Kibler et al. (2002) were the first to describe a visually based test for rating scapular dysfunction, which involves bilateral arm elevation in scaption and abduction at a rate of 45°/second for three repetitions. The system incorporates three different types of abnormal scapular motion (type I to III) and one normal type to classify different types of scapular dyskinesis (type IV). Definitions of the four types of scapular motion are displayed in Table 2.5. Initial investigations into the reliability of this method of assessment reported relatively low levels of inter-tester reliability using the kappa (κ) coefficient, $\kappa = 0.31$ ($p < 0.01$) between two physicians, and $\kappa = 0.42$ ($p < 0.001$) between two physical therapists (Kibler et al., 2002). This was possibly due to fact that the classification of scapular dyskinesis attempted to distinguish differences between subtypes of scapular dyskinesis forcing testers to classify a subject's scapular dyskinesis into one type when it may have been a combination of two or more.

Ellenbecker et al. (2012) repeated the Kibler et al. (2002) test with the aim to improve the interrater reliability by refining the process. Ellenbecker et al. (2012) added greater detail to the evaluation process in a number of ways including, using a 4 second count to assist participant into elevation in a more controlled way, followed by a 1 second pause in full elevation before repeating the 4 second count during the lowering phase. They also included the classification of both scapulae for symmetry to the assessment protocol. However these modifications did not improve the tests reliability with poor to fair results, $\kappa = 0.18$; 95% CI: 0.11, 0.31 and $\kappa = 0.24$; 95% CI: 0.17, 0.31.

Table 2.5 Kibler et al. (2002) scapular dyskinesis system used to categorise abnormal scapular motion

Pattern	Definitions
Inferior angle (type I)	At rest, the inferior medial scapular border may be prominent dorsally. During arm motion, the inferior angle tilts dorsally and the acromion tilts ventrally over the top of the thorax. The axis of the rotation is in the horizontal plane.
Medial border (type II)	At rest, the entire medial border may be prominent dorsally. During arm motion, the medial scapular border tilts dorsally off the thorax. The axis of the rotation is vertically in the frontal plane.
Superior border (type III)	At rest, the superior border of the scapula may be elevated and the scapula can also be anteriorly displaced. During arm motion, the shoulder shrug initiates movement without significant winging of the scapular occurring. The axis of this motion occurs in the sagittal plane.
Symmetric scapulohumeral (type IV)	At rest, the position of both scapulae are relatively symmetrical, taking into account that the dominant arm may be slightly lower. During arm motion, the scapulae rotate symmetrically upward such that the inferior angles translate laterally away from the midline and the scapular medial border remains flush against the thoracic wall. The reverse occurs during lowering of the arm.

While the reliability of the Kibler et al. (2002) test did not prove to be high, it did set the foundation for subsequent visually based scapular dyskinesis evaluation protocols, which have tried to improve the reliability and/or specificity of assessment of scapular dyskinesis (McClure et al., 2009; O'Connor et al., 2015; Uhl et al., 2009).

Uhl et al. (2009) used the same classification system developed by Kibler et al. (2002) but simplified the 4-type classification into a 2-type classification. All three dyskinesis categories (types I to III) were grouped into a single “yes” category (an abnormal dyskinesis pattern was observed), and type IV was relabelled as “no” (normal scapular motion was observed). This yes/no assessment removed the requirement of the tester to decide on a single predominant pattern when multiple planes of asymmetry may have been observed. The yes/no assessment method yielded a 79% agreement, with a κ correlation of 0.44 ($p < 0.01$), and a sensitivity ranging from 74% to 78%. However, the specificity decreased in comparison to the 4-type classification, ranging from 31% to 38% for scaption and flexion respectively.

McClure et al. (2009) devised an alternative dynamic scapular dyskinesis assessment test, the scapular dyskinesis test (SDT), which did not attempt to distinguish among

subtypes of dyskinesia, instead each scapula was rated independently of the other side using a simplified rating system which evaluated the absence or presence of scapular dysrhythmia and winging, and its severity. The SDT rated scapular movement as being a normal motion or having subtle or obvious abnormality (Table 2.6). Testers were instructed to assess each scapula independently of the other side as previous studies found that asymmetry did not indicate dysfunction (Koslow et al., 2003). Using this method McClure et al. (2009) found that of the 142 participants that volunteered 89 (62.6%) had obvious dyskinesia unilaterally and 32 (22.5%) had obvious dyskinesia bilaterally, proving that the rating of scapular dyskinesia should not be based on measures of asymmetry. In addition, this study showed good inter-rater reliability of the test, with 75-82% agreement and weighted kappa (κ_w) = 0.48-0.61, likely due to the use of standardised training using videotaped examples of normal and abnormal motion along with the improved classification system.

Table 2.6 McClure et al. (2009) operational definitions & rating scale for the Scapular Dyskinesis Test

Operational Definitions

Normal scapulohumeral rhythm: The scapula is stable with minimal motion during the initial 30° to 60° of humerothoracic elevation, then smoothly and continuously rotates upward during elevation and smoothly and continuously rotates downward during humeral lowering. No evidence of winging is present.

Scapular dyskinesis: Either or both of the following motions may be present.

Dysrhythmia: The scapula demonstrates premature or excessive elevation or protraction, non-smooth or stuttering motion during arm elevation or lowering, or rapid downward rotation during arm lowering.

Winging: The medial border and/or inferior angle of the scapula are posteriorly displaced away from the posterior thorax.

Rating Scale

Each test movement (flexion and abduction) is rated as:

- a. **Normal motion:** no evidence of abnormality.
- b. **Subtle abnormality:** mild or questionable evidence of abnormality, not consistently present.
- c. **Obvious abnormality:** striking, clearly apparent abnormality, evident on at least 3/5 trials (dysrhythmias or winging of 1 in (2.54 cm) or greater displacement of scapula from thorax.

Final rating is based on combined flexion and abduction test movements.

Normal: Both test motions are rated as normal or 1 motion is rated as normal and the other as having subtle abnormality.

Subtle abnormality: Both flexion and abduction are rated as having subtle abnormalities.

Obvious abnormality: Either flexion or abduction is rated as having obvious abnormality.

O'Connor et al., (2015) developed a field based screening tool to assess for scapular dyskinesis. This assessment test aimed to further develop the individual assessment of each scapula used by McClure et al. (2009). The testing protocol required testers to assess and rate each scapula individually for winging, control when lifting, control when lowering, and overall symmetry between the two scapulae. Each component was rated from 0-3 and Table 2.7 states how this scoring system was implemented for each component. Results of this new assessment tool indicated excellent inter-tester and good to excellent intra-tester reliability, with intraclass correlation coefficient (ICC) values of 0.80 – 0.97 and 0.60 – 0.92 respectively. O'Connor et al. (2015) also calculated Cohen's kappa (κ) scores for direct comparison with Kibler et al. (2002). Results found

moderate to very good reliability ($\kappa = 0.49, 0.59$), which was better than the moderate reliability reported by Kibler et al. (2002).

Table 2.7 O'Connor et al. (2015) scoring system for the Scapular Control Test

Score	Winging	Scapular Control when Lifting/Lowering	Symmetry
No issue (0)	Medial border of the scapulae flat on the thorax	Scapulae moves in a controlled manner with no shaking or abnormal scapulae positioning	Scapulae move in synchrony throughout the full range of motion with no lagging behind or speeding of a single scapula in relation to the corresponding scapula during lifting and lowering
Slight issue (1)	Medial border visible, with slight sulcus present	Slight loss of control, not consistently present	Slight asymmetry present during lifting and/or lowering
Moderate issue (2)	Medial border visible, with moderate sulcus present	Loss of control that is consistently present (each rep), but which is regained during the movement	Moderate asymmetry present during lifting and/or lowering
Severe issue (3)	Medial border visible, with severe sulcus present	Loss of control that is consistently present (each rep) and does not regain control at any point	Severe asymmetry present during lifting and/or lowering

Only the McClure et al. (2009) SDT went through validity testing in a follow-up study which assessed the same participants (Tate et al., 2009). Tate et al. (2009) only assessed participants judged as having either normal motion or obvious dyskinesia as per the McClure et al. (2009) guidelines. These participants underwent 3-D electromagnetic kinematic testing while performing the same weighted shoulder flexion and abduction movements as the scapular dyskinesia test. A group-by-group interaction for scapular upward rotation ($p < 0.001$), clavicular elevation ($p < 0.001$), and clavicular protraction ($p = 0.044$) was noted, indicating differences between those judged as possessing normal scapular motion, with the SDT, and those judged to possess obvious dyskinesia (Tate et al., 2009). The group judged to have dyskinesia had less upward rotation at rest and remained less upwardly rotated during arm elevation, with an approximate 9° difference between the groups at rest ($p < 0.001$), at 30° ($p < 0.001$), and 60° ($p = 0.01$) of humerothoracic elevation. In relation to clavicular elevation the dyskinesia group

began with less clavicular elevation, though not significant ($p = 0.05$), and remained less elevated during raising and lowering, with post hoc differences at 30° ($p = 0.03$) and 60° ($p = 0.03$) of elevation. The differences between groups at these angles were approximately 4° . Finally, in relation to clavicular protraction, the dyskinesia group began with more protraction and remained so compared to the normal group. Differences between the groups for protraction were seen at rest ($p = 0.02$), 30° ($p = 0.03$), 60° ($p = 0.008$), 90° ($p = 0.002$), and 120° ($p = 0.03$), with maximum differences between groups of 4.5° at 90° of humerothoracic elevation.

When comparing groups during shoulder abduction, group-by-group, interactions were noted for upward rotation, clavicular elevation, and posterior tilting, with the dyskinesia group having less scapular upward rotation ($p = 0.001$), less clavicular elevation ($p = 0.03$), and greater posterior tilting ($p < 0.001$) than the group judged to have normal motion (Tate et al., 2009). Similar to that observed during weighted flexion, the dyskinesia group demonstrated less upward rotation and less clavicular elevation than the normal group during weighted abduction. Post hoc analysis showed that these differences were only significant at rest for upward rotation ($p = 0.03$) and at rest ($p = 0.005$), 30° ($p = 0.04$), and 60° ($p = 0.05$) positions for clavicular elevation. There was between 5° to 7° actual between-group differences seen at these position for upward rotation and less than 3° for clavicular elevation. The dyskinesia group began with 5° greater posterior tilting at rest, but post hoc testing did not demonstrate any significant difference at rest or at any other angles. The finding of differences between the groups judged to have normal motion or obvious dyskinesia in several kinematic descriptors demonstrates that shoulders judged as having obvious dyskinesia actually possess different kinematics. This provided evidence for the validity of the SDT proposed by McClure et al. (2009).

2.2.5 Muscle Activation Patterns of the Shoulder Complex

In order to understand how active structures control, and generate movement, the timing and sequencing of MAPs have been well researched (Falla et al., 2004; Hodges and Richardson, 1999; Lucas et al., 2010; O'Sullivan et al., 1997b). The body is often viewed by clinicians as a series of segments that link together to form a kinetic chain (Lucas et al., 2004) however, these segments work in unison in a coordinated manner to control and produce movement. With regard to the upper limb, Kibler (1998) has

suggested that where there is a deficiency in a proximal segment of the kinetic chain altered workloads may be required in more distal segments in order to preserve the same movement outcome at the most distal segment. It is therefore important, that when assessing or analysing overuse or overload injuries at distal segments of the kinetic chain, such as the GH joint, to consider the proximal segments of the kinetic chain such as the thorax and scapula. Kibler (1998) suggested that the scapula and the muscles that attach it to the vertebrae and ribs (comprising of the trapezius, serratus anterior, rhomboids, levator scapulae and pectoralis minor) serve as the segments that link the trunk to the upper limb. In order for the scapula to fulfil this role successfully, it has been proposed that it must be located in the ideal position to effectively transfer forces from the trunk to the upper limb, and thus the scapular positioning muscles must be recruited with optimal MAPs (Lucas et al., 2004).

As the GH joint progresses from a rested position to full elevation there are distinct phases where muscles play a particular role, these are approximately 0°-90°, 90°-140°, and 140°- end of range (Bagg and William, 1988). As mentioned in previous sections, upward rotation of the scapula is a crucial motion that aids the positioning of the scapula and the muscles that stabilise the GH joint, the rotator cuffs. The muscles that are responsible for upward rotation of the scapula include all parts of the trapezius and the lower part of the serratus anterior. Bagg & Forrest (1986) demonstrated that in the early stages of scapular rotation, when the scapula's instantaneous centre of rotation (ICR) is located near the root of the spine of the scapula, the upper trapezius and lower serratus anterior are strongly activated, probably in accordance with their relatively large moment arms (compared to the middle and lower pars of the trapezius) at this stage of movement. In a later study, Bagg & William (1988) demonstrated that the ICR of the scapula shifted laterally at a relatively early stage of arm movement. This change in ICR increases the mechanical advantage of the lower fibres of the trapezius, though the upper fibres of the trapezius still maintain their mechanically favourable position (Bagg and William, 1988). The most common MAPs observed, involved a gradual increase in activity of the upper trapezius and lower serratus anterior in the early stages of elevation, with the lower trapezius remaining relatively quiet until the arm approached the 90° range (Bagg and Forrest, 1986).

Bagg and William (1988) demonstrated that scapular upward rotation contributes most to elevation of the arm in the middle phase of movement (average range, 82° to 139°). Bagg & William (1988) also stated that during this phase of elevation the scapular ICR migrates from the root of the scapular spine towards the acromion. The role of upward rotation during this phase of elevation is critical as the greater tubercle of the humerus can closely approximate the inferior surface of the acromion. It is therefore a necessity that the acromion continues to be elevated in order to preserve the subacromial space (Neumann, 2002). The key components during this stage of elevation, created by upward rotation of the scapula, is the increased mechanical advantage enjoyed by the upper trapezius and the lower serratus anterior compared to the GH abductors (deltoid and supraspinatus) (Neumann, 2002), along with a gradual improvement in the lower trapezius moment arm, as the ICR migrates toward the acromion (Bagg and William, 1988).

As the arm elevates above 90° there is a rapid increase in the activity of the lower trapezius, associated with a corresponding reduction in the electrical activity of both the upper trapezius and the lower serratus anterior towards the end of the middle phase of elevation (Bagg and William, 1988). Another reason for the decreased activity of the upper trapezius and lower serratus anterior muscles may be due to the changing resistance torque of the upper extremity as it elevates above 90°. In the final stage of elevation, as the ICR approximates the acromion, the force generating capacity of the upper trapezius is greatly diminished, both due to a minimum moment arm and an unfavourable length-tension relationship (Bagg and William, 1988). Bagg & William (1988) suggested that in this phase of elevation, the upper trapezius becomes a supporter of the shoulder girdle, opposing downward acting forces produced by the weight of the upper extremity and any load held in the hand. Conversely, the altered ICR significantly improves the moment arm of the lower trapezius, while its alteration does not negatively effects the lower serratus anterior. This allows these two muscles to maintain the upward rotation of the scapula (Bagg and William, 1988).

As the scapular upward rotators stabilise and position the scapula during elevation they also maintain the position of the glenoid fossa thereby providing optimal kinematics of the humeral head by maintaining the length-tension relationships of the rotator cuff muscles (Kibler, 1998). The rotator cuff muscles (supraspinatus, infraspinatus, teres

minor and subscapularis) also act to maintain the congruence between the humeral head and the glenoid fossa by producing a compressive force during GH movements (Michener et al., 2003). This compressive force results in an inferior translation to the head of the humerus counteracting the upward vector produced by the deltoid, particularly in the early phase of elevation, helping to preserve the subacromial space. It has also been proposed that the latissimus dorsi and teres major contribute to depression of the humeral head during elevation (Halder et al., 2001). However, there has been no conclusive evidence of this and the fact that both of these muscles are prime adductors of the GH joint, it seems unlikely that they would be strongly activated during elevation of the arm.

The main question that arises when abnormal movement patterns are associated with pain is whether it is a cause or an effect. It is unclear if abnormal MAPs cause the pain or if the abnormal MAPs is a result of the pain. Abnormal movement patterns and postures are obvious to and well identified by clinicians managing patients with musculoskeletal pain (Sterling et al., 2001). However, some changes in motor function that occur in the presence of pain are less apparent (Sterling et al., 2001). Such subtle alterations in motor function require the use of EMG sensors to assess the activity of specific muscles. Motor control deficits in the form of muscle inhibition and altered patterns of muscle recruitment, resulting in the loss of joint control, have been recognised in the lumbar spine, cervical spine, and knee in studies using such techniques (Hodges and Richardson, 1996; O'Sullivan et al., 1997a; Voight and Weider, 1991). Such loss of joint control may leave individuals biomechanically vulnerable to further injury or be the cause of ongoing pain (O'Sullivan et al., 1997a).

Both increases and decreases in muscle activity have been shown under certain conditions when nociceptive input is present (Sterling et al., 2001). Various models have been proposed in an attempt to explain motor responses in the presence of pain (Sterling et al., 2001). These models include the vicious cycle and pain adaptation models (Johansson and Sojka, 1991; Lund et al., 1991). Although these models may go some way towards explaining the effect of pain on motor activity their application in clinical settings is limited due to the complexity that is now understood to be associated with pain. Pain research has now embraced the complexity of pain with the wide implementation of the biopsychosocial model in the design and interpretation of such

studies. This model aims to look beyond assessing the possible biological aspect of pain and to incorporate possible psychological and social components. Research by Moseley (2007) and others have attempted to incorporate the variance of the individual's perception of what their pain means, how this makes them alter their movement and also the social context that the individual is currently placed. The progress that has been made in pain science in recent years further highlights its complex nature and one that may assist in furthering the understanding of pain that is thought to be of a myofascial origin.

While the effects of pain on MAPs have been well established it has been proposed that LTrPs are capable of altering MAPs when pain is not present. Given that LTrPs have been proposed to induce muscle cramping (Ge et al., 2008; Xu et al., 2010), muscle fatigue (Ge et al., 2012), restrict range of motion (Grieve et al., 2011; Montanez-Aguilera et al., 2009; Simons et al., 1999; Trampas et al., 2010), and cause muscle weakness (Ge and Arendt-Nielsen, 2011; Ge et al., 2012; Simons et al., 1999) it's plausible that they would therefore have an effect on MAPs. If the presence of LTrPs is demonstrated to cause alterations in MAPs, then effective treatment of these lesions would be of benefit in the potential prevention of common shoulder pathologies.

2.2.6 Measurement of Muscle Activation Patterns

Surface electromyography (sEMG) is used to measure MAPs and it has been utilised in a number of previous studies to investigate muscles of the shoulder complex (Christensen, 1986; Ebaugh et al., 2005; Elert et al., 2000; Lucas et al., 2004). This has been due to a combination of factors, better understanding of the physiological processes that contribute to the generation of the signal, more adequate signal processing techniques and a growing knowledge on how it can be applied in various clinical applications (Hermens et al., 2000).

Wadsworth & Bullock-Saxton (1997) and Lucas et al. (2010, 2004) have both investigated the time of onset of scapular rotators with the use of sEMG. These muscles are thought to be key to the optimal positioning of the scapula. Wadsworth & Bullock-Saxton (1997) investigated male swimmers with unilateral shoulder impingement syndrome during scapular plane elevation and observed that the timing of muscle activation was more variable in subjects with the condition compared to matched controls (Wadsworth and

Bullock-Saxton, 1997). Lucas et al. (2010, 2004) investigated the effects of LTrPs on MAPs of scapular positioning muscles during loaded scapular plane elevation. Their main findings were that the control group displayed a relatively stable sequence of muscle activation that was significantly different in timing and variability to that of the LTrP group (Lucas et al., 2010, 2004).

2.2.6.1 Surface EMG Placement

Due to the fact sEMG electrodes are the listening devices for picking up sEMG activity, knowing where to place the electrodes is a critical part of the process. Due to the increased use of sEMG both in research and clinically there has been huge variation in methodologies among different groups of users (Hermens et al., 2000). It was because of these variations and the possibility of it hindering the future growth and application of SEMG that Hermens et al. (2000) developed recommendations for sEMG sensors and sensor placement procedures by setting up the European concerted action surface EMG for a non-invasive assessment of muscles (SENIAM) in 1997. By standardising sEMG methodology the group hoped to make results more comparable and create a large common body of knowledge on the use of SEMG in various fields of application. Besides their general goal of creating more collaboration among the various European groups, the specific goal of the SENIAM was to develop recommendations on key items to enable a more useful exchange of data obtained with sEMG, including sensors, sensor placement, signal processing, and modelling.

Unfortunately, the SENIAM group do not currently have a comprehensive list of sensor placements for every muscle involved in controlling motion of the scapula or stabilising the GH joint. Currently there are recommendations for the trapezius and deltoid muscles only however, Cram & Criswell (2011) have produced an extensive list of sensor placement positions. A list of the recommended sensor placement sites for muscles pertaining to scapular motion and GH stabilisation can be found in the appendices (Appendix F). The placement recommendation by Cram & Criswell (2011) have been used in similar research conducted by Lucas et al. (2004). Cram & Criswell (2011) followed a number of criteria highlighted by Fridlund & Cacioppo (1986) to improve the fidelity of sEMG recordings (Table 2.8).

Table 2.8 Fidelity of sEMG recordings

Fidelity of sEMG recordings
Select the appropriate proximity of a proposed site to the underlying muscle mass, keeping the minimum amount of tissue between the electrodes and the muscle fibres themselves
Select the appropriate position of the electrodes relative to the muscle fibres. Whenever possible, the electrodes should be placed parallel to the fibres to maximise sensitivity and selectivity. Perpendicular placements tend to lead to greater common mode rejection and less selectivity
Avoid straddling the motor endplate region. If this is done, the amplitudes observed are typically lower owing to differential amplification. Placing electrodes a little off the centre of the muscle is better positioning
Choose sites that are easy to locate (sites that have good anatomical landmarks to facilitate reliable placement of electrodes during subsequent recording sessions)
Choose site that do not unduly obstruct vision or movement. Avoid areas that present problems owing to skin folds, bony obstruction, and other factors
Minimise cross-talk from proximal, deep or superficial muscles by selecting the best electrode size and interelectrode spacing

2.2.7 Scapular Dyskinesis or Natural Variation

This section will examine information which challenges current beliefs on shoulder stabilisation and scapular dyskinesis. Conservative treatment of most musculoskeletal injuries typically involves the implementation of an exercise based programme to improve the strength and/or tolerance of a tissue or to improve the ability of functionally related body segments to perform a desired task. The inclusion of stabilisation exercises have been a focus of such programmes in a bid to improve patients' ability to return to sport or activities of daily living. Typical examples of this have been the implementation of core stabilisation programmes for patients with low back pain (LBP) and scapula stabilisation programmes for patients with shoulder pain. While these programmes are well accepted and implemented by clinicians, there is a lack of research providing evidence for the practice. The use of stabilisation programmes in those with LBP became common following studies by Hodges and Richardson (1999, 1996) which suggested that those with LBP possessed a delayed activation of the transversus abdominis muscle compared to healthy people. From this came the advice that an exercise programme aimed at restoring this abnormality would resolve LBP. The rationale behind the use of core stability programmes was that this was a key muscle for stabilising the spine and that training it would have a positive effect on patients with LBP. However, a systematic review carried out by May & Johnson (2008) on stabilisation

exercises for LBP concluded that core stability exercises are unlikely to produce better outcomes over other forms of exercise. A similar rationale has been applied to patients with shoulder pain and the role of scapular dyskinesis, and in recent years the notion that an unstable scapula is related to shoulder dysfunction and pathology is well accepted (Kibler et al., 2009).

A recent publication by McQuade et al. (2016) took a critical perspective on scapular stabilisation and scapular dyskinesis, challenging the concept by applying biomechanical and motor control constructs. McQuade et al. (2016) questioned the idea that scapular dyskinesis is a sign of instability and that it is a result of weak or unbalanced ST muscles. They also questioned the evidence of training efforts to improve scapular stabilisation (McQuade et al., 2016). The premise that scapular dyskinesis is a sign of instability was first challenged based on the foundational definition of stability, which Reeves et al. (2007) defined as the degree to which a system can return to an orientation or movement trajectory following a perturbation. This definition is difficult to apply directly to the scapula, as in a normally functioning neuromuscular system the scapula does not go beyond its physiological limits (McQuade et al., 2016). McQuade et al. (2016) propose that the use of such terms influences how practitioners approach patient care, in that dyskinesis is a sign of muscle weakness, instability, and/or lack of motor control, and that stabilisation exercises will resolve symptoms and improve scapula motion. In fact, McQuade et al. (2016) stated that clinically the observation of “abnormal” motion should be described but not assumed to represent an “unstable scapula”, because the observed “abnormal” motion may simply represent normal kinematic variability.

Training programmes targeted at restoring what is considered normal scapular motion often focus on specific exercises for targeted muscles based on the belief that muscular imbalance is the cause (Cools et al., 2007). Like many areas of the body, these exercises are based on EMG studies which have found lower EMG responses in key muscles. However, because the scapula both translates and rotates in three dimensions, its ICR is constantly changing which effects the moment that each scapular muscle can generate to rotate the scapula (McQuade et al., 2016). This change in ICR has been detailed in section 2.2.5, where the ICR is located near the root of the spine of the scapula at rest and migrates laterally towards the acromion as GH elevation occurs. Thus, such exercises targeted at specific muscles may not replicate the functional role of the

muscle. This challenges clinical reasoning, where clinicians may tend to use scapular dyskinesis tests to assess for the presence of dyskinesis and initiate an exercises programme to resolve it. However, many individuals with scapular dyskinesis maintain healthy functional use of their upper limb despite its presence (McQuade et al., 2016).

As a result, McQuade et al. (2016) challenged the role of the scapula as a stable base of support. As the scapula has minimal anatomical constraints as it sits on the thorax suspended primarily by the musculotendinous attachments of 17 muscles, along with its direct attachment to the clavicle, this necessitates that forces generated in the arm must transfer to the axial skeleton primarily through the musculotendinous attachments (McQuade et al., 2016). They therefore put forward a model for load transfer suggesting that the scapula functions as the hub of a tensegrity structure (Levin, 2005), where forces coming from the arm are transferred to the axial skeleton through the soft tissues rather than the linked bond levers (McQuade et al., 2016). McQuade et al. (2016) used the analogy of the design of a bicycle wheel, the scapula, suspended in the “spokes” of the attached muscles and soft tissue, functioning as a hub for the arm and thorax. They postulate that the concept of the scapula supported within a musculoskeletal sling that can transfer forces from proximal to distal or distal to proximal is also plausible, and from this conceptualise that the ST joint functions as an energy transfer system rather than an anatomical structural base of support (McQuade et al., 2016). This alternative way of looking at the role of the scapula states that the role of the scapula is to maximise the overall degrees of freedom needed for placing the hand in space and to absorb the transfer of energy to and from the upper extremity (McQuade et al., 2016).

Viewing the role of the scapula in this way puts less importance on the stability of the scapula and the presence of scapula dyskinesis. This is also in line with suggestions made by Hasan (2005), that stability in the sense of quick resistance to perturbation often may not be necessary for successful control of forces, energy and movement (McQuade et al., 2016). Hasan (2005) instead proposed that movement variability creates resilience, which is more desirable than stability for the control of movement. McQuade et al. (2016) proposed that most observed scapula dyskinesis likely represents normal movement variability. They state that all human movement bears some form of individualisation, such as the variability in gait patterns between individuals. However, this acceptance of individual variability has not been extended to the scapula, where all

abnormal movement is identified as potentially problematic (McQuade et al., 2016). Bernstein's theory of movement behaviour states that movements can have different trajectories, velocities, and muscle activation profiles to achieve the same end result (Bongaardt and Meijer, 2000). Again, this theoretical model would question the relevance of scapular dyskinesis and any observations in altered MAPs. Furthering this model is the likelihood that the position of the scapula and its stabilisation would be a low priority for the central nervous system (CNS), it instead focusing on the hand and how it interacts with the environment to complete the desired task (McQuade et al., 2016). The scapula therefore would simply be a subservient to the hand and exercise programmes which place an emphasis on correcting scapula movement would be unlikely to have much carryover to daily tasks. McQuade et al. (2016) stated that the term robustness may be more appropriate than stability as it describes a system's tolerance for uncertainty, allowing for degrees of movement variability.

With the research that has been conducted on the shoulder in recent decades the theoretical connection between dyskinesis, scapula instability, and shoulder pathology has been widely accepted however, there is still a dearth of evidence that clarifies such a connection (McQuade et al., 2016). Specifically in relation to subacromial impingement syndrome, there has been a lack of a consistent connection between scapular dyskinesis and the pathology. A recent systematic review investigating the relationship between subacromial impingement syndrome and scapular position by Ratcliffe et al. (2014) found that no ideal scapula position exists and that deviations in scapular motion does not cause or contribute to subacromial impingement. This lack of clarity may pose three potential conclusions: that scapular dyskinesis could develop in response to pain rather than cause it; that the tests are not sensitive enough to clarify a relationship; that high variability in scapular motion is normal.

2.3 Conclusion

Following a review of the literature on both areas pertinent to this research, TrPs and the shoulder complex, it can be concluded that there is a clear requirement for further research in these areas. There is still uncertainty around the proposed hypotheses of TrPs and due to that uncertainty there have been challenges to the treatment interventions based on them. The current research on TrP treatment interventions demonstrates mixed results for their efficacy and a need for higher quality research.

Recent research on the latent form of TrPs has proposed that they cause clinical implications prior to their potential conversion into the active form. These proposed clinical implications of LTrPs offer researchers an opportunity to investigate the efficacy of the various treatment interventions, without pain potentially confounding results. This research aims to investigate the efficacy of LTrP dry needling at changing MAPs of key muscles in the shoulder complex, compared to manual release and placebo dry needling.

The review of the literature on the shoulder complex, and specifically scapular dyskinesis and MAPs, demonstrated that there are also points of contention in this area. There is uncertainty around the role of the scapular in functional movements and whether scapular dyskinesis is a cause or an effect of shoulder pathologies. As this research will collect MAP data on healthy individual without any shoulder pathology but with LTrPs present, it will gain an insight into the MAPs of healthy individuals with a potential abnormality and whether their MAPs change following treatment.

2.4 Summary

Results from this review of the literature suggest that treatment options for TrPs require further investigation, due to the uncertainties surrounding the proposed TrP hypotheses and the inconsistencies observed in the treatment studies. Recent proposals that LTrPs cause clinical implications in pain-free individuals provides an opportunity to investigate the efficacy of these treatment options. The aim of this research was to investigate the efficacy of two of the most widely used treatment interventions at altering MAPs in pain-free participants with LTrPs, and to include a clinically applicable outcome measure.

Chapter 3: Reliability and Validity of Two Clinical Scapular Dyskinesis Tests

3 Reliability and Validity of Two Clinical Scapular Dyskinesis Tests

3.1 Abstract

Background: Scapular dyskinesia is a term given to visible alterations in the scapula's position and motion; such alterations have been associated with shoulder injury (Tate et al., 2009). Previous research has correlated abnormalities in scapula positioning and motion with impingement symptoms, rotator cuff dysfunction, and instability (Kibler et al., 2013). Due to these associations a number of classification systems have been developed to assess scapular dyskinesia. While all developed scapular dyskinesia tests have been assessed for reliability, few have been assessed for both inter- and intra-tester reliability. Similarly, there is a dearth of research assessing the validity of these tests. As scapular dyskinesia is thought to be a result of abnormal MAPs there is a need to assess these tests to ascertain whether they are capable of identifying alterations in the MAPs of key periscapular muscles.

Aim: To establish the inter- and intra-tester reliability of the scapular dyskinesia test (SDT) and the scapular control test (SCT), and to establish their validity in assessing altered MAPs.

Methods: Thirty participants were videotaped and had sEMG data collected when performing the SDT and the SCT. Participants were assessed by two testers, both were examined for inter-tester reliability and one (the principal investigator) was assessed for intra-tester reliability. Participants were grouped into a normal and dyskinesia group based on the principal investigator's assessment. The validity of the two tests was then assessed by comparing the sEMG data of both groups.

Results: Both the SDT and the SCT demonstrated good to excellent inter- and intra-tester reliability, however there was no statistical difference in the MAPs between groups when assessing their validity.

Conclusion: While both tests demonstrated high inter- and intra-tester reliability neither proved to be valid at determining a difference in MAPs between groups. The results indicated that the use of these tests in a clinical setting to determine abnormalities in MAPs may be unwarranted.

3.2 Introduction

Shoulder pain and dysfunction are common complaints of those that seek care from clinicians working in the musculoskeletal domain. In cases where pain is not caused by a traumatic event or joint instability, the role of the scapula has received increased attention as a predisposing factor in the last decade or more. A number of studies have correlated abnormalities in scapula positioning and motion with impingement symptoms, rotator cuff dysfunction, and instability (Cools et al., 2007; Hallström and Kärrholm, 2006; Ludewig and Cook, 2000; Lukasiewicz et al., 1999; McClure et al., 2006; Warner et al., 1992). Scapular dyskinesis is the term given to visible alterations in scapular position and motion patterns and correcting these alterations has been a key focus for clinicians treating those with shoulder associated pathologies (Kibler et al., 2013).

The gold standard method for assessing altered scapular kinematics is the use of 3-D electromagnetic motion sensors, as utilised by a number of previous studies (Lawrence et al., 2014a, 2014b; Tate et al., 2009; Uhl et al., 2009), which have demonstrated that patients with shoulder pain possess altered kinematics in all joints involved in glenohumeral elevation. However, this type of scapular kinematic assessment involves the use of sophisticated technology which is impractical for clinical use, due to its invasive nature and expense. Therefore, in an attempt to provide clinicians with a means of assessing patients for scapular dyskinesis, a number of visual classification systems have been developed (Ellenbecker et al., 2012; Kibler et al., 2002; McClure et al., 2009; O'Connor et al., 2015; Uhl et al., 2009). Of these classification systems only the McClure et al. (2009) SDT has been validated using 3-D electromagnetic motion tracking (Tate et al., 2009).

Tate et al. (2009) demonstrated that those noted as having obvious dyskinesis, possessed different kinematics than those determined to have normal scapular motion, providing evidence for the validity of the SDT. Such information is of great benefit to clinicians designing injury prevention programmes for those with asymptomatic scapular dyskinesis and rehabilitation programmes for patients with pain that is associated with scapular dyskinesis. However, kinematic data can only infer as to how the muscles that control scapular motion are working. The scapula has minimal anatomical constraints and sits on the thorax suspended primarily by the

musculotendinous attachment of seventeen muscles along with some load transfer via the clavicle (McQuade et al., 2016). Therefore further investigation is necessary to determine whether those judged to possess scapular dyskinesis in turn demonstrate altered MAPs. Such information would also provide clinicians with invaluable information to direct them in implementing exercise based preventative and/or rehabilitation protocols.

The McClure et al. (2009) SDT and the O'Connor et al. (2015) SCT are two of the previously published clinical tests. The SDT involves participants performing bilateral overhead elevation in both the sagittal (forward flexion) and frontal (abduction) planes, while the O'Connor et al. (2015) SCT only involves bilateral elevation in the sagittal plane (abduction). The SDT and the SCT have demonstrated the best inter-tester reliability of the available tests (Kibler et al. 2002; Uhl et al. 2009; McClure et al. 2009; Ellenbecker et al. 2012; O'Connor et al. 2015) and the SCT demonstrated considerably better intra-tester reliability when compared to the only other study assessing it (Kibler et al., 2002). McClure et al. (2009) reported a κ_w score ranging from 0.48 to 0.61 for the SDT and, as mentioned previously, has been shown to be a valid measure of altered scapular kinematics (Tate et al., 2009). While the O'Connor et al. (2015) SCT has not been validated, it has achieved the highest inter-tester and intra-tester reliability of all published classification systems, with an ICC score ranging from 0.80 to 1.00 for inter-tester reliability and 0.60 to 1.00 for intra-tester reliability. The aim of this pilot study was to investigate the inter- and intra-tester reliability of both tests and establish whether either were valid measures for identifying asymptomatic subjects with altered MAPs in key muscles of the shoulder complex, with the use of sEMG sensors.

3.3 Methodology

3.3.1 Participants

Thirty healthy males were recruited from a convenience sample of collegiate staff, students and the general public (Table 3.1). Only males were recruited for this study to allow for the required unobstructed view of the skin overlaying the serratus anterior muscle during testing. To determine the study sample size, a priori power analysis was performed to provide a statistical power of 80% at an α level of 0.05. Equation 3.1 was implemented using previous data (Lucas et al., 2010) with a standard deviation (s) value of 0.11 seconds and a difference to be detected (d) value of 0.1 seconds, where time values represent the onset of muscle activation. From this calculation a sample size of 19 participants was determined to be sufficient, however a higher sample size of 30 participants was decided upon to allow for potential missing sEMG data. Collegiate staff and students were recruited by email (Appendix A), while the general public were recruited by word of mouth. All participants satisfied the inclusion and exclusion criteria outlined in Table 3.2. Ethical approval for this study was obtained from AIT's research ethics committee.

$$n = 16 \times \frac{s^2}{d^2}$$

Equation 3.1 Priori power analysis (s = standard deviation; d = difference to be detected)

Table 3.1 Pilot study: participant demographics (mean \pm SD)

Participant Demographics	(n = 30)
Age (y)	25 \pm 5
Hand Dominance	29 Right/1 Left
Height (m)	1.78 \pm 0.06
Weight (kg)	80.9 \pm 11.5
Body Mass Index (kg/m ²)	25.4 \pm 3.0
Left Overhead Elevation ($^{\circ}$)	168 \pm 7
Right Overhead Elevation ($^{\circ}$)	168 \pm 7
Left External Rotation 0 $^{\circ}$ ($^{\circ}$)	69 \pm 15
Right External Rotation 0 $^{\circ}$ ($^{\circ}$)	67 \pm 13
Left External Rotation 90 $^{\circ}$ ($^{\circ}$)	102 \pm 13
Right External Rotation 90 $^{\circ}$ ($^{\circ}$)	101 \pm 13
Left Internal Rotation (T-Spine)	4 \pm 2
Right Internal Rotation (T-Spine)	4 \pm 2
Left Horizontal Adduction (cm)	24 \pm 4
Right Horizontal Adduction (cm)	24 \pm 4

Table 3.2 Pilot study inclusion & exclusion criteria

Inclusion Criteria
Male
Aged 18-45 years
No upper limb, back, or neck pain 7 days prior to testing
Exclusion Criteria
Any pain in the back, neck, or upper limb 7 days prior to testing
Significantly increased thoracic kyphosis (excessive curvature of the thoracic spine)
History of any injuries to the neck or upper limb
History of any nerve injuries in the cervical or shoulder area
Any allergies to adhesives
Body mass index (BMI) of ≥ 30
<160 $^{\circ}$ arm elevation
Tenderness on the greater tuberosity of the humerus, AC joint
Positive impingement tests

3.3.2 Study Design

The layout of this study is illustrated in Figure 3.1.

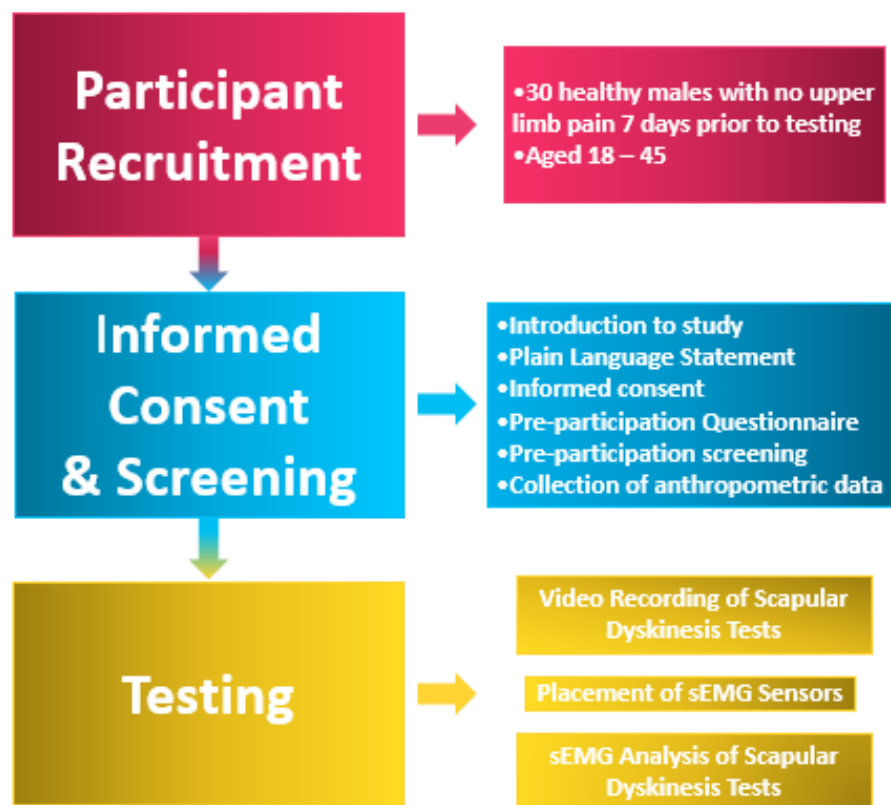


Figure 3.1 Pilot study layout

3.3.2.1 Pre-participation Screening

All testing was completed in the athletic therapy room. The purpose of the study, the testing procedure, participants' requirements, and the risks and benefits associated with the study were explained to all volunteers. Participants were given a plain language statement outlining these details and were given sufficient time to review it (Appendix B). After the dissemination of this information, participants were given the opportunity to ask any questions they may have had at this point. Volunteers willing to participate were then asked to sign an informed consent form prior to their progression to the screening component of the session (Appendix C). For those who required more time to consider their participation a screening session was organised at a later date.

Upon completion of the informed consent form, participants completed a pre-participation questionnaire (Appendix D). This questionnaire collected key data required for the study which included, date of birth, ethnicity, recent back, neck or upper limb pain, history of previous injuries, and allergies to adhesives. Participants were given a participant ID number, which was used for all further data collection to ensure anonymity and confidentiality. After participants completed the pre-participation

questionnaire it was reviewed by the principal investigator to determine if the participant satisfied the inclusion and exclusion criteria. Participants who did not satisfy all criteria to this point were excluded from the study, while compliant participants underwent a pre-participation screening. This screening involved the completion of components of a shoulder assessment form, developed by the American Shoulder and Elbow Surgeons (Richards et al., 1994) (Appendix E). This assessment collected data on each participants shoulder range of motion, signs of symptoms of shoulder pathology, strength and instability. Participants were required to remove their shirts to allow for identification of bony landmarks and to allow for unobstructed movement. Participants who did not comply with the criteria after the pre-participation screening were excluded from the study. Upon successful completion of the pre-participation screening, anthropometric data was collected, weight was measured in kilograms (kg), to the nearest 0.1 kg, using a Seca 761 scales (Seca, Birmingham, United Kingdom), and height was measured in metres (m), to the nearest 0.01 m, using a Seca 213 height measure (Seca, Birmingham, United Kingdom). Participants who satisfied all inclusion and exclusion criteria progressed to the scapular dyskinesis tests and the sEMG analysis.

3.3.2.2 Scapular Dyskinesis Tests

Participants completed two scapular dyskinesis tests, the SDT developed by McClure et al. (2009) and the SCT developed by O'Connor et al. (2015). For both tests, participants were instructed to stand assuming a normal resting posture. Participants were positioned approximately 2 metres directly in front of a video camera (model HC-V100, Panasonic Corporations, Osaka, Japan) that was mounted level on a tripod, approximately 1.5 metres from the floor. Participants were required to remain topless to allow for an unobstructed view of their posterior thorax. Participants were positioned with their backs to the camera so that they could not be identified on the video recording. Participants were then required to elevate their hands as detailed by each scapular dyskinesis test. Details of the SDT are shown in Table 3.3 and of the SCT in Table 3.4, while the tests are depicted in Figure 3.4, Figure 3.5 and Figure 3.6.

All participants performed the SDT first followed by the SCT. The recorded video clips were viewed and analysed by two testers, as per the McClure et al. (2009) and O'Connor et al. (2015) guidelines. The SDT uses a three point ordinal scale (normal motion, subtle dyskinesis, and obvious dyskinesis) while the SCT utilises a four point ordinal scale (no

issue, slight issue, moderate issue, and severe issue) and a summary of the rating guidelines are displayed in Table 3.5 and Table 3.6. Both testers were certified athletic and rehabilitation therapists, with 3 and 5 years clinical experience and underwent the same standardised training prior to analysing the video footage (see section 3.4.1).

Table 3.3 Protocol instructions for the Scapular Dyskinesia Test

McClure et al. (2009) SDT

1. Male participants are asked to remove their shirts to allow unobstructed observation of the posterior thorax
2. Participants place their arms at the side of the body, elbows straight, and shoulder in neutral rotation.
3. Participants are asked to simultaneously elevate their arms overhead as far as possible to a 4-second count using the “thumbs up” position and then lower to a 4-second count.
4. Planes of elevation:
 - a. 5 repetitions of bilateral, active, weighted shoulder flexion
 - b. 5 repetitions of bilateral, active, weighted shoulder abduction (frontal plane)
5. Weights used:
 - a. 1.4 kg (3lb) for those weighing < 68.1 kg (150 lb)
 - b. 2.3 kg (5 lb) for those weighing > 68.1 kg (150 lb)

Table 3.4 Protocol instructions for the Scapular Control Test

O'Connor et al. (2015) SCT

1. Male participants are asked to remove their shirts to allow unobstructed observation of the posterior thorax
2. Participants place their arms by their sides with palms facing their thighs
3. Participants are asked to simultaneously elevate their arms to 180° to a 4-second count and then lower them to a 4-second count
4. Planes of elevation:
 - 3 repetitions of bilateral, active, unweighted shoulder abduction (frontal plane)

Table 3.5 Operational definitions & rating scale for the Scapular Dyskinesis Test

Scapular Dyskinesis Test Rating Guidelines
<p>Operational Definitions</p> <p>Normal scapulohumeral rhythm: The scapula is stable with minimal motion during the initial 30° to 60° of humerothoracic elevation, then smoothly and continuously rotates upward during elevation and smoothly and continuously rotates downward during humeral lowering. No evidence of winging is present.</p> <p>Scapular dyskinesis: Either or both of the following motions may be present.</p> <p>Dysrhythmia: The scapula demonstrates premature or excessive elevation or protraction, non-smooth or stuttering motion during arm elevation or lowering, or rapid downward rotation humeral lowering.</p> <p>Winging: The medial border and/or inferior angle of the scapula are posteriorly displaced away from the posterior thorax.</p>
<p>Rating Scale</p> <p>Each test movement (flexion and abduction) is rated as:</p> <ol style="list-style-type: none">Normal motion: no evidence of abnormality.Subtle abnormality: mild or questionable evidence of abnormality, not consistently present.Obvious abnormality: striking, clearly apparent abnormality, evident on at least 3/5 trials (dysrhythmia or winging of 1 inch (2.54 cm) or greater displacement of the scapula from the thorax. <p>Final rating is based on combined flexion and abduction test movements.</p> <p>Normal motion: Both test motions are rated as normal or one motion is rated as normal and the other as subtle abnormality.</p> <p>Subtle abnormality: Both flexion and abduction are rated as having subtle abnormalities.</p> <p>Obvious abnormality: Either flexion or abduction is rated as having obvious abnormality.</p>

Table 3.6 Scoring system for the Scapular Control Test

Scapular Control Test Rating Guidelines			
Score	Winging	Scapular Control when lifting/lowering	Symmetry
No issue (0)	Medial border of the scapulae flat on the thorax	Scapulae moves in a controlled manner with no shanking or abnormal scapulae positioning	Scapulae move in synchrony throughout the full range of motion with no lagging behind or speeding of a single scapula in relation to the corresponding scapula during lifting and lowering
Slight issue (1)	Medial border visible, with slight sulcus present	Slight loss of control, not consistently present	Slight asymmetry present during lifting and/or lowering
Moderate issue (2)	Medial border visible, with moderate sulcus present	Loss of control that is consistently present (each rep), but which is regained during the movement	Moderate asymmetry present during lifting and/or lowering
Severe issue (3)	Medial border visible, with severe sulcus present	Loss of control that is consistently present (each rep) and does not regain control at any point	Severe asymmetry present during lifting and/or lowering

3.3.2.3 Surface Electromyography and Accelerometer Data Collection

After the scapular dyskinesis tests, participants were fitted with 10 sEMG sensors on specific scapular and GH muscles (Figure 3.2). This was performed separately from the video recording of the scapular dyskinesis test as the sensors would have caused a partial obstruction when viewing motion of the scapula. Prior to the placement of sEMG sensors participants had their skin prepared in order to provide good sensor to skin contact. This was required to obtain good sEMG signals and to limit artefacts in sEMG data. All locations were first assessed for debris and hair, if necessary the location was shaved with a disposable razor prior to cleaning the skin with an alcohol wipe. The muscles chosen for analysis were upward scapular rotators, a muscle of the rotator cuff group, and a prime mover of the GH joint. The upper and lower fibres of the trapezius muscle along with the lower fibres of the serratus anterior muscle were chosen for their role in scapular upward rotation. The infraspinatus muscle was chosen as the only rotator cuff muscle that was accessible for sEMG analysis. The middle deltoid was chosen for its role as a prime mover of the GH joint. Trigno wireless EMG sensors (Delsys Inc., Boston, MA, United States of America) were attached with double sided tape (Figure 3.3) according to methods described by Cram & Criswell (2011) (Appendix F). The

Trigno wireless EMG sensors are capable of recording both sEMG signals and tri-axial accelerometer data. Eight of the sensors recorded raw sEMG signals only (upper and lower trapezius, infraspinatus, and serratus anterior on the non-dominant and dominant sides) at 1926 samples/s. The two sensors on the middle deltoid muscles were selected as a dual sensors which recorded both sEMG signals (1926 samples/s) and accelerometer signals (148 samples/s). The accelerometer data collected from these sensors was used to identify when movement of the GH joints were initiated. This allowed the time at the onset of muscle activity to be normalised to the start of movement. Once the sensors were in place participants repeated the SDT followed by the SCT to capture their MAPs during each test. On completion of the scapular dyskinesis tests the sEMG sensors were removed.



Figure 3.2 *Sensor placement (sensors on the serratus anterior muscles not visible)*



Figure 3.3 *Delsys Trigno Wireless EMG sensors and adhesive tape*

3.4 Data Analysis

3.4.1 Video Analysis

Both testers underwent standardised training via two self-directed slide presentations on each scapular dyskinesis test, as used by McClure et al. (2009) and O'Connor et al. (2015). Both presentations detail the motion that is available at the ST joint and what is considered normal or abnormal. The presentations, each taking approximately 30 minutes to complete, included operational definitions pertinent to each test, along with photographs and embedded videos providing examples. As part of the presentation, testers were required to evaluate sample participants and submit their answers for assessment. This ensured that after completing the self-directed presentations that they were interpreting the information correctly. On successful completion of the self-directed presentations each tester viewed the recorded videos of all participants and independently rated the test movement for each shoulder according to each test (Figure 3.4, Figure 3.5, and Figure 3.6). The principal investigator, one of the two testers, also rated all videos one week after their initial assessment to determine intra-tester reliability.



Figure 3.4 McClure et al. (2009) Scapular Dyskinesis Test: flexion component

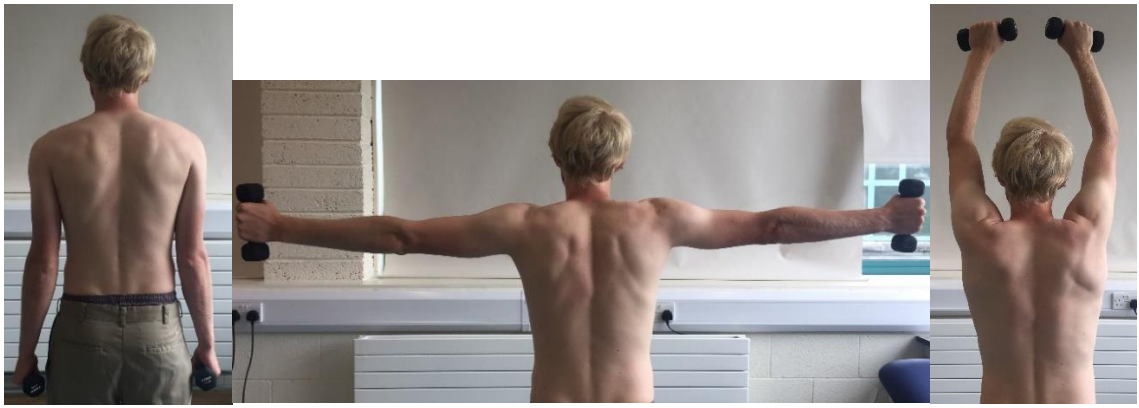


Figure 3.5 McClure et al. (2009) Scapular Dyskinesia Test: abduction component



Figure 3.6 O'Connor et al. (2015) Scapular Control Test

3.4.2 Surface Electromyography and Accelerometer Data Analysis

The raw sEMG signal from each muscle was collected using EMG acquisition software (EMGworks acquisition, Delsys Inc., Boston, MA, United States of America). The raw sEMG files were exported as text files and imported to data analysis software (LabChart 8, ADInstruments, Castle Hill, New South Wales, Australia). The sEMG signal was filtered (low pass = 500 Hz, high pass = 10 Hz), rectified, and smoothed using a root mean squared (RMS) calculation. The standard deviation of the smoothed RMS values was then calculated. These values were used to calculate the detection thresholds for muscle activation, which was set at 2 standard deviations (Hodges and Bui, 1996). A find loop was used to identify the first smoothed RMS value that exceeded the RMS detection threshold (onset of muscle activation). Following this, the first smoothed RMS value less than the smoothed RMS threshold was located. Once this point was identified muscle activation was considered to have returned to a resting level and a RMS value that exceeded the RMS detection threshold was searched for again (Hodges and Bui, 1996). This process was repeated for each muscle.

The accelerometer data was also collected using the EMG acquisition software (EMGworks acquisition, Delsys Inc., Boston, MA, United States of America) and exported in the same way to data analysis software (LabChart 8, ADInstruments, Castle Hill, New South Wales, Australia). To identify the point at which movement of the GH joint occurred, the x-axis accelerometer data was examined and a threshold was identified for each file where it exceeded the resting value. The time of initiation of movement was recorded for each rep and this was then used to normalise the onset of muscle activation.

The data collected from the sEMG analysis was then used to determine whether the SCT and the SDT were valid measures of scapular dyskinesis. Participants were grouped into a normal group or a dyskinesis group, based on the results of the principal investigators assessment, to determine whether there was a significant difference in onset of muscle activation (MAPs) between groups. This assessment was completed for both the dominant and non-dominant limbs. For the SCT, participants were grouped in the normal group if they scored 0 (normal) in all components of the test and the dyskinesis group if they scored a 1 (slight), 2 (moderate), or 3 (severe) for any component of the test. For the SDT, participants were grouped in the normal group if they scored normal and the dyskinesis group if they scored either subtle dyskinesis or obvious dyskinesis.

3.4.3 Statistical Analysis

All statistical analysis, excluding the manual calculations mentioned below, were performed using IBM SPSS statistics version 22 (IBM, New York, United States of America). To assess inter- and intra-tester reliability ICC and 95% CI were computed. A two-way random effects model with absolute agreement was used to calculate ICC and was classified according to Fleiss (1999) (Table 3.7).

Table 3.7 Fleiss (1999) intraclass correlation coefficient classification

Classification	ICC
Poor	< 0.40
Good	0.40 - 0.75
Excellent	> 0.75

To assess validity independent samples t-tests were then performed. The mean onset of muscle activation of each muscle was used to determine if there was a significant difference between those determined to possess normal scapular motion and those

with scapular dyskinesis. Prior to interpretation of the independent samples t-tests all data was assessed for the assumptions of normality, via Shapiro-Wilk's test, histograms, normal Q-Q plots and detrended normal Q-Q plots, and for homogeneity of variance, via Levene's test for equality of variance. Effect size was calculated using Cohen's d to indicate the relative magnitude of difference between the means (Cohen, 1988) (Equation 3.2). The Cohen (1988) criteria was then used to determine the classification of effect size (Table 3.8).

$$d = \frac{|\bar{x}_1 - \bar{x}_2|}{SD_{average}}$$

Equation 3.2 Effect size calculation for Cohen's d

Table 3.8 Cohen's d effect size criteria

Effect Size	d
Trivial	< 0.20
Small	> 0.20
Medium	> 0.50
Large	> 0.80

3.5 Results

Table 3.9 and Table 3.10 below illustrate the number of participants that fell into each category of the SDT and the SCT, based on the principal investigator's first assessment.

Table 3.9 Scapular Dyskinesia Test: Number of participants per category

Rating	Non-Dominant			Dominant		
	N	S	O	N	S	O
Flexion	8	9	13	14	8	8
Abduction	18	9	3	23	6	1
Final	13	4	13	18	4	8

N = Normal; S = Subtle dyskinesia; O = Obvious dyskinesia

Table 3.10 Scapular Control Test: Number of participants per category

Score	Non-Dominant				Dominant			
	0	1	2	3	0	1	2	3
Winging	25	3	2	0	25	4	1	0
Control of the Scapula Lifting	30	0	0	0	28	2	0	0
Control of the Scapula Lowering	20	7	3	0	20	8	2	0
Score	0		1		2		3	
Symmetry between both Scapulae	23		7		0		0	

0 = No issue; 1 = Slight issue; 2 = Moderate issue; 3 = Severe issue

3.5.1 Inter-tester and Intra-tester Reliability

ICC values indicated excellent inter-tester and intra-tester reliability of the SDT (Table 3.11). The inter-tester reliability of the SDT had excellent ICC scores and narrow 95% CI ranges on both the dominant and non-dominant sides (dominant side ICC = 0.97 [95% CI: 0.93, 0.99], non-dominant side ICC = 0.89 [95% CI: 0.77, 0.95]).

Intra-tester reliability of the SDT also demonstrated excellent ICC scores and narrow 95% CI ranges for both the dominant and non-dominant sides, ICC = 0.97 [95% CI: 0.93, 0.99] and ICC = 0.96 [95% CI 0.92, 0.98] respectively.

Table 3.11 ICC Inter-tester & intra-tester reliability of the Scapular Dyskinesis Test

Scapular Dyskinesis Test	Inter-tester Reliability		Intra-tester Reliability	
	ICC	ICC 95% CI	ICC	ICC 95% CI
Dominant	0.97*	0.93, 0.99	0.97*	0.93, 0.99
Non-dominant	0.89*	0.77, 0.95	0.96*	0.92, 0.98

All measures were ordinal data: 1 = normal, 2 = subtle, 3 = obvious

ICC classification: Poor (<0.40) = ®, Good (0.40-0.75) = #, Excellent (>0.75) = *

The ICC values for the SCT indicated good to excellent inter-tester and excellent intra-tester reliability (Table 3.12). Both dominant and non-dominant winging demonstrated excellent inter-tester ICC reliability, while the dominant side had a narrower 95% CI range compared to the non-dominant. Control of the scapula when lifting demonstrated excellent ICC inter-tester reliability with varying 95% CI ranges, with scores of ICC = 0.79 [95% CI: 0.57, 0.90] for the dominant side and ICC = 1.00 [95% CI: perfect agreement] for the non-dominant. Control of the scapula when lowering scored excellent ICC inter-tester reliability on both the dominant and non-dominant sides, with narrow 95% CI ranges. Inter-tester ICC reliability for symmetry between both scapula was the worst element of the SCT, as it demonstrated good reliability and the widest 95% CI range, ICC = 0.64 [95% CI: 0.26, 0.83].

In terms of intra-tester ICC reliability, the SCT scored excellent for all components and had very narrow 95% CI ranges (Table 3.12). All components scored above 0.90 for ICC and with three components reporting perfect agreement, both dominant and non-dominant control of the scapula lifting, and dominant control of the scapula when lowering.

Table 3.12 ICC Inter-tester & intra-tester reliability of the Scapular Control Test

Scapular Control Test	Side	Inter-tester Reliability		Intra-tester Reliability	
		ICC	ICC 95% CI	ICC	ICC 95% CI
Winging	D	0.90*	0.78, 0.95	0.97*	0.93, 0.98
	ND	0.86*	0.67, 0.94	0.92*	0.84, 0.96
Control of Scapula when Lifting	D	0.79*	0.57, 0.90	1.00*	PA
	ND	1.00*	PA	1.00*	PA
Control of Scapula when Lowering	D	0.94*	0.88, 0.97	1.00*	PA
	ND	0.90*	0.78, 0.95	0.98*	0.96, 0.99
Symmetry between both scapulae		0.64 [#]	0.26, 0.83	0.91*	0.81, 0.96

All measures were ordinal data: 0 = normal, 1 = slight, 2 = moderate, 3 = severe

ICC classification: Poor (<0.40) = [®], Good (0.40-0.75) = [#], Excellent (>0.75) = ^{*}

PA = Perfect agreement; D = Dominant; ND = Non-dominant

3.5.2 Validity

Results of the MAPs of the normal group and the dyskinesia group for the abduction component of the SDT are displayed in Table 3.13, Figure 3.7, and Figure 3.8. Table 3.13 also displays the 95% CI, significant differences, and effect size for all muscles. In all figures displaying MAPs, 0.00 seconds represents movement of the limb, negative values represent muscle activation prior to the start of movement, and positive values represent muscle activation after the start of movement. Results demonstrated no significant differences in any muscle between the normal and dyskinesia groups. MAPs for the middle deltoid muscle on the non-dominant side trended towards a later activation in the dyskinesia group, however, similarly to all other muscles this was not found to be significant ($p = 0.095$), but it did report a medium effect ($d = 0.643$), as did the infraspinatus muscle on the dominant side ($d = 0.691$).

Table 3.13 Scapular Dyskinesia Test MAPs during abduction: mean (SD) times of muscle activation for normal & dyskinesia groups

Muscle	MD		TU		TL		Inf		SA	
	Normal	Dyskinesia	Normal	Dyskinesia	Normal	Dyskinesia	Normal	Dyskinesia	Normal	Dyskinesia
ND (s)	0.166 (0.299)	0.371 (0.337)	0.158 (0.449)	0.392 (0.452)	0.259 (0.314)	0.367 (0.571)	-0.243 (0.288)	-0.207 (0.443)	0.866 (0.433)	0.900 (0.306)
95% CI	-0.031, 0.366	0.198, 0.544	-0.141, 0.456	0.160, 0.625	0.060, 0.475	0.074, 0.660	-0.426, -0.060	-0.435, 0.021	0.631, 1.176	0.742, 1.057
n	13	17	13	17	13	17	12*	17	13	17
Sig.	0.095		0.169		0.544		0.808		0.803	
d	0.643		0.454		0.244		0.098		0.092	
D (s)	0.342 (0.283)	0.402 (0.540)	0.482 (0.478)	0.309 (0.628)	0.383 (0.358)	0.258 (0.562)	0.091 (0.587)	-0.196 (0.242)	0.716 (0.469)	0.626 (0.550)
95% CI	0.202, 0.483	0.059, 0.745	0.244, 0.720	-0.091, 0.708	0.205, 0.561	-0.099, 0.615	-0.201, 0.383	-0.350, -0.042	0.483, 0.949	0.276, 0.976
n	18	12	18	12	18	12	18	12	18	12
Sig.	0.695		0.398		0.463		0.122		0.634	
d	0.145		0.313		0.271		0.691		0.177	

MD = Middle Deltoid; TU = Trapezius Upper; TL = Trapezius Lower; Inf = Infraspinatus; SA = Serratus Anterior; ND = Non-dominant; D = Dominant; s = seconds; 95% CI = 95% Confidence Interval; n = number per group; minus "-" = muscle activated before movement; Sig. = Significance; d = Cohen's d; * = missing data

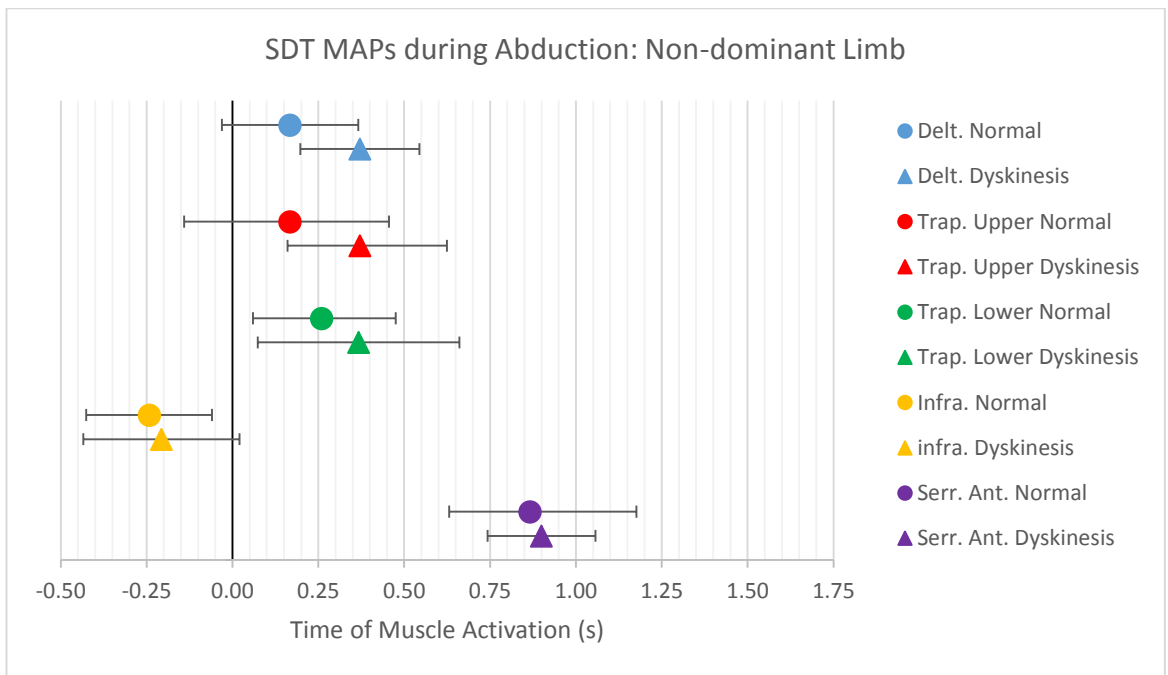


Figure 3.7 SDT MAPs during abduction: non-dominant limb mean and 95% confidence interval times of muscle activation for normal (n = 13) & dyskinesia (n = 17) groups

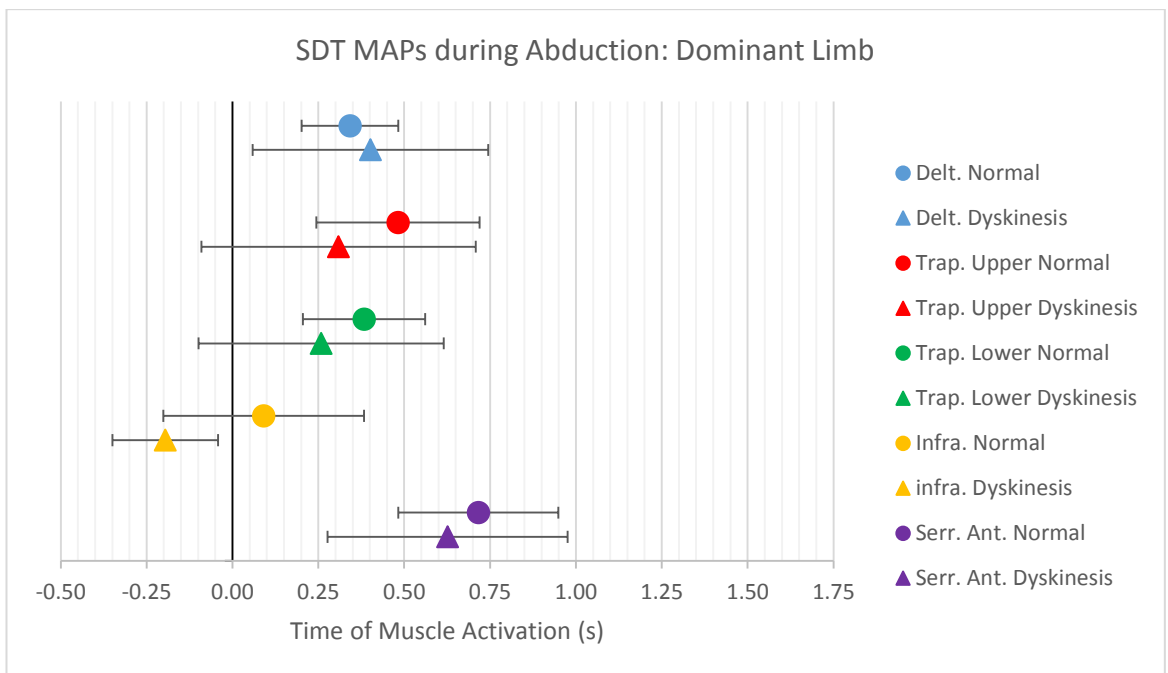


Figure 3.8 SDT MAPs during abduction: dominant limb mean and 95% confidence interval times of muscle activation for normal (n = 12/13) & dyskinesia (n = 6) groups

Results of the MAPs of the normal group and the dyskinesia group for the flexion component of the SDT are displayed in Table 3.14, Figure 3.9, and Figure 3.10. Similar to that of the abduction component, the flexion component of the SDT demonstrated no significant differences in any muscle between the normal and dyskinesia groups. This corresponded with no large effect sizes, where all values demonstrated either small or medium effects.

Table 3.14 Scapular Dyskinesia Test MAPs during flexion: mean (SD) times of muscle activation for normal & dyskinesia groups

Muscle	MD		TU		TL		Inf		SA	
	Normal	Dyskinesia	Normal	Dyskinesia	Normal	Dyskinesia	Normal	Dyskinesia	Normal	Dyskinesia
ND (s)	1.138 (0.489)	0.933 (0.311)	0.331 (0.741)	0.334 (0.498)	0.120 (0.482)	0.077 (0.600)	-0.032 (0.331)	-0.134 (0.422)	0.776 (0.456)	0.675 (0.422)
95% CI	0.766, 1.416	0.773, 1.093	-0.138, 0.853	0.077, 0.590	-0.244, 0.432	-0.231, 0.386	-0.254, 0.210	-0.351, 0.083	0.450, 1.111	0.458, 0.892
n	12*	17	13	17	13	17	12*	17	13	17
Sig.	0.179		0.989		0.836		0.493		0.537	
d	0.511		0.005		0.079		0.270		0.229	
D (s)	1.138 (0.489)	0.933 (0.311)	0.331 (0.741)	0.334 (0.498)	0.120 (0.482)	0.077 (0.600)	0.190 (0.460)	-0.021 (0.310)	0.462 (0.594)	0.490 (0.574)
95% CI	0.672, 1.104	0.432, 1.376	0.358, 1.023	-0.207, 0.992	-0.193, 0.234	-0.144, 0.682	-0.039, 0.419	-0.218, 0.175	0.167, 0.757	0.125, 0.854
n	18	12	18	12	18	12	18	12	18	12
Sig.	0.179		0.989		0.836		0.176		0.899	
d	0.027		0.370		0.462		0.548		0.048	

MD = Middle Deltoid; TU = Trapezius Upper; TL = Trapezius Lower; Inf = Infraspinatus; SA = Serratus Anterior; ND = Non-dominant; D = Dominant; s = seconds; 95% CI = 95% Confidence Interval; n = number per group; minus "-" = muscle activated before movement; Sig. = Significance; d = Cohen's d; * = missing data

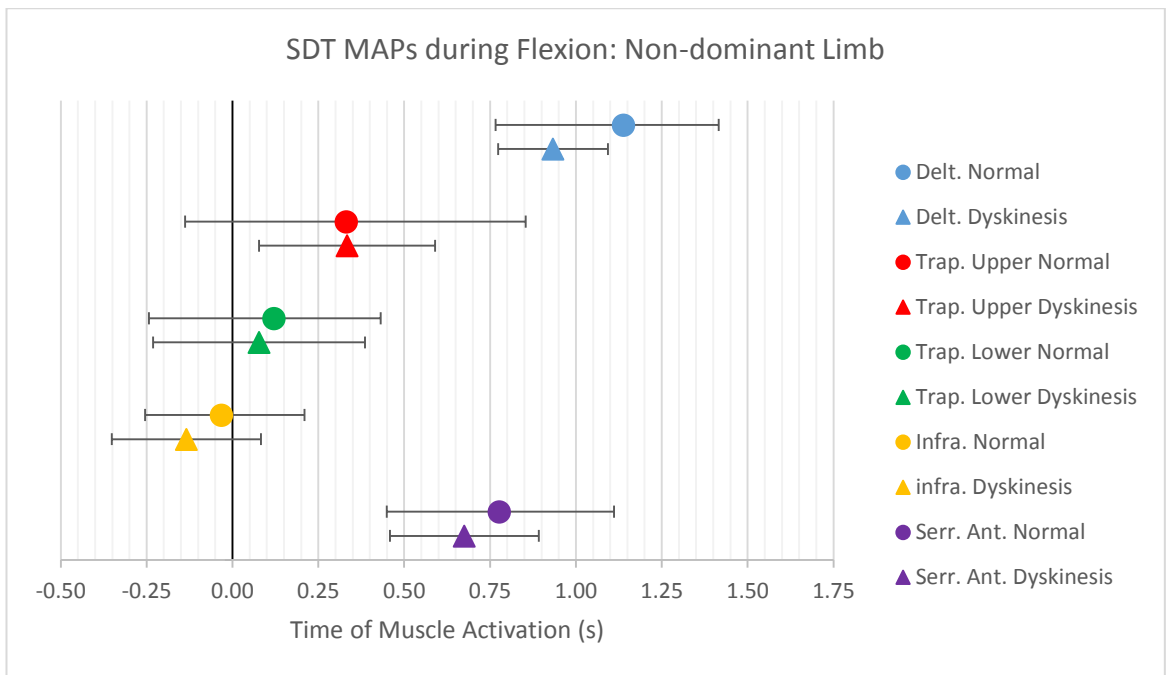


Figure 3.9 SDT MAPs during flexion: non-dominant limb mean and 95% confidence interval times of muscle activation for normal (n = 12/13) & dyskinesia (n = 17) groups

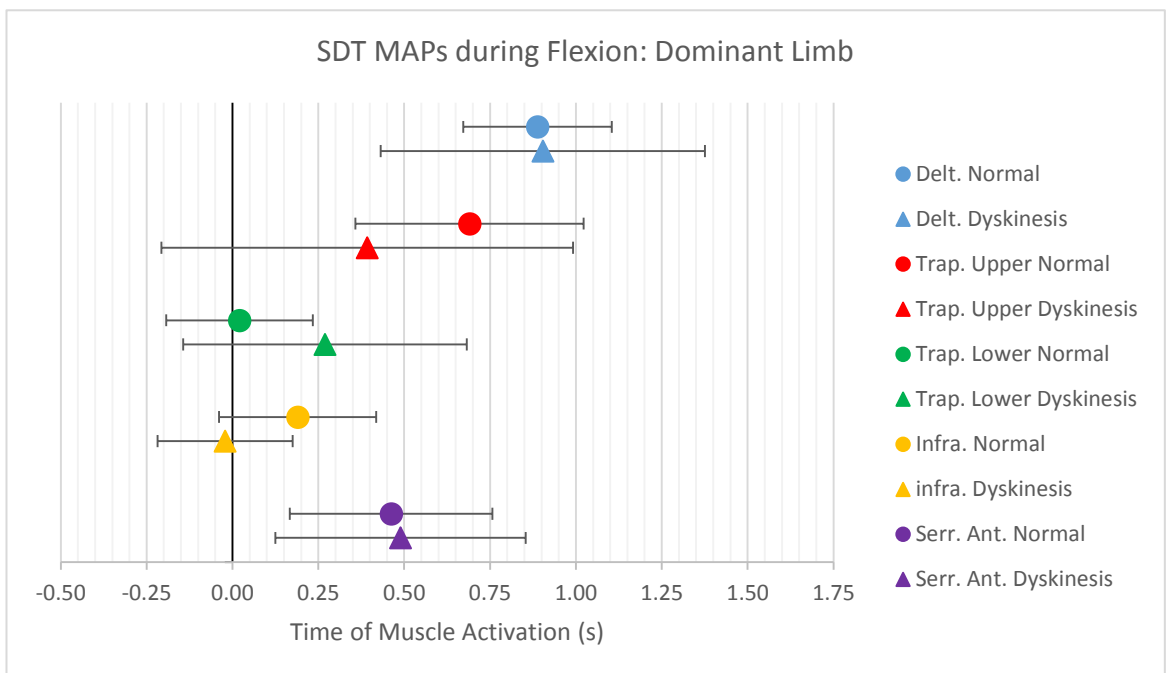


Figure 3.10 SDT MAPs during flexion: dominant limb mean and 95% confidence interval times of muscle activation for normal (n = 18) & dyskinesia (n = 12) groups

Results of the MAPs of the normal group and the dyskinesia group during the SCT are displayed in Table 3.15, Figure 3.11, and Figure 3.12. Results demonstrated a significant difference ($p = 0.014$) between groups in the non-dominant limbs' infraspinatus muscle only, with a mean onset of muscle activity in the normal group of 0.389 seconds [95% CI: 0.193, 0.586], -0.037 seconds [95% CI: -0.331, 0.257] in the dyskinesia group, and a mean difference of 0.427 seconds [95% CI: 0.094, 0.760]. This significant difference was associated with a large effect size of 1.079. However, as illustrated in Figure 3.11, the 95% CIs of the normal and dyskinesia groups overlapped, creating uncertainty as to whether the difference observed in the MAPs of the infraspinatus muscle can be considered a significant result. There was no significant difference in the MAPs or large effect sizes of any other muscles, which demonstrated similar mean onsets of muscle activity and large overlaps in the 95% CI ranges.

Table 3.15 Scapular Control Test MAPs during abduction: mean (SD) times of muscle activation for normal & dyskinesia groups

Muscle	MD		TU		TL		Inf		SA	
	Normal	Dyskinesia	Normal	Dyskinesia	Normal	Dyskinesia	Normal	Dyskinesia	Normal	Dyskinesia
ND (s)	0.399 (0.338)	0.472 (0.383)	0.570 (0.612)	0.603 (0.492)	0.805 (0.437)	0.867 (0.428)	0.389 (0.408)	-0.037 (0.383)	1.235 (0.435)	1.427 (330)
95% CI	0.236, 0.562	0.189, 0.830	0.275, 0.865	0.278, 1.066	0.595, 1.016	0.578, 1.277	0.193, 0.586	-0.331, 0.257	1.026, 1.445	1.144, 1.657
n	19	11	19	11	19	11	19	9▪	19	11
Sig.	0.592		0.878		0.711		0.014*		0.210	
d	0.202		0.061		0.142		1.079		0.500	
D (s)	0.565 (0.468)	0.502 (0.332)	0.648 (0.502)	0.698 (0.585)	0.797 (0.456)	0.830 (0.586)	0.605 (0.769)	0.278 (0.670)	1.132 (0.552)	1.013 (0.449)
95% CI	0.318, 0.795	0.279, 0.726	0.398, 0.898	0.305, 1.091	0.557, 1.023	0.436, 1.223	0.176, 0.857	-0.172, 0.728	0.832, 1.389	0.711, 1.315
n	19	11	18▪	11	19	11	19	11	19	11
Sig.	0.698		0.810		0.546		0.251		0.547	
d	0.158		0.091		0.063		0.454		0.239	

MD = Middle Deltoid; TU = Trapezius Upper; TL = Trapezius Lower; Inf = Infraspinatus; SA = Serratus Anterior; ND = Non-dominant; D = Dominant; s = seconds; 95% CI = 95% Confidence Interval; n = number per group; minus "-" = muscle activated before movement; Sig. = Significance; d = Cohen's d; sig <0.05 = *; ▪ = missing data

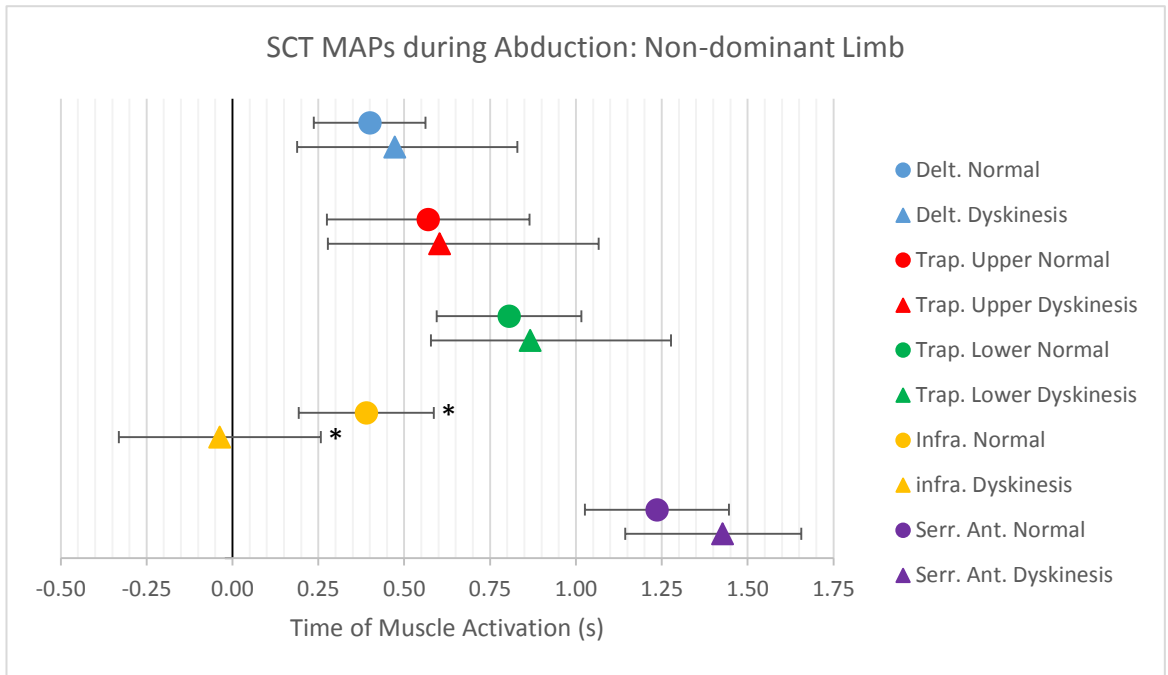


Figure 3.11 SCT MAPs during abduction: non-dominant limb mean and 95% confidence interval times of muscle activation for normal (n = 19) & dyskinesia (n = 9/11) groups (*: p = 0.014)

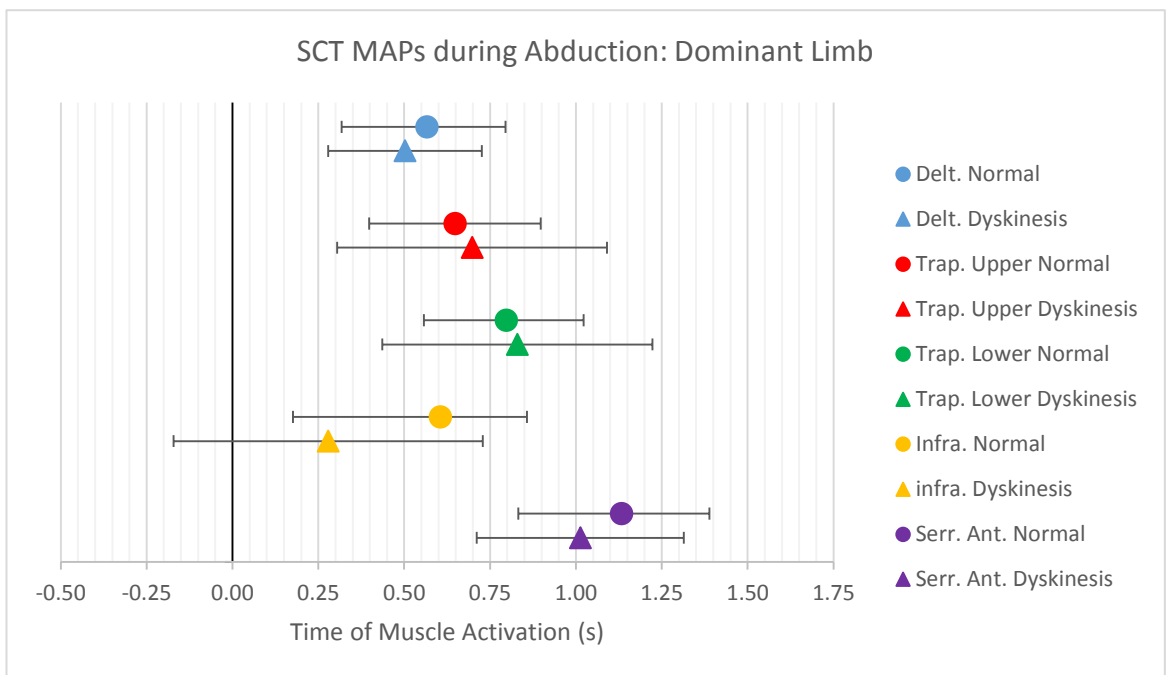


Figure 3.12 SCT MAPs during abduction: dominant limb mean and 95% confidence interval times of muscle activation for normal (n = 18/19) & dyskinesia (n = 11) groups

3.6 Discussion

This pilot study investigated the reliability of two clinical assessment tests for scapular dyskinesis; the McClure et al. (2009) SDT and the O'Connor et al. (2015) SCT. It also investigated the validity of these two tests by examining whether those judged to display scapular dyskinesis had altered MAPs compared to those determined to display normal scapular motion.

3.6.1 Inter-tester and Intra-tester Reliability

Clinical scapular dyskinesis tests have been developed to provide a standardised method for visually assessing altered motion of the scapula. Inter-tester reliability has been established in all previously published scapular dyskinesis tests (Ellenbecker et al., 2012; Kibler et al., 2002; McClure et al., 2009; O'Connor et al., 2015; Uhl et al., 2009) however, only the Kibler et al. (2002) 4-type method and the O'Connor et al. (2015) SCT have been assessed for intra-tester reliability. This study conducted both inter- and intra-tester reliability to fully examine the reliability of the SDT and the SCT. Previous studies have used one of three methods to assess the reliability of scapular dyskinesis tests, kappa (κ) coefficients, weighted kappa (κ_w) coefficients, or intraclass correlation coefficients (ICC). In tests such as the 4-type method and 2-type method, investigated by Kibler et al. (2002), Uhl et al. (2009) and Ellenbecker et al. (2012), data was nominal and the seriousness of disagreement between testers was equal, thus κ coefficients were used. However, in both the SDT and the SCT the data is ordinal and the level of disagreement between testers has the potential to vary in its seriousness. As mentioned in section 3.3.2.2 the SDT uses a three point ordinal scale (normal, subtle dyskinesis, and obvious dyskinesis), while the SCT utilises a four point ordinal scale (none, slight, moderate, and severe). Thus, if one tester identifies a subject to possess normal scapular motion and the other tester identifies the same subject to possess subtle dyskinesis, the seriousness of disagreement is not the same as if one tester identifies a subject to possess normal scapular motion and the other tester obvious dyskinesis. Due to the potential for varying seriousness, the SDT and SCT are typically examined using κ_w coefficients and ICC, as both methods account for the severity of disagreement between testers. While both κ_w and ICC are equally effective for assessing such data, this study has primarily used ICC calculations for inter- and intra-tester reliability, but has also computed κ_w for direct comparison with previous publications. Table 3.16 displays the inter-tester reliability

results from all previous classification systems for clear comparison with the results from this study.

The inter-tester reliability for the SDT demonstrated excellent ICC inter-tester reliability (ICC = 0.89, 97), which was accompanied by narrow ICC 95% CI ranges that were above the threshold for the excellent classification. The SDT completed in this study demonstrated greater inter-tester reliability compared to ICC scores reported by O'Connor et al. (2015) for the SCT (ICC = 0.80, 1.00). As the initial publication of the SDT by McClure et al. (2009) utilised κ_w calculations for reliability they have also been calculated for a direct comparison. The κ_w inter-tester reliability scores for the SDT in the present study demonstrated excellent reliability with $\kappa_w = 0.76$ (95% CI: 0.56, 0.97) for the non-dominant limb and $\kappa_w = 0.92$ (95% CI: 0.82, 1.00) for the dominant limb. While the 95% CI, specifically for the non-dominant limb, had a wide range, both the κ_w and 95% CI values computed for the SDT in this study showed higher results to those reported by McClure et al. (2009) (see Table 3.16). The improved κ_w scores reported in the present study may have been due to testers undergoing standardised training for both the SDT and the SCT, in comparison to only the SDT in the McClure et al. (2009) study, which could indicate a greater understanding of scapular dyskinesis and how it is assessed. Taking into consideration the excellent inter-tester reliability demonstrated in both the ICC and κ_w calculations for the SDT in the present study it performed better in comparison to other previously published classifications systems (see Table 3.16).

The original publication of the SCT, by O'Connor et al. (2015), only reported ICC values for inter-tester reliability making direct comparison with the McClure et al. (2009) study, which used κ_w , difficult. The ICC inter-tester results for the SCT in the present study (ICC = 0.64-1.00 [95% CI: 0.26, 1.00]) are similar to those demonstrated by O'Connor et al. (2015) (ICC = 0.80-1.00 [95% CI: 0.55, 1.00]). However, one component of the test, symmetry between both scapulae, reduced from excellent inter-tester reliability, reported by O'Connor et al. (2015), to good inter-tester reliability in the present study (ICC = 0.64 [0.26, 0.83]). Symmetry between both scapulae also demonstrated the widest 95% CI range. This is potentially due to the fact that O'Connor et al. (2015) conducted the test live compared to the present study, where testers observed video recordings of participants. In the O'Connor et al. (2015) study testers observed five repetitions however, in the present study testers could have reviewed the video as many

times as needed. The more times testers viewed each participants they potentially could have been more critical of the quality of the motion. There were also differences in how the two studies trained the testers. In the O'Connor et al. (2015) study testers underwent three training sessions where the instructions and scoring system of the test were explained, demonstrations of the test were given and opportunities to practice the test were provided, along with a standardised self-directed presentation. In comparison, training for testers in the present study only involved the use of the standardised self-directed presentation, which detailed normal and abnormal motion of the ST joint, instructions for the test and its scoring system, examples of how to implement the test and an assessment of the testers ability to implement it. The results from the present study demonstrated that testers that only undergo standardised self-directed training can produce similar inter-tester reliability results to testers whom undergo three live training session as well as the standardised self-directed training. These results can thus infer that self-directed training should be utilised as a cost effective method for the training of therapists.

Table 3.16 Inter-tester reliability values of all scapular dyskinesis classification systems

Study	(N)	Assessment Method	Plane of Motion	Live/ Video	Testers	Inter-tester Value
Kibler et al. (2002)	26	4-type method	Frontal & Scapular	Video	2 physicians	$\kappa = 0.31$ ($p < .01$)
					2 physical therapists	$\kappa = 0.42$ ($p < .001$)
Uhl et al. (2009)	56	4-type method	Sagittal & Scapular	Live	1 physical therapist & 1 physician	$\kappa = 0.44$ ($p < .01$)
		2-type method	Sagittal & Scapular			$\kappa = 0.41$ ($p < .01$)
McClure et al. (2009)	90	SDT	Sagittal & Frontal	Video	2 athletic trainers & 4 physical therapists	R $\kappa_w = 0.61$ 95%CI= 0.43, 0.78 L $\kappa_w = 0.48$ 95%CI= 0.29, 0.67
	142			Live	5 physical therapists	R $\kappa_w = 0.55$ 95%CI= 0.32, 0.78 L $\kappa_w = 0.58$ 95%CI= 0.38, 0.79
Ellenbecker et al. (2012)	71	4-type method	Scapular	Video	2 orthopaedic surgeons & 2 physical therapists	R $\kappa = 0.19$ 95%CI= 0.11, 0.21 L $\kappa = 0.25$ 95%CI= 0.18, 0.32
		2-type method				R $\kappa = 0.16$ 95%CI= 0.05, 0.27 L $\kappa = 0.26$ 95%CI= 0.13, 0.40
		symmetry				$\kappa = 0.08$ 95%CI= -0.01, 0.30
O'Connor et al. (2015)	15	SCT	Frontal	Live	2 athletic therapists & 1 physical therapist	ICC= 0.80-1.00 95%CI= 0.55, 1.00

(N) = number in study/group; SDT = Scapular Dyskinesis Test; SCT = Scapular Control Test; κ = Kappa; κ_w = Weighted Kappa; ICC = Intraclass Correlation coefficients; 95%CI = 95% Confidence Intervals

Intra-tester reliability is an important requirement for clinical tests however, only the Kibler et al. (2002) 4-type method and the O'Connor et al. (2015) SCT have been previously assessed for intra-tester reliability. To thoroughly examine both the SDT and the SCT, the present study conducted intra-tester reliability on the SDT for the first time, and repeated the analysis on the SCT. Results demonstrated intra-tester reliability, utilising ICC, to be excellent, with narrow 95% CI ranges for both tests, SDT ICC = 0.96-

0.97 [95% CI: 0.92-0.99] and SCT ICC = 0.91-1.00 [95% CI: 0.81-1.00]. O'Connor et al. (2015) reported good to excellent ICC intra-tester reliability for the SCT, there was however a large 95% CI distribution (ICC = 0.60-1.00 [95% CI: 0.12-0.97]) (Table 3.17). Methodological differences between each study may have influenced this change in 95% CI, as the O'Connor et al. (2015) study was conducted live compared to video recording utilised in the present study. Implementing a live methodology meant that participants returned for a second testing session one week following the first. Slight variations in participants' scapular motion from one session to the next may have affected the intra-tester reliability results. However, in the present study video tapes were used when assessing participants, meaning the principal investigator examined the same video tape for the second assessment. This removed any possibility of slight variations in scapular motion between tests.

Kibler et al. (2002) has been the only study to conduct intra-tester reliability on their 4-type method of assessing scapular dyskinesis. As data collected from their study was nominal, Kibler et al. (2002) utilised κ statistics for intra-tester reliability (Table 3.17). This makes a direct comparison with the present study with the same statistical measure unfeasible. However, a comparison can be made between the classifications reported by both studies. Kibler et al. (2002) reported κ values of 0.42 ($p < 0.001$) and 0.49 ($p < 0.001$), for two different testers, indicating good intra-tester reliability. In comparison both the SDT and the SCT achieved excellent classification, with all ICC values greater than 0.90. These higher intra-tester reliability results in the present study are likely due to a more extensive training procedure for testers. Testers in the Kibler et al. (2002) study only received a ten minute visual and verbal presentation along with written descriptions of the four patterns, which could be referred to when assessing participants.

Table 3.17 Intra-tester reliability values of all scapular dyskinesis classification systems

Study	(N)	Assessment Method	Plane of Motion	Live/ Video	Tester	Kappa	ICC
Kibler et al. (2002)	26	4-type method	Coronal & Scapular	Video	1 physician	$\kappa = 0.42$ ($p < .001$)	None
					1 physical therapist	$\kappa = 0.49$ ($p < .001$)	None
						$\kappa = 0.41-1.00$ 95%CI= 0.00-1.00	ICC= 0.60-1.00 95%CI= 0.16-1.00
O'Connor et al. (2015)	15	SCT	Coronal	Live	Tester A	$\kappa = 0.44-1.00$ 95%CI= 0.00-1.00	ICC= 0.63-1.00 95%CI= 0.06-1.00
					Tester B	$\kappa = 0.58-1.00$ 95%CI= 0.07-1.00	ICC= 0.75-1.00 95%CI= 0.23-1.00

(N) = number in study/group; SCT = Scapular Control Test; κ = Kappa; ICC = Intraclass Correlation coefficients; 95% CI = 95% Confidence Intervals

3.6.2 Validity

A main objective of this study was to determine whether participants who were perceived to possess altered motion of their scapulae also possessed altered MAPs. As no statistical difference in MAPs between groups was found in almost all muscles, neither test can be said to be valid, based on altered MAPs. Only during the SCT was there a significant difference ($p = 0.014$) and large effect size ($d = 1.079$) in the mean muscle activation times of the infraspinatus muscle on the non-dominant side. However, as the primary role of the infraspinatus muscle is to act as a stabiliser and external rotator of the GH joint it is unlikely that the difference observed between the normal and dyskinesis groups during the SCT would account for visual differences observed by the testers. Along with the large difference between groups observed in the mean muscle activation times of the infraspinatus, it also demonstrated narrow 95% CI ranges. All other muscles demonstrated either relatively similar mean muscle activation times or large differences with wide 95% CI ranges, causing considerable overlapping between the two groups. Despite the significant difference found in the infraspinatus muscle on the non-dominant side during the SCT, the results from this pilot study demonstrate that neither of the two scapular dyskinesis tests assessed are valid clinical tests for determining the presence of altered MAPs in key muscles of the shoulder girdle, in people with perceived altered scapular motion.

Validity of the SDT has been previously assessed by Tate et al. (2009) with the use of 3-D electromagnetic kinematic testing. The results from the Tate et al. (2009) study reported that differences were found between the normal and obvious dyskinesia groups. Their results specifically noted that those determined to possess obvious dyskinesia displayed less scapular upward rotation, less clavicular elevation, and greater clavicular protraction. These findings of differences between the normal and obvious dyskinesia groups in several kinematic descriptors provided evidence for validating the SDT. There are a number of differences between the present study, which did not validate either of the tests, and the Tate et al. (2009) study, aside from the different outcome measures. The present study did not recruit any participants that were currently experiencing pain, which is in contrast to Tate et al. (2009) where participants were only excluded if they had a current pain rating of 7/10 or greater on a numeric rating scale, where 0 represented no pain and 10 represented the worst pain possible. The inclusion of some participants with pain could have led to pain being a confounding factor, in that the presence of pain may have caused participants to alter their movement strategy and perhaps cause obvious dyskinesia. The present study did not include participants with pain as these tests are often used as screening tools in pain free subjects. Thus, the current study aimed to assess if a participant perceived to possess scapular dyskinesia, would they also possess significantly different MAPs to a participant preserved to possess normal scapular motion.

Another difference between the Tate et al. (2009) study and the present one was the exclusion of participants with subtle dyskinesia from the 3-D testing which the authors did due to their belief that this rating reflects an ambiguous clinical situation. The authors stated that in people with subtle dyskinesia, the decision to intervene in some way would likely be based on more factors other than motion assessment, whereas a rating of obvious dyskinesia would form a stronger basis for intervention (Tate et al., 2009). The present study grouped all participants with any degree of dyskinesia and assessed them against those determined to possess normal motion in order to decipher whether the presence of dyskinesia, to any degree, corresponded to an alteration in MAPs compared to those whom were considered to possess normal movement. While the different grouping strategies used by both studies makes the direct comparison of the results difficult, it produces another discussion around the use of scapular

dyskinesia: perhaps these subjective clinical tests are only valid at determining differences in extreme cases. Therefore, instead of striving to objectify and alter subtle deviations in what is considered normal scapular motion, perhaps clinicians should only take heed of those with severe or obvious dyskinesia. It is also important for clinicians to be cognisant of the fact that some individuals can possess severe or obvious dyskinesia without compromising their ability to perform functional activities and with a complete absence of pain.

There are a number of potential reasons why the current study did not validate these tests. One of the main reasons there was no statistical difference observed between groups, for all but one muscle, could have been due to the variation seen in the onset of muscle activity between participants. This variation can be seen in all figures displaying MAPs (Figure 3.7 to Figure 3.12) where there is considerable overlap in the 95% CI ranges of each muscle. The inability to identify significant differences between groups could demonstrate that variation in the sequencing of muscle activations is a normal and perhaps desired phenomenon. It has been stated by Latash et al. (2002) that functional systems that are stable and adaptable use all their degrees of freedom effectively in order to optimise task performance. This can be seen in everyday tasks of human movement, where several attempts at the same tasks lead to somewhat different patterns of performance, including the kinematics, kinetics, and patterns of muscle activation (Latash et al., 2002). The variation in onset of muscle activation within groups, caused by individual variation, also meant that it was difficult to determine, with any certainty, whether there was a consistent order to which muscles activated. This was in contrast to studies carried out by Lucas et al. (2010, 2004), whom demonstrated quite consistent MAPs between participants resulting in clear sequencing of muscle activation, due to the narrow standard deviations reported. Lucas et al. (2010) demonstrated an average standard deviation of 0.144 seconds for both their control and LTrP groups during unloaded elevation in the scapular plane of the dominant side. However, the current study demonstrated a standard deviation that ranged from 0.470 seconds to 0.581 seconds on the dominant side during the SDT and the SCT. Table 3.18 illustrates the inconsistencies seen in the MAPs during both the SDT and the SCT in comparison to those reported by Lucas et al. (2010). There are a number of potential

reasons for the variation in MAPs demonstrated in the study compared to the Lucas et al. (2010) study; primarily due to differences in the methodologies used.

Table 3.18 MAPs during the SDT & SCT compared to Lucas et al. (2010)

Muscle activation sequencing	Current Study												Lucas et al. (2010)	
	SDT Abduction				SDT Flexion				SCT Abduction				Scaption	
	Normal		Dyskinesia		Normal		Dyskinesia		Normal		Dyskinesia		Control	LTrP
	ND	D	ND	D	ND	D	ND	D	ND	D	ND	D	D	D
1	Inf.	Inf.	Inf.	Inf.	Inf.	TL	Inf.	Inf.	Inf.	MD	Inf.	Inf.	TU	Inf.
2	TU	MD	TL	TL	TL	Inf.	TL	TL	MD	Inf.	MD	MD	Inf.	TU
3	MD	TL	MD	TU	TU	SA	TU	TU	TU	TU	TU	TU	MD	MD
4	TL	TU	TU	MD	SA	TU	SA	SA	TL	TL	TL	TL	SA	SA
5	SA	SA	SA	SA	MD	MD	MD	MD	SA	SA	SA	SA	TL	TL

SDT = Scapular Dyskinesia Test; SCT = Scapular Control Test; LTrP = Latent Trigger Point Group; ND = Non-dominant side; D = Dominant side; MD = Middle Deltoid; TU = Trapezius Upper; TL = Trapezius Lower; Inf. = Infrapinatus; SA = Serratus Anterior

A possible reason for the wide 95% CI ranges and large SD in muscle activation could be due to inconsistent arm movement between reps and/or between trials. Participants were required to perform bilateral active shoulder elevation in the sagittal plane (flexion) or the frontal plane (abduction) however, slight variations in the angle to which participants performed these movements may have caused inconsistency in their MAPs. The use of a guide panel, as used by Lucas et al. (2010, 2004), set in the desired plane of motion may have mitigated such variations. With the use of a guide panel, participants were required to maintain continuous gentle contact with the guide as they moved through full range of motion. While the use of such guide panels are unlikely in a clinical setting, their use may be necessary when attempting to ascertain discrete variations in MAPs with sEMG sensors in a research setting. This may explain why Lucas et al. (2010, 2004) observed lower SD values in their study.

Another possible cause of the inconsistent MAPs, specifically during the abduction component of the McClure et al. (2009) SDT, may have been due to the requirement for participants to perform external rotation during glenohumeral elevation (the SDT instructs participants to adopt a “thumbs up” position during elevation). It was not specified in the tests guidelines at what stage of elevation participants were to adopt the “thumbs up” position; participants may therefore have adopted it immediately, before movement, or at any stage after movement occurred. This element of the SDT may have compounded the variance seen, specifically in the infraspinatus muscle which performs external rotation at the GH joint.

The results of this pilot study may indicate that there was an unidentified factor that caused the variation in MAPs in both groups; perhaps the presence of LTrPs. Lucas et al. (2010, 2004) investigated the impact of LTrPs on muscle function and reported distinct differences both in the sequencing of muscle activation and in the variation in onset of muscle activity between a LTrP group and a control group without any LTrPs. The Lucas et al. (2010, 2004) studies demonstrated that those without any LTrPs present displayed more consistent MAPs. This was indicated by consistent activation of specific muscles between participants in the control group, resulting in small standard deviation values. The range of standard deviations reported in the control group of the Lucas et al. (2010) study was 0.028-0.117 seconds. Conversely, the LTrP group displayed both within and between subject inconsistencies in the order of muscle activation, resulting in larger

standard deviation values of 0.91-0.401 seconds. The data in the present study may indicate that the presence of unidentified LTrPs could have accounted for the inconsistencies observed in the timing and order of muscle activations. It should also be noted that as mentioned above, the Lucas et al. (2010, 2004) studies incorporated the use of guide panels. This may also account for the more consistent MAPs reported in the Lucas et al. (2010, 2004) studies when compared to the present study, which had standard deviations which ranged between 0.283-0.769 seconds. As the aim of the pilot study was to determine the reliability and validity of the SDT and the SCT participants were not screened for the presence of LTrPs.

Finally, it is imperative to interpret the results from the present study with an important caveat. It was the focus of this study to investigate the validity of the SDT and the SCT using MAPs as an outcome measure. However, this only offers a brief understanding of the entire muscle activity of the assessed periscapular muscles. It does not offer any insight into their activity beyond the time at which they became activated. Therefore, it is not known whether there was a significant difference in the muscle activity of the two groups for the remainder of movement. It is plausible that there may have been differences in the level of motor unit activity between the normal and dyskinesia groups. Motor unit activity is typically measured by examining muscle amplitude, where the firing rate of muscles are observed (Cram and Criswell, 2011). Future research should consider whether those determined to possess scapular dyskinesia demonstrate differences in sEMG amplitude throughout range of motion compared to those with normal scapular motion. While the present study has demonstrated that the SDT and the SCT are not valid at determining abnormal MAPs in those with scapular dyskinesia, it is still unknown whether these tests are valid at assessing abnormal sEMG amplitude. As observable abnormalities in scapular motion, such as winging and dysrhythmia, typically occur at the early stages of GH elevation and/or when lowering the limb from an elevated position, future research should examine whether scapular dyskinesia tests are effective at assessing abnormal sEMG amplitude.

3.7 Limitations

The main limitation to the interpretation of the results of the present study is that it only compared the muscle activation in reference to the onset of movement and it did not examine what occurred within muscles during the entire movement. As abnormalities

in scapular motion typically occur during movement there is a need for future research to focus on determining whether there is any alteration in periscapular muscle activity throughout movements and whether it corresponds to the presence of scapular dyskinesis.

As mentioned in the discussion, this study did not use guide panels during testing to control participants' movement. This may have attributed to the large variation in MAPs, thus future studies should incorporate the use of such panels to mitigate potential variations in MAPs due to inconsistent arm position.

Finally, the recording of participants MAPs did not occur at the same time as the video recording which was used for scapular dyskinesis assessment. Participants were first video recorded performing both the SDT and the SCT, without any sensors on, they were then fitted with the sEMG sensors and repeated the SDT and SCT. Data was collected in this manner to allow testers clear sight of the totality of each participants posterior thorax. Therefore, there is a possibility that some variation in participants' scapular motion occurred from the collection of the video recording to the collection of the sEMG data.

3.8 Conclusion

Both the SDT and the SCT demonstrated high levels of inter-tester reliability and excellent intra-tester reliability. However, neither of these clinical tests for scapular dyskinesis were valid at determining the presence of altered MAPs between those perceived to possess dyskinesis and those without. This may have been caused by issues in the methodology, the absence of guide panels, the presence of LTrPs, and the inclusion of subjects with subtle/slight and moderate dyskinesis in the analysis. Alternatively, it may demonstrate that these tests might only be effective at determining differences at extreme ends of the spectrum, normal scapular movement and severe or obvious dyskinesis.

3.9 Summary

While both the SDT and SCT demonstrated high levels of reliability in the present study, their inability to be validated at determining the presence of altered MAPs may have been due to an unaccounted confounding factor. It is plausible that an unidentified deficiency, specifically the possible presence of LTrPs, caused the high variation in MAPs

across groups. Future research should take all necessary steps to mitigate variations in MAPs due to methodological issues. Findings from the present research suggest to utilise a clinical scapular dyskinesis test that does not involve a rotational element to the task of bilateral GH elevation and that guide panels ought to be used to standardise the plane of motion in which GH elevation is performed, both between and within subjects.

Chapter 4: An Investigation into the Efficacy of LTrP Dry Needling on Shoulder MAPs

4 An Investigation into the Effectiveness of LTrP Dry Needling on Shoulder MAPs

4.1 Abstract

Background: Despite extensive research into TrPs, there is still a lack of understanding behind their underlying pathophysiology. Due to this, the treatment options for TrPs are also under scrutiny. LTrPs, which do not cause pain, offer an avenue to investigate the effectiveness of TrP treatments without pain confounding results. One proposed negative outcome of both ATrP and LTrP forms, is that of altered MAPs. Thus examining how TrP treatments affect MAPs in pain free participants with LTrPs has the potential to add to the knowledge both on the potential negative effects of LTrPs and the efficacy of the suggested treatments.

Aim: To investigate the effectiveness of LTrP dry needling versus manual release and placebo dry needling in key muscles of the shoulder complex.

Methods: Sixty participants were randomly assigned to one of three treatment groups. The effectiveness of the three treatments on LTrPs was assessed pre- and post-intervention to determine their effect on MAPs. Both subjective (SCT) and objective (sEMG data) outcome measures were utilised to assess the effectiveness of each treatment. Wilcoxon signed rank and Kruskal-Wallis tests were carried out on the SCT and a mixed between-within ANOVA was used for the sEMG data.

Results: There were no statistically significant differences in MAPs as a result of any of the treatments, as assessed using the using the SCT and sEMG data.

Conclusion: Dry needling and manual release treatment interventions were not effective at altering the MAPs of participants with LTrPs. Therefore, further research is required to determine their value as a treatment intervention.

4.2 Introduction

TrPs have been proposed as the cause of muscular pain or tenderness, which patients often describe as diffuse and non-specific. TrPs are described as hyperirritable points located within a taut band of skeletal muscle or fascia, which cause the local tenderness. TrPs may also cause referred pain and autonomic symptoms (Bron and Dommerholt, 2012). There are two basic types of TrPs, active (ATrPs) and latent (LTrPs), the fundamental difference being when they cause pain or discomfort to the patient. ATrPs cause spontaneous pain at rest or with contraction or stretching of the involved muscle, while LTrPs only cause pain when stimulated manually, such as during manual compression or with the insertion of a needle (Simons et al., 1999). ATrPs have been researched extensively with numerous studies published on areas such as their pathophysiology (Bron and Dommerholt, 2012; Gautam et al., 2010; Gerwin, 2010; Gerwin et al., 2004; Hägg, 2000; Hocking, 2010; Hubbard and Berkoff, 1993; Shah et al., 2005; Simons et al., 1999) and treatments (Beckerman et al., 1992; Cummings and White, 2001; de las Peñas et al., 2005; Draper et al., 2010; Hong, 2004; Hong et al., 1993; Hsieh et al., 2007; Ilbuldu et al., 2004; Irnich et al., 2002; Itoh et al., 2007; Kietrys et al., 2013; Manca et al., 2014; Pal et al., 2014; Rodríguez-Fernández et al., 2011; Simons et al., 1999; Tekin et al., 2013; Tsai et al., 2010; Yeganeh Lari et al., 2016).

While it has generally been acknowledged that LTrPs precede ATrPs, research has only recently begun to focus on LTrPs in more detail and they have been shown to be significantly more complex in nature (Celik and Mutlu, 2013). Research has recently proposed that these minor, subclinical neuromuscular lesions, which do not cause the patient any pain until compressed, also have a negative effect on the muscle prior to their conversion into ATrPs (Celik and Mutlu, 2013). Recent studies have proposed that LTrPs cause: local tenderness with and without referred pain upon mechanical stimulation (Ge and Arendt-Nielsen, 2011), restricted range of motion (Grieve et al., 2011; Montanez-Aguilera et al., 2009; Simons, 2004; Trampas et al., 2010), muscle weakness (Ge and Arendt-Nielsen, 2011; Ge et al., 2012; Simons et al., 1999), muscle fatigue (Ge et al., 2012), alteration of MAPs (Ge et al., 2008; Lucas et al., 2010, 2004), and induced muscle cramping (Ge et al., 2008; Lucas et al., 2010; Xu et al., 2010). Thus, the potential negative implication of LTrPs on muscle function and the possible risk of injury highlights a need to investigate the potential benefits of their treatment.

Due to the presence of pain in the active form of TrPs, it has been challenging to determine the true effectiveness of the various treatment interventions. Many of the proposed clinical implications of TrPs, such as altered MAPs, may be a result of the pain the patient experiences. Thus to truly assess the effectiveness of the various treatment interventions, the LTrP form affords researchers and clinicians the opportunity to assess any improvements in the proposed implications following treatment with the absence of pain. Wadsworth & Bullock-Saxton (1997) have demonstrated the role of altered MAPs in shoulder pathologies such a chronic impingement syndrome. Wadsworth & Bullock-Saxton (1997) demonstrated that young elite male swimmers with unilateral chronic shoulder impingement syndrome possessed altered timing of muscle activation in the upward rotators of the scapula. However, it was established that the presence of the painful condition could have accounted for the change in MAPs and thus a cause and effect relationship could not be established. It was therefore necessary to establish whether pain-free subjects with some form of a deficiency could establish a cause and effect relationship. As a result Lucas et al. (2004) investigated whether LTrPs could be the responsible deficiency, due to their hypothesised development, muscle overload (Simons et al., 1999), and their pain-free nature (Lucas et al., 2004; Simons et al., 1999).

Numerous treatment interventions have been investigated for their effectiveness on ATrPs, with varying results, of which dry needling and manual release are the most established. The only study which investigated LTrP dry needling and MAPs was Lucas et al. (2004), however they utilised a combination of dry needling and passive stretch, and compared it to a sham ultrasound treatment and a control group whom did not have any LTrPs. This was due to the fact the Lucas et al. (2004) were investigating the possibility that LTrPs caused altered MAPs rather than the effectiveness of dry needling. Therefore this present study aimed to investigate the effectiveness of dry needling in comparison to manual release and placebo dry needling at altering MAPs.

4.3 Methodology

4.3.1 Participants

Sixty two healthy males volunteered from a convenience sample of collegiate staff, students, and the general public. To determine the study sample size, a priori power analysis was performed to provide a statistical power of 80% at an α level of 0.05. Equation 4.1 was implemented using previous data (Lucas et al., 2010) with a standard deviation (s) value of 0.11 seconds and a difference to be detected (d) value of 0.1 seconds, where time values represent the onset of muscle activation. From this calculation a sample size of 19 participants per group was determined to be sufficient. Therefore a sample size of 60 participants, with group sizes of 20 participants, was decided. Collegiate staff and students were recruited by email (Appendix G), and with advertising posters around the institute (Appendix H). The general public were recruited by word of mouth. All but two of the volunteers satisfied the inclusion and exclusion outlined in Table 4.1 (due to upper limb pain and the absence of LTrPs), thus the study was conducted on 60 participants (Table 4.2).

$$n = 16 \times \frac{s^2}{d^2}$$

Equation 4.1 Priori power analysis (s = standard deviation; d = difference to be detected)

Table 4.1 Main study inclusion & exclusion criteria

Inclusion Criteria
1. Male
2. Aged 18-45 years
3. No upper limb, back, or neck pain 7 days prior to testing
Exclusion Criteria
1. Any pain in the back, neck, or upper limb 7 days prior to testing
2. Significantly increased thoracic kyphosis (excessive curvature of the thoracic spine)
3. History of any injuries to the neck or upper limb
4. History of any nerve injuries in the cervical or shoulder area
5. Any allergies to adhesives or metals
6. Needle phobia or those with a history of fainting from needling therapies
7. Body mass index (BMI) of ≥ 30
8. $< 160^\circ$ arm elevation
9. Tenderness on the greater tuberosity of the humerus, AC joint
10. Positive impingement tests
11. Any active trigger points

Table 4.2 Main study: participant demographics (mean \pm SD)

Participant Demographics	(n = 60)
Age (y)	25 \pm 5
Hand Dominance	57 Right & 3 Left
Height (m)	1.80 \pm 0.06
Weight (kg)	83.1 \pm 10.7
Body Mass Index (kg/m ²)	25.6 \pm 2.4
Left Overhead Elevation (°)	167 \pm 6
Right Overhead Elevation (°)	167 \pm 6
Left External Rotation 0° (°)	66 \pm 24
Right External Rotation 0° (°)	69 \pm 23
Left External Rotation 90° (°)	99 \pm 19
Right External Rotation 90° (°)	101 \pm 11
Left Internal Rotation (T-Spine)	3 \pm 1
Right Internal Rotation (T-Spine)	4 \pm 2
Left Horizontal Adduction (cm)	26 \pm 4
Right Horizontal Adduction (cm)	26 \pm 4

4.3.2 Study Design

The study consisted of an introductory/screening session and a testing session, Figure 4.1 illustrates the layout.

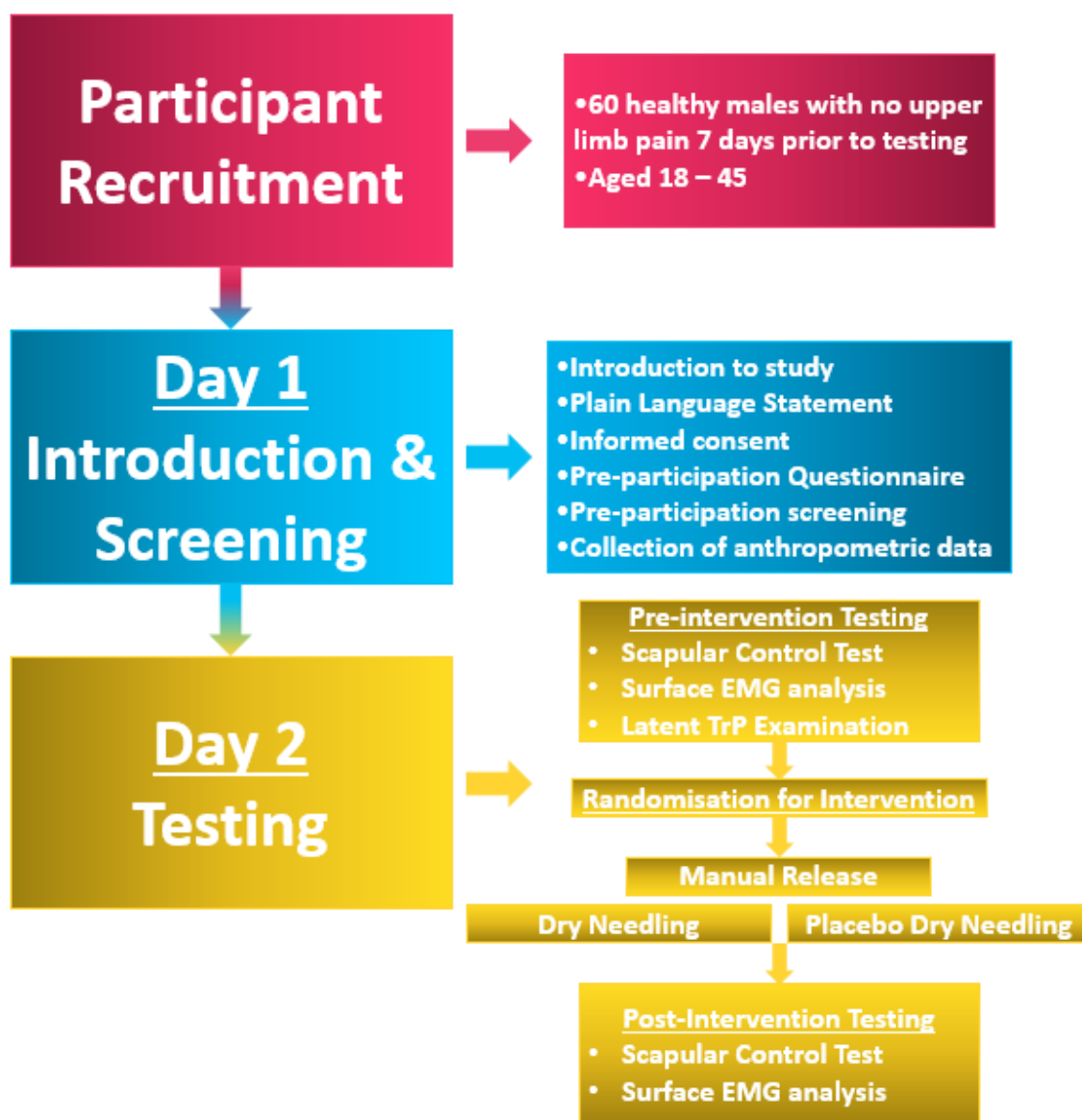


Figure 4.1 Main study layout

4.3.2.1 Introductory, Screening and Familiarisation Session

Volunteers for the study reported to the athletic therapy room for the introductory, screening and familiarisation session. The introductory component of this session outlined the purpose of the study, the testing procedure, participants' requirements, and the risks and benefits associated with the study. After the dissemination of this information participants were given the opportunity to ask any questions they may have had at this point. Participants were also given a plain language statement which outlined the details of the study (Appendix I). Volunteers willing to participate completed an informed consent form prior to their progression to the screening component of the

session (Appendix J). For those who required more time to consider their participation a screening session was organised at a later date.

Upon completion of the informed consent form, participants completed a pre-participation questionnaire, similar to that used in the pilot study, (Appendix K). At this point each participant was given a participant ID number which was used for all further data collection, to ensure anonymity and confidentiality. This questionnaire collected key data required for the study including, date of birth, ethnicity, recent back, neck, or upper limb pain, history of previous injuries, allergies to adhesives and/or metals, and phobia of needles. Participants were required to be pain free seven days prior to assessment, as well as free from previous injuries to the back, neck, and upper limb, which may have affected their MAPs. The questionnaire was then reviewed by a principal examiner to determine if they satisfied the inclusion and exclusion criteria at this point. Participants who did not satisfy all criteria at this point were excluded from the study. Participants that met the inclusion criteria underwent a pre-participation screening. The pre-participation screening utilised was identical to that of the pilot study, and involved the completion of components of a shoulder assessment form, developed by the American Shoulder and Elbow Surgeons (Richards et al., 1994) (Appendix E). Participants who did not comply with the criteria after the pre-participation screening were excluded from the study. Upon successful completion of the pre-participation screening, participants' had their weight measured in kilograms (kg), to the nearest 0.1 kg, using a Seca 761 scales (Seca, Birmingham, United Kingdom), and height was measured in metres (m), to the nearest 0.01 m, using a Seca 213 height measure (Seca, Birmingham, United Kingdom). Participants were required to have a BMI of less than thirty, those that did not comply were excluded from the study. Participants who satisfied all inclusion and exclusion criteria progressed onto the familiarisation component of the session.

The familiarisation component was included in the main study following results obtained from the pilot study. It was found that participants demonstrated high variability in MAPs, which was identified by large standard deviations of the onset of muscle activity, with both the SDT and the SCT. As a result of this guide panels (Figure 4.2) were used to standardise the plane of motion for every participant and the familiarisation session would ensure that they were comfortable with the procedure

before the main testing session. The time period between the familiarisation session and the testing session ranged from three to seven days.

4.3.2.2 Testing Session

Participants were required to report to the athletic therapy room for the testing session on their designated day. Participants first completed the SCT, with the use of guide panels, and this was then repeated with sEMG sensors placed on ten muscles, the middle deltoid, upper trapezius and lower trapezius, infraspinatus, and serratus anterior, on both the non-dominant and dominant sides. The upper and lower fibres of the trapezius muscle were chosen along with the lower fibres of the serratus anterior muscle for their role in scapular upward rotation. The infraspinatus muscle was chosen for its role as a GH joint stabiliser and as the only rotator cuff muscle that is accessible for sEMG analysis. Finally, the middle deltoid muscle was chosen for its role as a prime mover of the GH joint. Participants were asked to remove their shirts for the duration of the testing session; this allowed an unobstructed view of their posterior thorax.

4.3.2.2.1 Scapular Control Test

Following the completion of the pilot study a number of recommendations were proposed to reduce variations in MAPs between trials. The SCT was chosen over the SDT as participants were required to remain in a constant GH rotation throughout GH elevation. It was not specified in the SDT guidelines at what stage of elevation participants should adopt the “thumbs up” position therefore it was not standardised for each participant. This element of the SDT may have contributed to the variance seen, specifically in the infraspinatus muscle which performs external rotation at the GH joint. Another recommendation following the pilot study was to include guide panel for participants again due to the high variability in MAPs observed. The guide panels guaranteed that each participant performed GH abduction in the exact same position for each repetition (Figure 4.2).

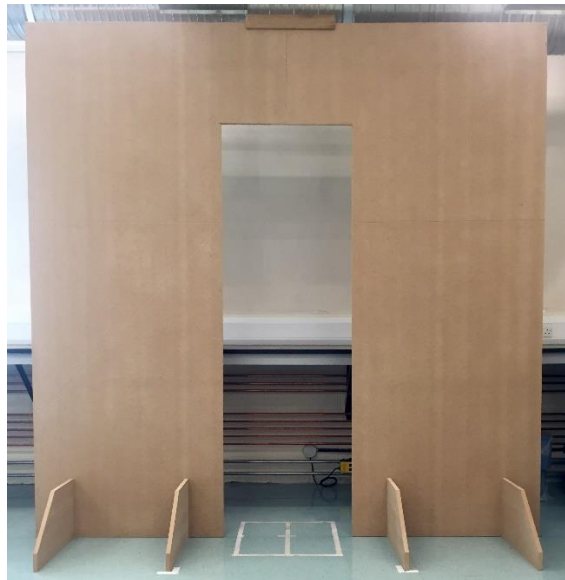


Figure 4.2 Guide panels for the SCT

For the SCT, participants were instructed to stand in a square marked out on the floor, which was between two guide panels, and adopt a normal resting position, with their palms by their sides (Figure 4.3). The square was positioned approximately 2 metres directly in front of a video camera (model HC-V100, Panasonic Corporations, Osaka, Japan) that was mounted level on a tripod, approximately 1.5 metres from the floor. Participants were positioned with their backs to the camera so that they could not be identified on the video recording. Participants were required to perform bilateral active shoulder elevation in the frontal plane, while maintaining their elbows in an extended position, at a rate of 45° per second, while maintaining gentle contact against the guide panels with their index fingers and their hands in neutral rotation (Figure 4.3). A metronome, set at 60 beats per minute, was used to control the participants' speed and the guide panels had three grooves cut at 45° , 90° , and 135° to assist participants in maintaining a constant tempo throughout the movement. Participants paused at maximal elevation for 1 second before returning to the start position, at the same tempo as the elevation component and again maintaining gentle contact against the guide panel with their index fingers. Once participants returned to the start position they relaxed for 4 seconds before beginning the next repetition. This rest period between repetitions was necessary during sEMG analysis, to allow muscle activity to return to a rested state (Lucas et al., 2004). Participants performed 5 repetitions of bilateral active shoulder abduction, which was recorded for analysis by the principal investigator. The video camera was adjusted to capture the posterior aspect of the participants' waist,

head, and elbows throughout the full range of motion. All videos were assessed by the principal investigator, a certified athletic and rehabilitation therapist, with three years clinical experience. The principal investigator was assessed for inter- and intra-tester reliability in the pilot study (Chapter 3).

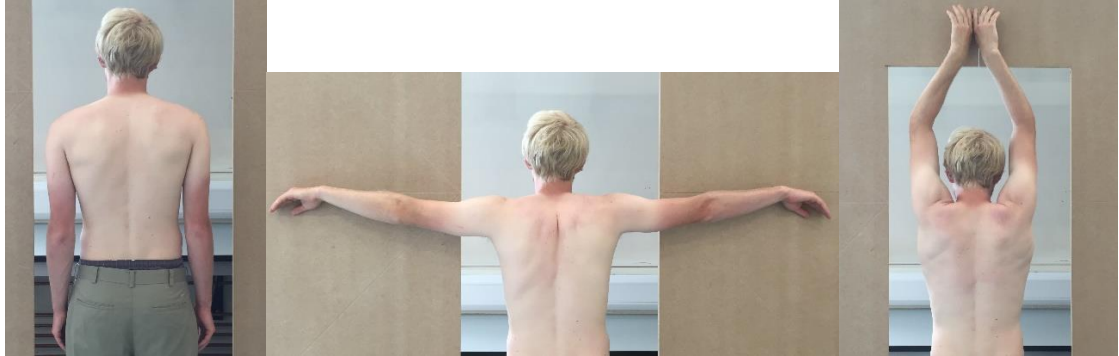


Figure 4.3 Scapular Control Test with guide panels

4.3.2.2.2 Surface Electromyography and Accelerometer Data Collection

Following the SCT, participants were fitted with sEMG sensors on the middle deltoid, upper and lower trapezius, infraspinatus and serratus muscles for four reasons outlined at the start of section 4.3.2.2. Prior to the placement of sEMG sensors participants had their skin prepared in order to provide good sensor to skin contact. This was required to obtain good sEMG signals and to limit artefacts in sEMG data. All locations were first assessed for debris and hair, if necessary the location was shaved with a disposable razor prior to cleaning the skin with an alcohol wipe. Trigno wireless EMG sensors (Delsys Inc., Boston, MA, United States of America) were attached with double sided tape (Figure 4.4) according to methods described by Cram & Criswell (2011) (Appendix F). The Trigno wireless EMG sensors were capable of recording both sEMG signals and tri-axial accelerometer data. Eight of the sensors recorded raw sEMG signals only (upper and lower trapezius, infraspinatus, and serratus anterior muscles on the non-dominant and dominant sides) at 2000 samples per second (Hz). The sensors on the middle deltoid muscles were selected as a dual sensor, which recorded both sEMG signals (1926 Hz) and accelerometer signals (148 Hz). The accelerometer data collected from these sensors were used to identify when movement of the GH joints initiated. This allowed the time at which muscle activity occurred to be normalised to the start of GH joint movement. Once the sensors were in place participants repeated the SCT as previously described. Before the sensors were removed for LTrP examination their exact locations

were recorded, by marking the bottom right corner of each sensor on the participants' skin, ensuring accurate replacement of each sensor following the participants' treatment intervention. Both the SCT and the sEMG analysis were repeated after the participants' specific treatment intervention, for pre-post intervention analysis.



Figure 4.4 Delsys Trigno Wireless EMG sensors & adhesive tape

4.3.2.2.3 Latent Myofascial Trigger Point Examination

Following the removal of the sEMG sensors, participants had their scapular and glenohumeral muscles examined for the presence of LTrPs. Prior to the LTrP examination the principal investigator explained the procedure to the participant again. In accordance with Simons et al. (1999), participants lay on an examination table in a warm and relaxed state. Their position when lying was altered as required for each muscle. The participant's upper limb was positioned to lengthen the muscle being examined to the point of a perceptible increase in resistance to movement. In this position, the normal muscle fibres are said to remain slack while the fibres of any taut bands, containing the LTrPs, are placed under additional tension rendering them more easily distinguishable (Simons et al., 1999). Cross-fibre palpation was then used to identify any taut bands, using "flat palpation" (trapping the LTrP between the principal investigator's fingertips and underlying bone, see Figure 4.5) or using "pincer palpation" (trapping the LTrP between the principal investigator's thumb and fingers, see Figure 4.5) (Simons et al., 1999).



Figure 4.5 Palpation techniques: flat palpation & pincer palpation

If a taut band was identified, the principal investigator then palpated along the taut band searching for a slightly enlarged point or the “focus” of the contraction. When the principal investigator had identified this point, the participant was asked if the point was tender when compressed manually. In the event of an affirmative response, a pressure pain threshold reading was taken of the point using an algometer (Figure 4.6), using the procedure validated by Fisher (1987). The principal investigator applied a slow, gradual increase in pressure with the algometer. Participants were instructed to say “now” when they first began to feel the pressure change to discomfort. The application of pressure was ceased immediately and the peak pressure reading was recorded as the pressure pain threshold. Pressure pain threshold measurements were repeated three times and the mean recorded (Appendix L). All pressure pain threshold measurements were taken in quick succession (within approximately 30 seconds) to avoid deactivating the LTrP by sustained pressure (manual release technique). The position of each LTrP was documented on an enlarged body diagram (Appendix L). Participants were also asked if they experienced any referred sensations elsewhere in their body. Following this, snapping palpation was applied in an attempt to elicit a local twitch response. The presence of referred sensations and/or a local twitch response were used as additional confirmation of the presence of an LTrP.



Figure 4.6 Digital algometer

4.3.2.2.4 Intervention

Following LTrP examination participants were randomly allocated into one of the three intervention groups, manual release, dry needling, or placebo dry needling, by choosing from a selection of shuffled envelopes containing the group titles. During all interventions the documentation of the locations of LTrPs during the examination were used to relocate each LTrP. The same process used for the identification of the LTrPs was again implemented to relocate the LTrP prior to treatment. The principal investigator who implemented all interventions had prior training in both TrP manual release and dry needling. The TrP manual therapy training was conducted as part of their undergraduate education and involved 16 hours over 2 days. The TrP dry needling course was a continual professional development (CPD) course run by the David G Simons Academy and involved a total of 48 hours of training, over two 3 day modules.

4.3.2.2.5 Manual Release

Prior to LTrP manual release the principal investigator explained the procedure to the participant again and they were encouraged to give feedback to and maintain communication with the principal investigator throughout the treatment. The manual release technique followed the methodology described by Fryer and Hodgson (2005), who demonstrated that the technique was effective at significantly increasing the pressure pain thresholds of participants compared to a control group. Participants were treated lying down and positioned in a suitable manner to access the muscles being treated. Similar to the examination, the muscle being treated was positioned optimally

to allow skilled palpation of any taut bands and the LTrPs. Verbal communication with the participant was maintained to assess their response to the procedure. Upon locating a LTrP the principal investigator applied a gradual increase in pressure, with the use of the digital algometer, until the participant reported a moderate but easily tolerable discomfort. A subjective value of 7 on a 0-10 cm visual analogue scale (VAS) was used to identify this point (Appendix M). The principal investigator sustained this pressure until the tenderness had reduced. Once the participant reported that the pain decreased to a value of 3 or 4 out of 10 on the VAS, the principal investigator slowly increased the pressure to restore the perceived discomfort to the original value of 7 out of 10. The second pressure reading was then recorded. This process was repeated for each identified LTrP.

4.3.2.2.6 Dry Needling

Prior to commencing LTrP dry needling the principal investigator washed their hands and put on nitrile examination gloves. The principal investigator then explained the procedure to the participant again and they were encouraged to give feedback to and maintain communication with the principal investigator throughout the treatment. During the treatment the participant was positioned in a stable position to minimise movement of their body during the procedure. Similar to the LTrP examination and the LTrP manual release technique, the targeted muscle was positioned to allow skilled palpation of any taut bands and LTrPs. Before the treatment of any LTrPs with dry needling, the skin was cleaned with an alcohol based disinfectant solution. Once an LTrP was located it was immobilised with either flat palpation or pincer grip, depending on the muscle being treated (Figure 4.5). Dry needles were inserted perpendicularly through the skin and moved forward to the location of the LTrP (Dommerholt and de las Peñas, 2013). Once a local twitch response was elicited the dry needle was removed. A guide tube aided the insertion of all needles (0.3 mm x 30 mm). In order to minimise any discomfort, the participant may have felt on insertion of the dry needle, a degree of pressure was applied to the skin with the guide tube prior to each dry needle being inserted. A dynamic needling technique was used with a slow and steady motion to locate the LTrP and elicit a LTR. Dynamic needling involves a deliberate lancing motion in and out of the muscle (Dommerholt and de las Peñas, 2013). Treatment of the LTrP was ceased when no more LTRs were observed. The area treated was then lightly

compressed for 10-20 seconds following the withdrawal of the needle, to ensure haemostasis.

4.3.2.2.7 Placebo Dry Needling

Participants who were randomised into the placebo group received the same explanation of the treatment as the dry needling group. LTrPs were located in the same manner as the genuine dry needling treatment however; a “Streitberger” needle was used to perform the placebo treatment. This needle looked identical to a typical dry needling needle but had a blunted tip, which did not break through the skin, and a shaft that retracted into the dry needle’s handle (Figure 4.7). The placebo dry needling treatment followed the same process as the genuine dry needling process but as the needle did not penetrate the skin the LTrP was not treated and no local twitch response was elicited.

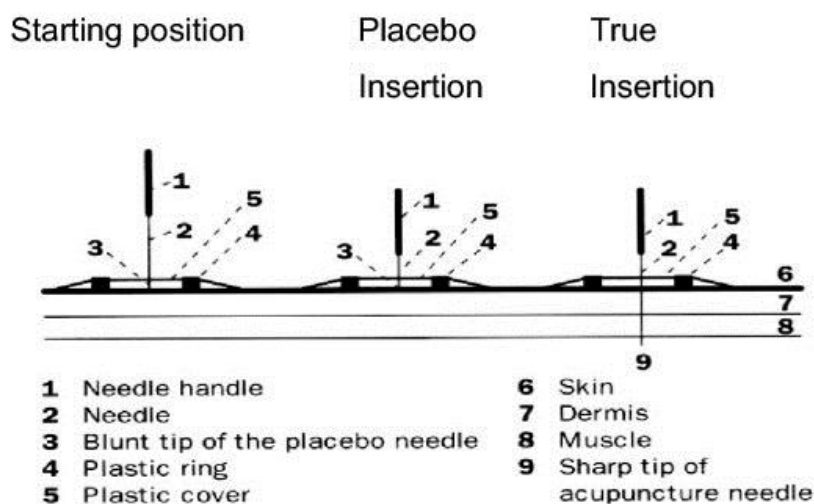


Figure 4.7 Streitberger Placebo Needle [reproduced from Streitberger & Kleinhenz (1998)]

4.3.2.2.8 Post-intervention Re-testing

After participants received their treatments they repeated the SCT and sEMG data collection. The markings that were applied to the participants’ skin before the sEMG sensors were removed were used to ensure that they were positioned in the same place for the post-intervention sEMG data collection.

4.4 Data Analysis

4.4.1 Scapular Control Test Video Analysis

The principal investigator, who completed the inter- and intra-tester reliability testing in the pilot study (Chapter 3) assessed all participants' pre- and post-intervention videos. They therefore underwent standardised training via a self-directed slide presentation on the SCT as outlined in section 3.4.1. The principal investigator viewed all 60 pre-intervention videos of the participants and independently rated the test movement for each shoulder according to the rating guidelines (Table 4.3). The principal investigator viewed and rated all post-intervention videos of the participants 3 days later. Statistical analysis was then conducted to determine whether a significant change occurred within and between intervention groups.

Table 4.3 Scoring system for the Scapular Control Test

Scapular Control Test Rating Guidelines			
Score	Winging	Scapular Control when lifting/lowering	Symmetry
No issue (0)	Medial border of the scapulae flat on the thorax	Scapulae moves in a controlled manner with no shanking or abnormal scapulae positioning	Scapulae move in synchrony throughout the full range of motion with no lagging behind or speeding of a single scapula in relation to the corresponding scapula during lifting and lowering
Slight issue (1)	Medial border visible, with slight sulcus present	Slight loss of control, not consistently present	Slight asymmetry present during lifting and/or lowering
Moderate issue (2)	Medial border visible, with moderate sulcus present	Loss of control that is consistently present (each rep), but which is regained during the movement	Moderate asymmetry present during lifting and/or lowering
Severe issue (3)	Medial border visible, with severe sulcus present	Loss of control that is consistently present (each rep) and does not regain control at any point	Severe asymmetry present during lifting and/or lowering

4.4.2 Surface Electromyography and Accelerometer Data Analysis

All sEMG and accelerometer data was collected and analysed as it was in the pilot study and is detailed in section 3.4.2. The data collected from the sEMG analysis was then used to determine whether there were within and between group differences in participants'

MAPs pre- and post-intervention. Participants were grouped into their respective groups depending on the intervention they received, dry needling, manual release, or placebo dry needling. Both the non-dominant and dominant shoulders of participants were examined for MAPs pre- and post-intervention.

4.4.3 Statistical Analysis

All statistical analysis, excluding the manual calculations mentioned below, were performed using IBM SPSS statistics version 22 (IBM, New York, United States of America). A significance level of at $p < 0.05$ was set for all tests, unless stated otherwise.

4.4.3.1 Scapular Control Test Statistical Analysis

As the SCT involved a four point ordinal scale with seven criteria, which could not be amalgamated, non-parametric tests were utilised for the statistical analysis (McCrum-Gardner, 2008). Prior to carrying out any statistical analysis the data was examined using histograms for symmetry of the distributions of differences between all related groups. A Wilcoxon Signed Rank Test was employed for each intervention group, to determine whether there was a within-subjects difference in participants' SCT scores for each of the seven criteria pre- and post-intervention. The treatment groups, dry needling, manual release, and placebo dry needling, were selected as the independent variable and each of the seven criteria in the SCT were set as the dependant variable. The within-subjects factor for this analysis was the two time points, pre- and post-intervention. The effect size for each intervention group was then calculated using Equation 4.2, where N was equal to the total number of observations over the two time points (Pallant, 2010). The Cohen (1988) criteria was then used to determine the classification of effect sizes (Table 4.4).

$$r = \frac{z}{\sqrt{N}}$$

Equation 4.2 Effect size calculation for Wilcoxon Signed Rank Test

Table 4.4 Cohen's r effect size criteria

Effect Size	r
Trivial	< 0.10
Small	> 0.10
Medium	> 0.30
Large	> 0.50

The Kruskal-Wallis Test was utilised to determine whether there was a between-subjects difference in participants' SCT scores for any of the seven criteria, both pre- and post-intervention. The treatment groups were selected as the independent variables and each of the seven criteria were set as the dependant variables.

4.4.3.2 Surface Electromyography and Accelerometer Statistical Analysis

Prior to carrying out any statistical analysis, all data was examined for the assumption of normality, via Shapiro-Wilk's test, normal Q-Q and detrended Q-Q plots, and box plots. Ten of the sixty assessments violated the Shapiro-Wilk's test for normality however, following assessment of the normal Q-Q and detrended Q-Q plots, box plots, and due to the robustness of analysis of variance (ANOVA) tests to violations of normality (Schmider et al., 2010), the data was deemed appropriated for analysis via an ANOVA test. A mixed between-within ANOVA, was employed for each muscle to determine the effect of each intervention on altering MAPs pre- and post-intervention. The intervention groups were set as the independent variables and the time of muscle activations were set as the dependant variables. The within-subjects factor for this analysis was the two time points, pre- and post-intervention. All muscles were examined for equal variances, using Levene's Test of Equality of Error Variances, and for homogeneity of intercorrelations, using Box's M Test with a significance level set at $p < 0.001$ due to its high sensitivity. Wilk's Lambda was used to assess if there was an interaction effect between the interventions. If the interaction between groups was not significant the main effects for each intervention group was then examined, again using Wilk's Lambda. The Cohen (1988) criteria was used to determine the classification of the Partial Eta Squared effect sizes (Table 4.5).

Table 4.5 Partial Eta Squared effect size criteria

Effect Size	Partial Eta Squared
Trivial	< 0.01
Small	> 0.01
Medium	> 0.06
Large	> 0.14

4.5 Results

4.5.1 Prevalence of Latent Trigger Points

All groups demonstrated similar LTrPs prevalence in all muscles (Table 4.6). The infraspinatus and upper trapezius muscles demonstrated the highest prevalence of LTrPs, with 88% of participants possessing them in both non-dominant muscles, and 85% and 83% respectively in the dominant muscles.

Table 4.6 Latent Myofascial Trigger Point prevalence per intervention group: mean (SD)

Group	ND MD	D MD	ND TU	D TU	ND TL	D TL	ND Inf	D Inf	ND SA	D SA	Total
Dry Needling	0.0 (0.0)	0.0 (0.0)	1.2 (0.5)	1.4 (0.7)	0.5 (0.7)	0.7 (0.7)	1.3 (0.8)	1.2 (0.6)	0.6 (0.6)	0.7 (0.6)	7.5 (3.5)
Manual Release	0.0 (0.0)	0.0 (0.0)	1.0 (0.6)	1.1 (0.7)	0.4 (0.5)	0.4 (0.5)	1.4 (1.0)	1.1 (0.7)	0.4 (0.5)	0.8 (0.6)	6.5 (3.2)
Placebo Dry Needling	0.0 (0.0)	0.1 (0.2)	1.1 (0.6)	1.2 (0.8)	0.5 (0.6)	0.4 (0.6)	1.2 (0.7)	1.0 (0.6)	0.3 (0.5)	0.4 (0.6)	6.0 (3.0)
Total	0.0 (0.0)	0.0 (0.1)	1.1 (0.6)	1.2 (0.7)	0.4 (0.6)	0.5 (0.6)	1.3 (0.8)	1.1 (0.6)	0.4 (0.5)	0.6 (0.6)	6.6 (3.2)
% of participants	0	2	88	83	38	42	88	85	40	52	100

ND = Non-dominant; D = Dominant; MD = Middle Deltoid; TU = Trap Upper; TL = Trapezius Lower; Inf = Infraspinatus; SA = Serratus Anterior; % of participants = percentage of participants whom presented with at least one LTrP in the muscle.

4.5.2 Prevalence of Scapular Dyskinesis

Table 4.7 illustrates the distribution of dyskinesia within each of the intervention groups prior to treatment.

Table 4.7 Prevalence of scapular dyskinesis in each group pre-intervention

Group	Non-dominant		Dominant	
	Normal	Dyskinesis	Normal	Dyskinesis
Dry Needling	12	8	15	5
Manual Release	8	12	12	8
Placebo Dry Needling	11	9	11	9

4.5.3 Scapular Control Test

The within-subjects results of the Wilcoxon Signed Rank Tests and effect size calculation conducted for each intervention group (dry needling, manual release, and placebo dry needling) are displayed in Table 4.8, Table 4.9, and Table 4.10. Only the symmetry between both scapulae component of the SCT in the placebo dry needling group achieved statistical significance ($z = -2.00$, $p = 0.046$), with a medium effect size ($r = 0.32$). All other components in all three intervention groups did not reach statistical significance and presented with small effect sizes.

Table 4.8 Dry needling group within-subjects effects

Dry Needling Group	z-score	p-value	r-value
ND Winging	0.00	1.00	0.00
D Winging	0.00	1.00	0.00
ND Control of Scapula when Lifting	0.00	1.00	0.00
D Control when Lifting	0.00	1.00	0.00
ND Control of Scapula when Lowering	0.00	1.00	0.00
D Control of Scapula when Lowering	-0.58	0.56	0.09
Symmetry between both Scapulae	-1.00	0.32	0.16

ND = Non-dominant; D = Dominant

Trivial effect: $r < 0.10$; Small effect: $r > 0.10$; Medium effect: $r > 0.30$; Large effect: $r > 0.50$

Table 4.9 Manual release group within-subjects effects

Manual Release Group	z-score	p-value	r-value
ND Winging	0.00	1.00	0.00
D Winging	-1.00	0.32	0.16
ND Control of Scapula when Lifting	0.00	1.00	0.00
D Control when Lifting	0.00	1.00	0.00
ND Control of Scapula when Lowering	-1.00	1.32	0.16
D Control of Scapula when Lowering	-1.14	0.16	0.22
Symmetry between both Scapulae	-0.58	0.56	0.09

ND = Non-dominant; D = Dominant

Trivial effect: $r < 0.10$; Small effect: $r > 0.10$; Medium effect: $r > 0.30$; Large effect: $r > 0.50$

Table 4.10 Placebo dry needling group within-subjects effects

Placebo Dry Needling Group	z-score	p-value	r-value
ND Winging	-1.00	0.32	0.16
D Winging	-1.00	0.32	0.16
ND Control of Scapula when Lifting	0.00	1.00	0.00
D Control when Lifting	0.00	1.00	0.00
ND Control of Scapula when Lowering	0.00	1.00	0.00
D Control of Scapula when Lowering	0.00	1.00	0.00
Symmetry between both Scapulae	-2.00	0.05*	0.32‡

ND = Non-dominant; D = Dominant; * = $p < 0.05$; ‡ = medium effect size

Trivial effect: $r < 0.10$; Small effect: $r > 0.10$; Medium effect: $r > 0.30$; Large effect: $r > 0.50$

The between-subjects results of the Kruskal-Wallis Tests are displayed in Table 4.11. The Kruskal-Wallis Tests did not reveal any statistically significant differences across the three intervention groups in any of the seven components of the SCT, either pre- or post-intervention.

Table 4.11 Between-subject effects pre- and post-intervention

	Pre-intervention		Post-intervention	
	X ²	p-value	X ²	p-value
ND Winging	(2) 2.53	0.28	(2) 2.05	0.36
D Winging	(2) 4.48	0.11	(2) 4.05	0.13
ND Control of Scapula when Lifting	(2) 0.00	1.00	(2) 0.00	1.00
D Control when Lifting	(2) 0.00	1.00	(2) 0.00	1.00
ND Control of Scapula when Lowering	(2) 2.01	0.37	(2) 1.12	0.57
D Control of Scapula when Lowering	(2) 0.89	0.64	(2) 1.66	0.44
Symmetry between both Scapulae	(2) 0.52	0.81	(2) 1.06	0.59

4.5.4 Onset of Muscle Activation

Mixed between-within ANOVA's were conducted to assess the impact of the three interventions (dry needling, manual release, and placebo dry needling) on participants' onset of muscle activity in all five muscles on the non-dominant and dominant sides. The results of the mixed between-within ANOVA's are presented in Table 4.12. Data for each intervention group's mean muscle activation times, along with standard deviations and 95% confidence intervals, are presented in Table 4.13 to Table 4.15, and illustrated in Figure 4.9 to Figure 4.14. Overall the results of the mixed between-within ANOVAs demonstrated that there was only a significant interaction effect between groups for one muscle and no significant difference in the effectiveness of any of the three interventions. There were two other muscles that showed subtle differences, discussed below.

There was a significant interaction between intervention groups and time (pre- and post-intervention) and a moderate effect size in the non-dominant lower trapezius muscles (Wilks' Lambda = 0.89, $F(2, 55) = 3.36$, $p = 0.04$, partial eta squared = 0.11). This was due to the earlier mean time of muscle activation seen in the placebo dry needling group (pre-intervention = 1.205 seconds [95% CI: 0.930, 1.479] and post-intervention = 0.973 seconds [0.695, 1.252]) in comparison to the later mean time of muscle activation seen in the dry needling group (pre-intervention = 1.230 seconds [0.956, 1.505] and post-intervention = 1.304 seconds [1.025, 1.582]) (Figure 4.8). However, there was no significant main effect for time (Wilks' Lambda = 0.97, $F(1, 55) = 1.66$, $p = 0.20$, partial eta squared = 0.03). The main effect between groups was not significant either ($F(2, 55)$

= 0.45, $p = 0.58$, partial eta squared = 0.02), suggesting no significant difference in the effectiveness of the three interventions.

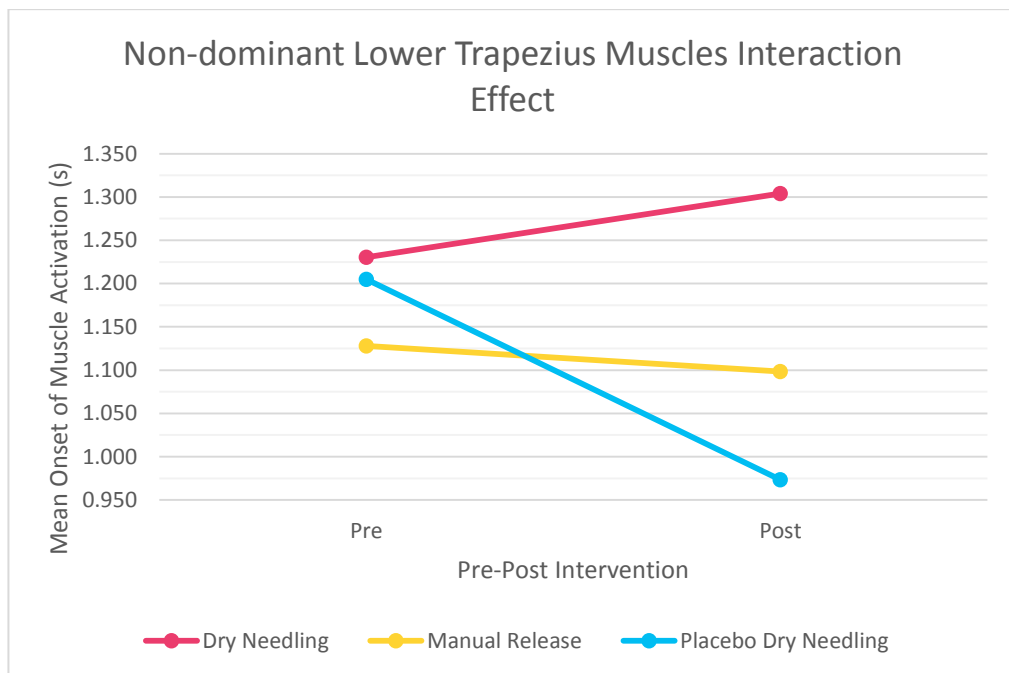


Figure 4.8 Non-dominant lower trapezius muscles interaction effect

There was no significant interaction effect between intervention groups and time (pre- and post-intervention) in the dominant lower trapezius muscles (Wilks' Lambda = 0.98, $F(2, 55) = 0.56$, $p = 0.57$, partial eta squared = 0.02). However, there was a main effect for time with statistical significance found along with a large effect size (Wilks' Lambda = 0.85, $F(1, 55) = 9.56$, $p = 0.003$, partial eta squared = 0.15), with all groups demonstrating an earlier time of muscle activation post-intervention (Figure 4.10, Figure 4.12, and Figure 4.14). However, the main effect between groups was not significant ($F(2, 55) = 0.03$, $p = 0.97$, partial eta squared < 0.01), suggesting no difference in the effectiveness between of the three groups.

There was no significant interaction effect between intervention groups and time (pre- and post-intervention) in the non-dominant infraspinatus muscles (Wilks' Lambda = 0.94, $F(2, 57) = 1.79$, $p = 0.18$, partial eta squared = 0.06). In addition, there was no significant main effect for time however, there was a large effect size (Wilks' Lambda = 0.98, $F(1, 57) = 0.92$, $p = 0.34$, partial eta squared = 0.16), with the manual release and placebo dry needling groups demonstrating a later mean muscle activation time post-intervention and dry needling establishing an earlier mean muscle activation time. While

the main effect between groups was not significant, there was a medium effect size found ($F(2, 57) = 2.41, p = 0.10, \text{partial eta squared} = 0.08$) Table 4.12 Results of the mixed between-within ANOVAs

Muscle	Interaction between intervention and time				Time				Intervention Groups		
	Wilks' Lambda	F-value	P-value	Partial Eta Squared	Wilks' Lambda	F-value	P-value	Partial Eta Squared	F-value	P-value	Partial Eta Squared
ND MD	0.99	0.18	0.84	0.01	0.97	1.84	0.18	0.03	0.16	0.85	0.01
D MD	0.93	2.23	0.12	0.07	0.99	0.39	0.54	0.01	0.36	0.70	0.01
ND TU	0.91	2.76	0.07	0.09	0.99	0.53	0.47	0.01	0.51	0.60	0.02
D TU	1.00	0.12	0.89	0.00	1.00	0.22	0.64	0.00	1.27	0.28	0.04
ND TL	0.89	3.36	0.04*	0.11	0.97	1.66	0.20	0.03	0.54	0.58	0.02
D TL	0.98	0.56	0.57	0.02	0.85	9.56	0.00*	0.15†	0.03	0.97	0.00
ND Inf	0.94	1.79	0.18	0.06	0.98	0.92	0.34	0.16†	2.41	0.10	0.08
D Inf	0.97	0.77	0.47	0.03	0.98	0.99	0.32	0.02	0.68	0.51	0.02
ND SA	1.00	0.14	0.87	0.01	1.00	0.00	0.99	0.00	0.40	0.53	0.02
D SA	0.97	0.91	0.41	0.03	1.00	0.15	0.70	0.00	0.74	0.48	0.03

ND = Non-dominant; D = Dominant; MD = Middle Deltoid; TU = Trap Upper; TL = Trapezius Lower; Inf = Infraspinatus; SA = Serratus Anterior; † = Large Partial Eta Squared Effect Size; * = $p < 0.05$

Partial Eta Squared= Trivial: < 0.01 ; Small: > 0.01 ; Medium: > 0.06 ; Large: > 0.14

Table 4.13 Dry needling group MAPs: mean (SD) time of muscle activation pre- & post-intervention

Muscle Side	MD		TU		TL		Inf		SA	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
ND (s)	0.566 (0.487)	0.548 (0.419)	0.643 (0.520)	0.496 (0.537)	1.230 (0.430)	1.304 (0.424)	0.350 (0.626)	0.296 (0.661)	1.139 (0.603)	1.117 (0.548)
95% CI	0.383, 0.749	0.367, 0.729	0.426, 0.859	0.273, 0.718	0.956, 1.505	1.025, 1.582	0.086, 0.615	0.036, 0.556	0.849, 1.428	0.834, 1.399
n	20		20		19		20		20	
D (s)	0.550 (0.330)	0.575 (0.331)	0.658 (0.466)	0.605 (0.485)	1.311 (0.554)	1.154 (0.543)	0.423 (0.936)	0.594 (0.820)	1.023 (0.777)	1.113 (0.728)
95% CI	0.369, 0.731	0.407, 0.743	0.377, 0.938	0.395, 0.815	1.006, 1.615	0.831, 1.478	0.015, 0.831	0.241, 0.947	0.688, 1.359	0.820, 1.406
n	20		20		19		20		19	

MD = Middle Deltoid; TU = Trapezius Upper; TL = Trapezius Lower; Inf = Infraspinatus; SA = Serratus Anterior; ND = Non-dominant; D = Dominant; s = seconds; 95% CI = 95% Confidence Interval; n = number per group; minus “-“ = muscle activated before movement

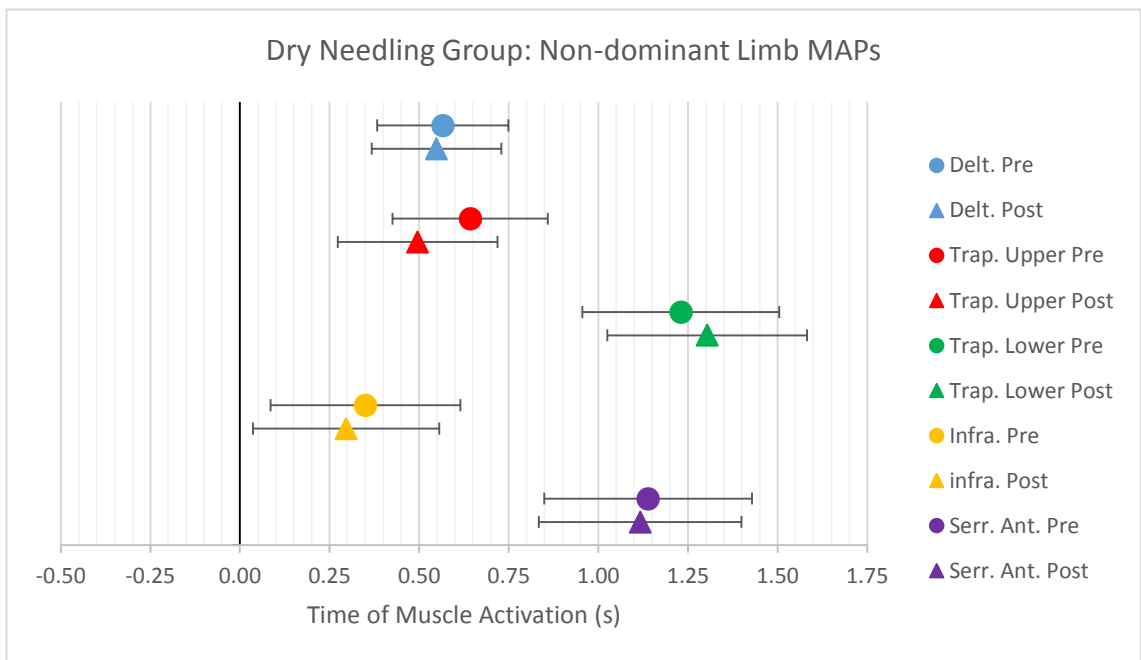


Figure 4.9 Dry needling group MAPs: non-dominant limb mean and 95% confidence interval times of muscle activation pre- & post-intervention

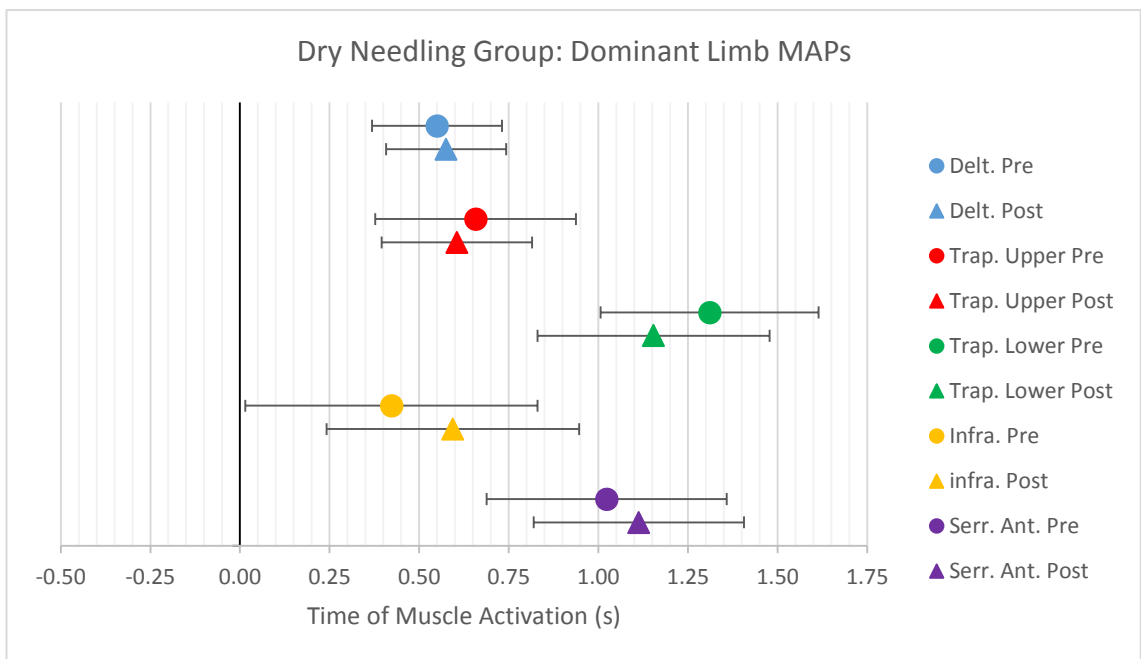


Figure 4.10 Dry needling group MAPs: dominant limb mean and 95% confidence interval times of muscle activation pre- & post-intervention

Table 4.14 Manual release group MAPs: mean (SD) time of muscle activation pre- & post-intervention

Muscle Side	MD		TU		TL		Inf		SA	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
ND (s)	0.605 (0.398)	0.543 (0.421)	0.617 (0.494)	0.705 (0.519)	1.128 (0.745)	1.098 (0.694)	0.114 (0.604)	0.166 (0.627)	1.272 (0.657)	1.260 (0.684)
95% CI	0.422, 0.788	0.362, 0.723	0.400, 0.833	0.482, 0.927	0.860, 1.396	0.827, 1.370	-0.150, 0.379	-0.094, 0.425	0.982, 1.561	0.977, 1.543
n	20		20		20		20		20	
D (s)	0.592 (0.439)	0.618 (0.405)	0.650 (0.448)	0.608 (0.417)	1.245 (0.682)	1.157 (0.705)	0.693 (0.973)	0.697 (0.886)	1.045 (0.704)	0.933 (0.511)
95% CI	0.411, 0.773	0.450, 0.785	0.370, 0.930	0.398, 0.817	0.941, 1.549	0.834, 1.481	0.251, 1.135	0.315, 1.080	0.718, 1.372	0.647, 1.219
n	20		20		20		17		20	

MD = Middle Deltoid; TU = Trapezius Upper; TL = Trapezius Lower; Inf = Infraspinatus; SA = Serratus Anterior; ND = Non-dominant; D = Dominant; s = seconds; 95% CI = 95% Confidence Interval; n = number per group; minus “-“ = muscle activated before movement

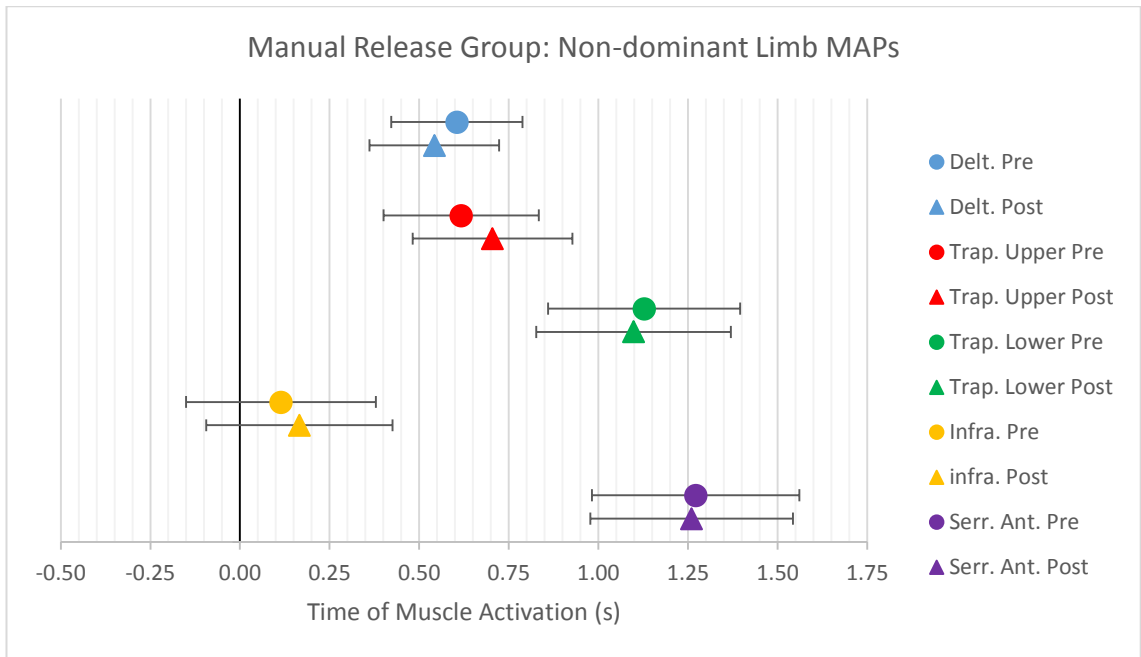


Figure 4.11 Manual release group MAPs: non-dominant limb mean and 95% confidence interval times of muscle activation pre- & post-intervention

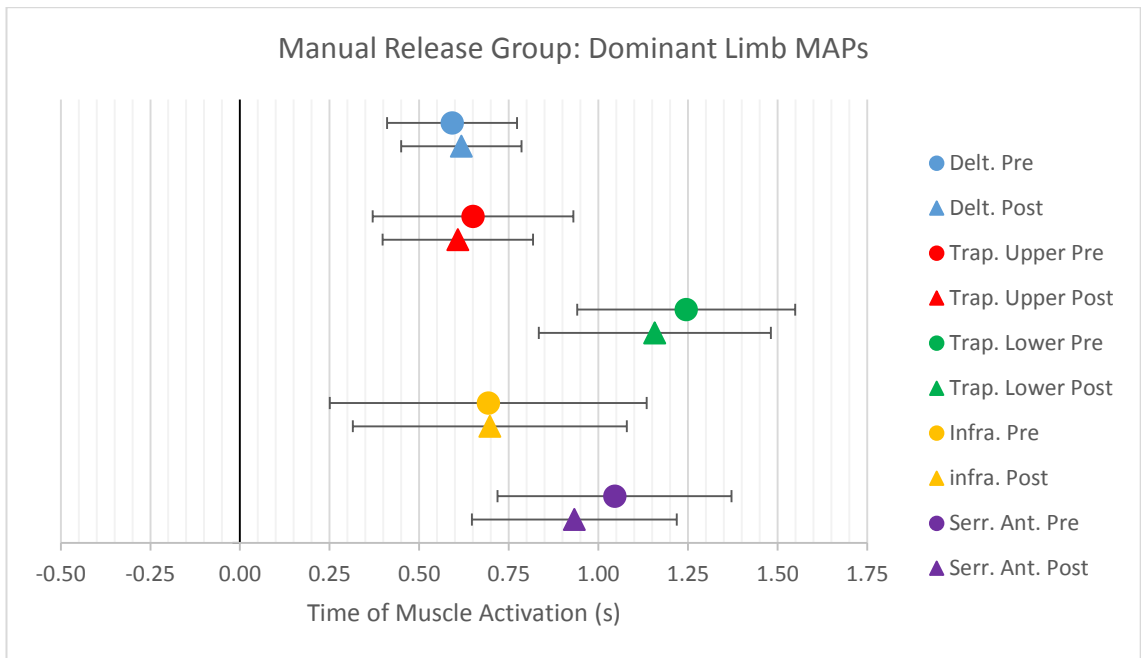


Figure 4.12 Manual release group MAPs: dominant limb mean and 95% confidence interval times of muscle activation pre- & post-intervention

Table 4.15 Placebo dry needling group MAPs: mean (SD) time of muscle activation pre- & post-intervention

Muscle Side	MD		TU		TL		Inf		SA	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
ND (s)	0.537 (0.325)	0.478 (0.370)	0.529 (0.432)	0.499 (0.429)	1.205 (0.566)	0.973 (0.658)	-0.123 (0.539)	-0.022 (0.423)	1.032 (0.677)	1.067 (0.655)
95% CI	0.353, 0.720	0.297, 0.659	0.313, 0.746	0.277, 0.722	0.930, 1.479	0.695, 1.252	-0.387, 0.142	-0.282, 0.238	0.742, 1.321	0.784, 1.350
n	20		20		19		20		20	
D (s)	0.557 (0.436)	0.452 (0.383)	0.404 (0.870)	0.416 (0.500)	1.286 (0.731)	1.071 (0.858)	0.367 (0.809)	0.380 (0.631)	0.849 (0.707)	0.798 (0.660)
95% CI	0.376, 0.739	0.284, 0.619	0.124, 0.684	0.206, 0.626	0.989, 1.582	0.755, 1.386	-0.063, 0.796	0.008, 0.751	0.522, 1.176	0.513, 1.084
n	20		20		20		18		20	

MD = Middle Deltoid; TU = Trapezius Upper; TL = Trapezius Lower; Inf = Infraspinatus; SA = Serratus Anterior; ND = Non-dominant; D = Dominant; s = seconds; 95% CI = 95% Confidence Interval; n = number per group; minus “-“ = muscle activated before movement

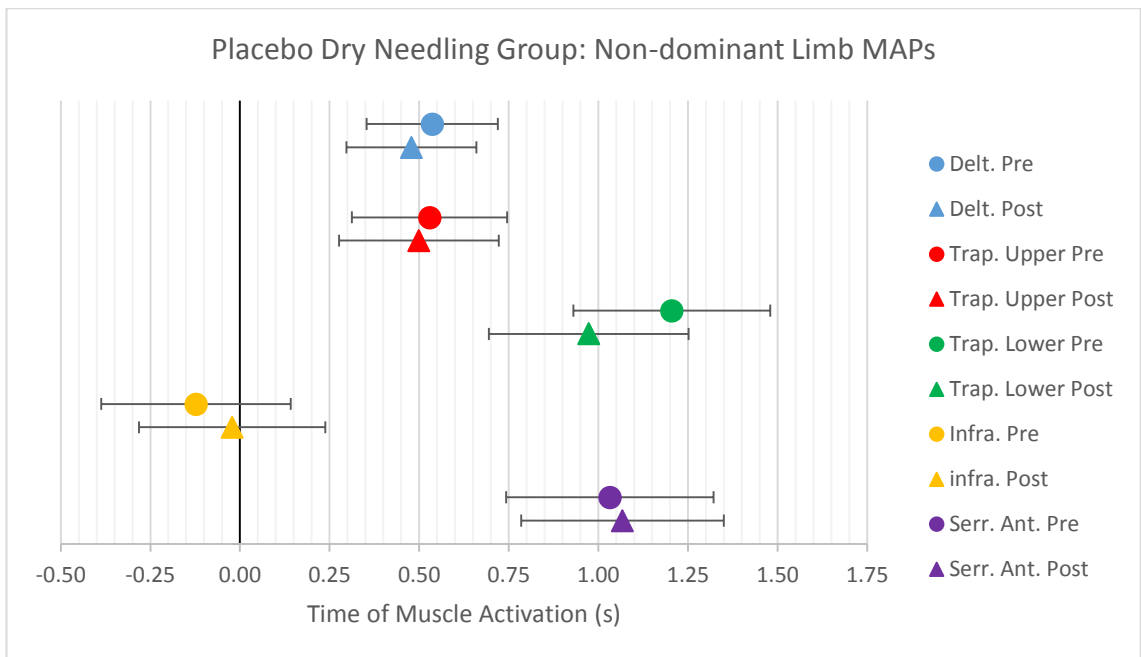


Figure 4.13 Placebo dry needling group MAPs: non-dominant limb mean and 95% confidence interval times of muscle activation pre- & post-intervention

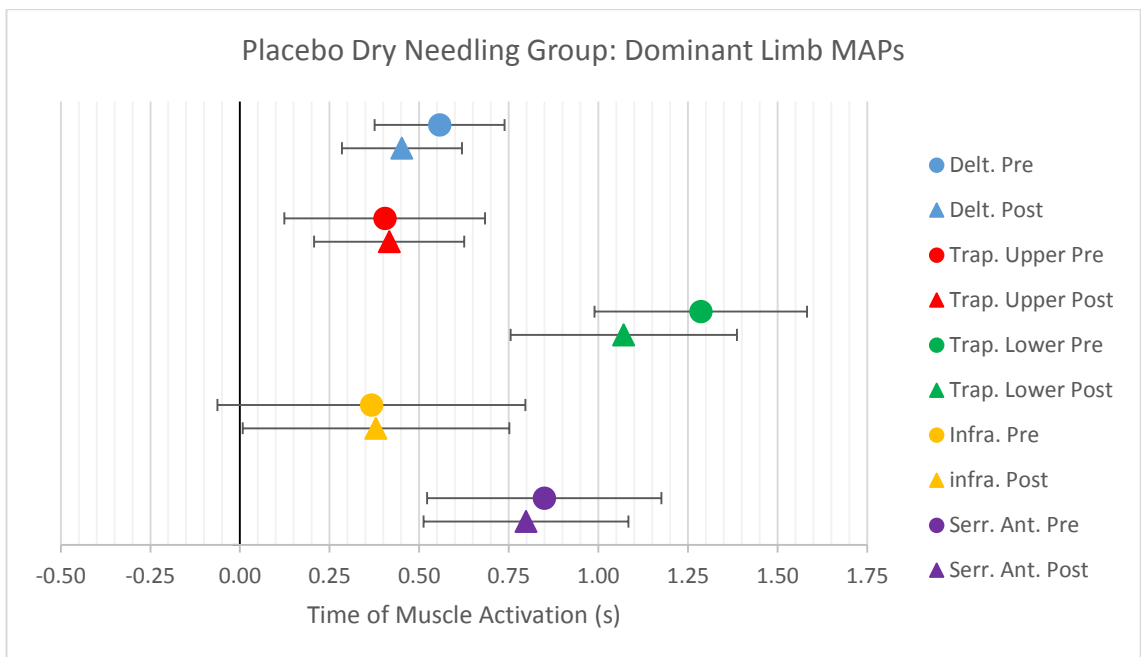


Figure 4.14 Placebo Dry needling group MAPs: dominant limb mean and 95% confidence interval times of muscle activation pre- & post-intervention

4.6 Discussion

Recent research investigating LTrPs suggests that these minor, subclinical neuromuscular lesions also have a negative effect on the muscle prior to their conversion into ATrPs (Celik and Mutlu, 2013). The present study investigated whether LTrP dry needling was an effective treatment at improving, one of the proposed negative effects of LTrPs, MAP alterations (Ge et al., 2008; Lucas et al., 2010, 2004).

4.6.1 Scapular Control Test

The SCT was implemented to determine whether any visual alterations that may occur following the treatment of LTrPs could be observed visually using a clinical test. The results of the SCT demonstrated that no visual changes could be observed in the motion of the scapula after any of the interventions. Only the between-subjects effects in the placebo dry needling group demonstrated a significant difference ($p = 0.046$), with a medium effect size ($r = 0.32$), for the symmetry between both scapulae component of the test.

This was unexpected as the LTrPs in this group did not receive a real treatment. The changes in this component of the SCT in the placebo dry needling group may be due to, slight natural variation in participants' symmetry between both scapulae before and after the intervention, reliability issues in this component from the principal investigator's perspective, or a true placebo effect. As demonstrated in the intra-tester reliability study carried out in the pilot study (Chapter 3), the symmetry between both scapulae component of the SCT demonstrated the lowest ICC score of all seven components and with the widest 95% CI, however it still achieved an excellent reliability classification (ICC = 0.91 [95% CI: 0.81, 0.96]). It is also important to note that previous research reported that asymmetry between scapulae did not indicate dysfunction (Koslow et al., 2003), thus isolated changes in this component alone may not be noteworthy. No data was collected on whether participants had experience of any of the interventions or whether they had positive or negative expectations of their selected treatment. Patients' expectations on the benefits of treatments have been shown to effect the outcome of the treatment (Kalauokalani et al., 2001), thus it is not known whether either prior experience of the treatment or perceived benefits could have accounted for the changes observed in the placebo group.

The six other components in the placebo dry needling group did not reach significance and were associated with only trivial or small effect sizes. All components of the SCT in the dry needling and the manual release groups were similar to those from the six non-significant components in the placebo dry needling group, with no significant differences and trivial or small effect sizes. Similarly the between-subjects effects, both pre- and post- intervention, did not yield any significant changes, highlighting that the subtle changes observed, if any, were consistent across all three groups.

The use of scapular dyskinesis tests is common place in the clinical setting. However, there is a paucity of research on the ability of either treatment interventions or exercise programmes at altering scapular motion. Research in this area has typically focused on the effectiveness of treatments at improving pain (Balci et al., 2016; Ellenbecker et al., 2008), muscle strength (Ellenbecker et al., 2008), and range of motion (Balci et al., 2016; Ellenbecker et al., 2008), or how specific exercises increase EMG activity of targeted muscles (Cools et al., 2007). Balci et al. (2016) did assess the ability of proprioceptive neuromuscular facilitation (PNF) techniques and classic shoulder exercises, such as wand, pendulum, and isolated scapular exercises, at altering the scapula's motion using the lateral scapular slide test. However, this was only a 2-D assessment method and did not assess scapular motion during a functional task.

The results of the present study, using the SCT, indicate that the treatment of LTrPs with dry needling and manual release is not effective, as there is no significant or clinically relevant change in observed scapular motion following their use. However, it should be noted that LTrPs may not cause scapular dyskinesis, as their treatment does not result in any significant or clinically relevant changes in scapular motion. Finally, it should be considered that visual tests such as the SCT may not be capable of determining subtle changes in MAPs following the treatment of LTrPs with dry needling and manual release.

4.6.2 Onset of Muscle Activation

The aim of the sEMG analysis was to determine whether any potential change in MAPs could be observed following the treatment of LTrPs with an objective measure. Similar to the SCT results, treatment of LTrPs with two real treatments, dry needling and manual release, did not demonstrate a significant change in the MAPs of key muscles of the shoulder complex when compared to a placebo treatment.

These findings are in direct contrast to those reported by Lucas et al. (2010, 2004) who showed that the presence of LTrPs in scapular rotator muscles caused an alteration in MAPs and that a treatment intervention to remove the LTrPs normalised the MAPs in line with a control group, whom did not have any LTrPs present. Lucas et al. (2010, 2004) concluded that where LTrPs existed, participants demonstrated altered timing in muscle activation along with more variable activation times. Furthermore, Lucas et al. (2010, 2004) demonstrated that treating the LTrP group with dry needling and passive muscle stretching normalised their muscle activation times. These normalised muscle activation times showed no significant difference when compared to the muscle activation times of the control group. Along with the normalisation of MAPs in those treated with dry needling and passive stretch there was also a reduction in the variability of muscle activation times (indicated by comparing the standard deviations of the onset of muscle activity times) within the treatment group, which again replicated the control group. The present study demonstrated greater variation in the muscle activation times across all muscles in all groups pre-interventions (ranging from 0.330 s to 0.973 s, in the middle deltoid and infraspinatus muscles respectively) in comparison to those reported by Lucas et al. (2010) (ranging from 0.132 s to 0.401 s, in the upper and lower fibres of the trapezius muscles respectively). The variation in the muscle activation times post-intervention remained high in the present study (ranging from 0.331 s, in the middle deltoid muscles, to 0.886 s, in the infraspinatus muscles), again this was in contrast to the Lucas et al. (2010) study which reported significant ($p < 0.05$) reductions in the standard deviation times post-intervention (unfortunately Lucas et al. (2010) only graphically illustrated times for post-intervention).

Pain is known to cause negative effects on motor control and performance (Sterling et al., 2001), and the findings by Lucas et al. (2010, 2004) demonstrated the possibility that LTrPs could cause altered MAPs before the presence of pain. They did this by demonstrating how, in the absence of pain, LTrPs caused alterations in MAPs and importantly that their removal restored what was considered “normal” MAPs. The present study utilised this finding to assess the effectiveness of dry needling as a treatment of LTrPs by assessing pre- and post-intervention changes in MAPs. Dry needling was compared to a treatment that has also been extensively researched as a treatment for ATrPs, manual release, and a placebo treatment, which acted as a control.

However, from the results generated by the present study the null hypothesis must be accepted, that the treatment of LTrPs with dry needling only and manual release only are not effective at altering MAPs in muscles of the shoulder complex.

There are important points to note when drawing direct comparisons with the Lucas et al. (2004) study. There was a slight difference in how the current study analysed the sEMG data obtained from participants. The current study utilised accelerometer data, built into the Trigno Wireless EMG sensors placed on the middle deltoid muscles, to determine movement of the upper limb whereas Lucas et al. (2004) utilised a custom built micro switch on the subjects thigh. It was chosen to do this as the accelerometer and sEMG data were synced to the same data acquisition software which could record data for the initiation of movement and the onset of muscle activity in tandem. The use of standardised equipment such as the Trigno Wireless EMG sensors allows for easy replication of the current study. The determination of the onset of muscle activity was also different, with the Lucas et al. (2004) study utilising 1 standard deviation and the present study using 2 standard deviations (Hodges and Bui, 1996). Following pilot data collection it was observed that using 1 standard deviation was not a viable option to determine the onset of muscle activity due to an inability to reliably identify the muscle activation for each repetition.

Due to the null hypothesis being accepted and the results of the current study contradicting those reported by Lucas et al. (2004) there is uncertainty as to whether LTrPs truly cause altered MAPs in key muscles of the shoulder complex. These results also show the uncertainty that surrounds both TrPs and their treatments, as well as the relevance of optimal MAPs. In relation to the shoulder complex, the results demonstrate that the now widely accepted connection between scapular dyskinesis and shoulder pathology should be questioned. These results demonstrated that there was considerable variability in the MAPs of healthy, pain free participants in all groups. It is still inconclusive whether altered MAPs are a cause or an effect of shoulder pathology and these results add weight to the possibility that a true optimal muscle activation strategy does not exist. Thus the use of clinical tests which observe scapular dyskinesis in screening or diagnostic assessments, as a potential connection with shoulder instability, may lead to unwarranted conclusions about pathomechanisms and the use

of irrelevant interventions that stifle clinical reasoning and decision-making (McQuade et al., 2016).

Similar to the uncertainty that surrounds TrPs, the lack of clear evidence on the relevance of scapular dyskinesis has led to alternative theories on the role of the scapula. It is possible that too much emphasis is being placed on the muscles that control and stabilise the scapula as a potential cause of shoulder pathology (McQuade et al., 2016). Hasan (2005) states that stability in the sense of quick resistance to perturbation often may not be necessary for successful control of forces, energy and movement, instead that movement variability creates resilience, which is more desirable than stability for the control of movement. The variability seen in the MAPs of participants in this study perhaps highlights this concept, that movement strategies are plastic and that individuals have the ability to alter and refine how they move based on the situation presented to them. This plasticity also means that when presented with identical situations, such as with the pre- and post-intervention task performed by participants in this study, there is uncertainty whether individuals will utilise the same control and stabilisation strategies or whether the learned experience causes them to adapt to the task. With this in mind McQuade et al. (2016) used the term robustness instead of stability when making reference to motor control, as it describes a systems tolerance for uncertainty, allowing for degrees of movement variability.

Movement variability is thought to be pervasive throughout the multiple levels of movement organisation and it occurs both within and between individuals (Newell and Corcos, 1993) and it is thought to exist because of the many complex systems and constraints that interact with each other in order to produce movement (James, 2004). Movement variability has been viewed as both detrimental and beneficial to skilled coordinated movement (James, 2004). The view that it is a benefit emerged from the study of the behaviour of chaotic nonlinear dynamic systems and applied to human movement (James, 2004). From this dynamic systems perspective, at least four benefits of variability have been suggested (James, 2004). Firstly, variability determines the stability of a movement pattern around an attractor. With this perspective, large amounts of variability are thought to suggest unstable movement patterns, while small amounts of variability indicate stable patterns. Currently researchers do not know what is considered a large amount of variability in the onset of muscle activation, thus, cannot

say with any certainty that certain MAPs are detrimental. Secondly, variability is thought to allow for flexibility within the neuromotor system to permit the learning of a new movement pattern through adjusting the appropriate parameters. Thirdly, variability is thought to allow for flexibility to select or change to new, previously learned movement patterns by rescaling parameters so that different movements can be assessed. Fourthly, variability is thought to provide random patterns that allow for constant sampling of different movement patterns. Suggestions two to four highlight the ability to learn new skills and how individuals can hone and enhance them over time as their expertise grows.

Variability has been observed and examined in a number of biological systems, such as heartbeat, respiration, menstrual cycle, sleep-wake cycle, and gait (James, 2004). In these systems the current prevailing viewpoint is that biological variability in the correct amount is essential for health, however, variation outside the normal limits, either too great or too small, may lead to a class of disease (James, 2004). Research in these areas led to the understanding of normal and altered variability, where previously the most optimal state was thought to be ordered and regular. In relation to MAPs researchers do not know whether more or less variability is advantageous or where the limits are, thus further research is needed.

4.7 Limitations

There are a number of limitations to the current study. Due to the use of standardised movements to examine altered MAPs, the ability to apply the findings to real world situations is hampered. Amasay & Karduna (2009) reported that scapular orientation recorded during constrained planar motion is different during functional tasks. They compared scapula orientations at the same plane and elevation angle achieved during both constrained and functional movements and reported angular differences ranging from 3.2° to 9.7° between movement conditions. Thus any association between shoulder pathology or dyskinesia and scapula orientations from studies using constrained movements may not directly translate to functional tasks.

This study only examined and treated muscles for which sEMG data was collected for LTrPs. As there was a limit to the number of muscles that could be examined by sEMG sensors, due to both sensor availability and the accessibility of muscles for sEMG analysis, it was decided to only assess and treat muscles which could be objectively examined. Theoretically, LTrPs in other muscles, such as the pectoralis minor, may have prevented any potential change in kinematics of the scapula and thus the MAPs of the examined muscles.

No other forms of assessment were used other than the SCT and sEMG data to determine the effectiveness of the interventions. Dry needling was ceased once no more LTRs could be elicited at each identified LTrP and manual release was ceased after two cycles of increased pressure. Future studies should reassess each LTrP following treatment to determine whether there has been an increase in their pressure pain thresholds and/or the elimination of a LTR.

During the process of collecting sEMG, pre- and post-intervention, it was necessary to remove the sEMG sensors to allow for unobstructed LTrP examination and to carry out the relevant interventions. While steps were taken to limit any variation in sensor placement this process of removal and reapplication may have added unintentional errors in the sEMG data collected.

Finally, results from the sEMG data should be interpreted with caution, as it only assessed changes within and between intervention groups at the onset of muscles activation. Therefore this data does not detail whether there was any changes in muscle

function during the rest of glenohumeral elevation. This is a limitation to the study as scapular dyskinesis and altered kinematics typically presents above 30° of elevation and during the lower stage of elevation. Thus the objective measure does not give a complete picture of potential changes in periscapular muscles.

4.8 Conclusion

Neither subjective observation of scapular motion, with the use of the SCT, nor the objective measurement of periscapular muscle's MAPs, with the use of sEMG analysis, identified that LTrP dry needling or manual release were effective at changing observable scapular motion or the onset of muscle activation. No significant differences or treatment effects were observed when comparing the treatment of LTrPs with dry needling, manual release or placebo dry needling. Therefore, the null hypothesis was accepted, that the treatment of LTrPs with dry needling, manual release, and placebo dry needling had no significant effect on scapular motion and the MAPs of key muscles of the shoulder complex.

4.9 Summary

Results from this study highlight the uncertainty that surrounds LTrP dry needling and manual release treatments as effective methods to alter MAPs of key periscapular muscles. In addition the role of altered MAPs as a clinical implication of LTrPs in key periscapular muscles is unclear. These results should be considered with caution as it is not known whether there was any change in the activity of muscles during the remainder of GH elevation, as only the onset of muscle activation was examined.

Chapter 5: Conclusion and Future Recommendations

5 Conclusion and Future Recommendations

5.1 Conclusion

Two clinical assessment tests for scapular dyskinesis, the SDT and the SCT, which are used to assess abnormal scapular movement, were found to demonstrate medium to high inter- and intra-tester reliability. However, no significant differences were found using sEMG analysis to compare MAPs between those determined to possess normal scapular motion versus those with scapular dyskinesis. Therefore, these tests are not validated to identify participants with altered MAPs. It is therefore unclear whether altered MAPs of periscapular muscles truly play a role in altered kinematics of the shoulder and whether scapular dyskinesis tests are of use when trying to assess for potential muscular abnormalities.

LTrPs have been proposed to possess a number of clinical implications, such as altered MAPs, which have also been related to shoulder pathologies. The use of dry needling and manual release in the treatment of participants with LTrPs present in key periscapular muscles did not result in any significant or clinically relevant changes in MAPs. These results indicate that TrP dry needling and manual release treatments were not effective at changing one of the proposed clinical implications of LTrPs.

5.2 Future Recommendations

Following the completion of this research a number of recommendations for future investigations are proposed. Research is needed to clarify whether LTrPs truly cause altered MAPs of periscapular muscles. At present Lucas et al. (2004) is the only study that has investigated the MAPs of participants with LTrPs versus a control group without any LTrPs. Since the results of this study did not demonstrate that treatment of LTrPs resulted in improved MAPs, in terms of reduced group standard deviations, further research is warranted. As research on TrPs continues, both in their active and their latent forms, researchers and clinicians should continue to critique the effectiveness of treatment interventions based on the current hypotheses. Randomised control trials of high quality should continue to assess the effectiveness of treatment interventions, examining the specific clinical implications that are proposed by the active and latent forms. The inconsistencies in results in the current literature do not definitively state whether the use of treatments for TrPs are effective in comparison to control or placebo.

Current rehabilitation programmes for patients with shoulder pathologies tend to focus on restoring what is considered normal scapular motion and often include specific exercises for particular muscles, based on the belief that muscular imbalance is the cause (Cools et al., 2007). However, the results from this study highlighted a high variability in the MAPs of participants during a standardised task. This high variability may be due to the fact that there is limited potential for precise voluntary control of the periscapular muscles during functional movements, due to their innervation. Taking this into consideration, along with the perspective that the CNS likely places the position and stability of the scapula as a low priority during functional movements, perhaps alternative avenues for the treatment of shoulder pathologies should be considered. Exercises that are intended to recruit and strengthen individual periscapular muscles may not be as effective as more general functional movements, such as pushing and pulling exercises, which do not specifically target the periscapular muscles. Thus, other forms of exercises which involve the upper extremity may produce better outcomes over exercises targeted at specific muscles and/or scapular stabilisation and should be further examined.

Finally, as the current study and the Lucas et al. (2004) study only investigated MAPs there is a lack of knowledge as to whether LTrPs cause any changes in muscle activation after the onset of activity. This is of particular interest as scapular dyskinesis is typically observed after initial movement, when the arm is being raised and/or lowered. Future research should investigate whether LTrPs cause altered EMG activity in periscapular muscles during movement and if so, whether treatment interventions for TrPs are effective at normalising them.

Chapter 6: Bibliography

6 Bibliography

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Chapter 7: Appendices

7 Appendices

Appendix A: Pilot Study Recruitment Email

Dear staff and students,

Would you like to have your shoulder function analysed?

I'm looking for male participants to take part in a short pilot study analysing shoulder function using video analysis and surface electromyography (sEMG) sensors.

This is an ideal opportunity for all athletes who use their shoulder to compete (Gaelic football, hurling, rugby, basketball, tennis, archery, etc.), gym goers who do weights, or if you're just interested to see if your shoulder is functioning as it should.

Candidates must be between the ages of 18-45, have a healthy shoulder at the time of testing, and be available for testing Wednesday 25/11/15 or Thursday 26/11/15.

Candidates will undergo:

A shoulder evaluation

Visual shoulder analysis

Surface electromyography (sEMG) analysis

After all tests are completed and analysed full feedback will be given to participants detailing their results, if requested. Advice on appropriate strategies to improve your shoulder function will then be provided.

If this is of interest to you please contact the research student by email or phone:

email: m.donohoe@research.ait.ie

phone: [087 654 8243](tel:0876548243)

Kind regards,

Michael Donohoe.

Postgraduate research student.

Appendix B: Plain Language Statement



Plain Language Statement

Supervisors: Dr. Niamh Ni Chéilleachair

Dr. Siobhán O'Connor

Dr. Giles Warrington

Prof. Neil Rowan

Investigator: Mr. Michael Donohoe

Purpose

The aim of this pilot study is to determine the reliability and validity of three scapular dyskinesis tests.

What is required of you?

As part of this study you will be required to attend one testing session. After the details of the study have been explained you will be required to sign an informed consent form. Prior to inclusion in this study you will be required to undergo screening by completing a pre-participation questionnaire and a pre-participation physical examination. These are in order to ensure you meet the requirements necessary for participation in the study.

Introduction

Once you have been given full details of the study you will be given the opportunity to ask any questions you may have. At this point you will be required to sign an informed consent form before the screening component of the session begins.

Pre-participation Screening

- You will be required to fill out a questionnaire related to the study.
- You will be required to go through a physical examination of the upper limb. This is to verify that you do not have any upper limb issues which may cause you to be excluded from the study.
- Your height and weight will be measured.

Scapular Dyskinesia Tests

Once you have completed the pre-participation screening you will progress onto the main purpose of the study. This will consist of three separate scapular dyskinesia tests. A scapular dyskinesia test is designed to evaluate the movement of your scapula (shoulder blade) as you move your arm through a full range of motion. Each of the tests will be recorded by a video camera for evaluation by an experienced practitioner. The video camera will only capture the back of your head, waist, and elbows.

- Male participants will be required to remove their shirts and female participants will be required to wear a halter top (provided if necessary).
- You will be required to perform a series of overhead movements guided by the instruction of the researchers.

Surface Electromyography (sEMG) Analysis

Following the scapular dyskinesia tests your shoulder muscles will be analysed using sEMG sensors. This will require a number of wireless sensors to be stuck to your skin with adhesive tape. Once applied the three scapular dyskinesia test will be repeated.

- You will be required to be fitted with wireless sEMG sensors on specific muscles involved in movement of your arms.
- You will not feel any sensation, other than the presence of the sensor on your skin, during the sEMG analysis.
- Following the application of the sEMG sensors you will be required to repeat the movements of the three scapular dyskinesia tests.

Potential Risks and Benefits

Risks

During both sessions males will be required to remove their t-shirts and females will be required to wear a halter top. You may experience some distress, discomfort or embarrassment as a result of this. To minimise this all procedures will take place behind a curtained off area.

Benefits

As part of the screening process you will go through a number of tests used to assess any abnormalities in the shoulder joint. The results of these test will be explained to you after your examination. Should you need any further assessment because of any issues which may have been discovered you will be given appropriate advice.

As part of the testing session your scapular control and the muscles involved will be assessed for any abnormalities. Upon completion of the testing the results of the testing will be explained to

you. Should you have any abnormalities these issues will be explained and you will be advised how to best correct them.

Confidentiality

The results and information received from this study are regarded as confidential and will be used by the investigating team only. It will be stored in a secure filing cabinet and password protected computer which will only be accessible to the investigating team. Your data will be kept anonymous through your participant ID code. Your data will be destroyed 5 years after publication of this study.

Freedom of Withdrawal

Participation in the study is voluntary. Therefore you are free to withdraw from the study at any time without prejudice or reason. If you have any queries prior to consenting participation or during the study please ask any of the investigating team.

You can contact the researcher at any time should you have any questions.

Contact information:

Michael Donohoe
Department of NHS
Athlone Institute of Technology
Athlone
m.donohoe@research.ait.ie
087 654 8243

Appendix C: Pilot Study Informed Consent Form



Informed Consent Form

A pilot study investigating the reliability and validity of three scapular dyskinesis tests.

- I have read and understand all the information in the **Volunteer Information Sheet**.
- I understand what the project is about and what the results will be used for.
- I am fully aware of all testing procedures and they have been verbally explained to me in detail.
- I am aware of the potential **risks and benefits** associated with this study.
- I understand that any information about me will be kept confidential and my information will be coded with a subject ID.
- I understand that the results of the research study may be published but that my identity will not be revealed.
- I know that my participation in this study is voluntary and that I can withdraw from the study at any time without giving a reason.
- I understand that if I have any questions regarding any aspect of this research study I can contact any of the investigators involved with this study.

Volunteer's name: _____

Volunteer's signature: _____

Date: _____

Witness' Name: _____

Witness' Signature: _____

Date: _____

Investigator's signature: _____

Date: _____

Appendix D: Pilot Study Pre-participation Questionnaire



Pre-participation Questionnaire

Participant ID: _____

1 What is your gender?

- Male
Female

2 What is your date of birth?

DOB **DD/MM/YYYY**
____/____/____

3 What is your ethnicity?

- White/Caucasian
Black/African American
Asian
Other (Please specify) _____

4 Have you had any pain in the back, neck, or upper limb in the past 7 days?

- Yes
No

5 i) Have you a history of any injuries to your neck or upper limb?

- Yes
No

ii) if yes, tick where relevant.

- | | | | | | |
|-------------------|--------------------------|---------------------------------|--------------------------|----------------------|--------------------------|
| Rotator cuff tear | <input type="checkbox"/> | Labral tear | <input type="checkbox"/> | Shoulder dislocation | <input type="checkbox"/> |
| Fractured scapula | <input type="checkbox"/> | Fractured humerus | <input type="checkbox"/> | Fractured clavicle | <input type="checkbox"/> |
| Other | <input type="checkbox"/> | If other, please specify: _____ | | | |

6 i) Have you a history of any nerve injuries in the cervical or shoulder area?

- Yes
No

ii) If yes, tick where relevant.

- | | | | | | |
|---------------|--------------------------|------------------|--------------------------|---------------------|--------------------------|
| Long thoracic | <input type="checkbox"/> | Spinal accessory | <input type="checkbox"/> | Cervical nerve root | <input type="checkbox"/> |
|---------------|--------------------------|------------------|--------------------------|---------------------|--------------------------|

7 Have you any allergies to adhesives?

- Yes
No

Appendix E: Shoulder Assessment Form



Shoulder Assessment Form American Shoulder and Elbow Surgeons

Participant ID:		Date:	
Age:	Hand Dominance: R L Ambi	Sex: M F	

RANGE OF MOTION Total shoulder motion goniometer preferred				
	Right		Left	
	Active	Passive	Active	Passive
Forward elevation (maximum arm-trunk angle)				
External rotation (arm comfortably at side)				
External rotation (arm at 90 degree abduction)				
Internal rotation (highest posterior anatomy reached with thumb)				
Cross-body adduction (antecubital fossa to opposite acromion)				
Abduction				

SIGNS 0 = none; 1 = mild; 2 = moderate; 3 = severe								
	Right				Left			
	0	1	2	3	0	1	2	3
Supraspinatus/greater tuberosity tenderness								
AC joint tenderness								
Biceps tendon tenderness (or rupture)								
Other tenderness – list:								
Impingement I (passive forward elevation in slight internal rotation)								
Impingement II (passive internal rotation with 90 degree flexion)								

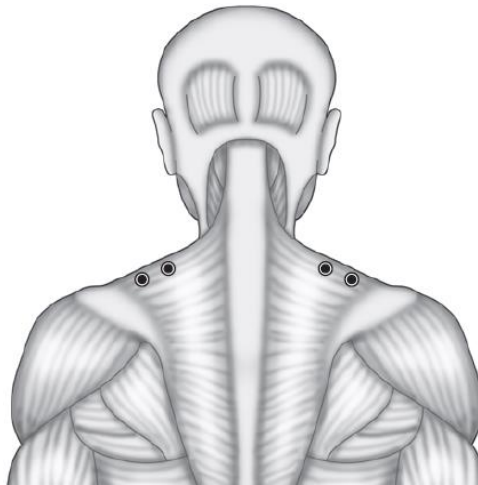
STRENGTH (record MRC grade)		
0 = no contraction; 1 = flicker; 2 = movement with gravity eliminated 3 = movement against gravity; 4 = movement against some resistance; 5 = normal power		
	Right	Left
Testing affected by pain?	Y N	Y N
Forward elevation	0 1 2 3 4 5	0 1 2 3 4 5
Abduction	0 1 2 3 4 5	0 1 2 3 4 5
External rotation (arm comfortably at side)	0 1 2 3 4 5	0 1 2 3 4 5
Internal rotation (arm comfortably at side)	0 1 2 3 4 5	0 1 2 3 4 5
Shoulder elevation (shoulder shrug)	0 1 2 3 4 5	0 1 2 3 4 5
Scapular Adduction	0 1 2 3 4 5	0 1 2 3 4 5
Scapular Protraction	0 1 2 3 4 5	0 1 2 3 4 5
Scaption (prone shoulder flexion in scapular plane)	0 1 2 3 4 5	0 1 2 3 4 5
Impingement III (90 degree active abduction – classic painful arc)	Y N	Y N
Subacromial crepitus	Y N	Y N
Scars – location:	Y N	Y N
Atrophy – location:	Y N	Y N
Deformity – describe:	Y N	Y N

INSTABILITY		
0 = none; 1 = mild (0 – 1 cm translation) 2 = moderate (1 – 2 cm translation or translates to glenoid rim) 3 = severe (>2 cm translation or over rim of glenoid)		
	Right	Left
Anterior translation	0 1 2 3	0 1 2 3
Posterior translation	0 1 2 3	0 1 2 3
Inferior translation (sulcus sign)	0 1 2 3	0 1 2 3
Anterior apprehension	0 1 2 3	0 1 2 3
Reproduces symptoms?	Y N	Y N
Voluntary instability?	Y N	Y N
Relocation test positive?	Y N	Y N
Generalized ligamentous laxity?	Y N	
Other physical findings:		

Anthropometric Data	
Height	cm
Weight	kg

Appendix F: Sensor Placement

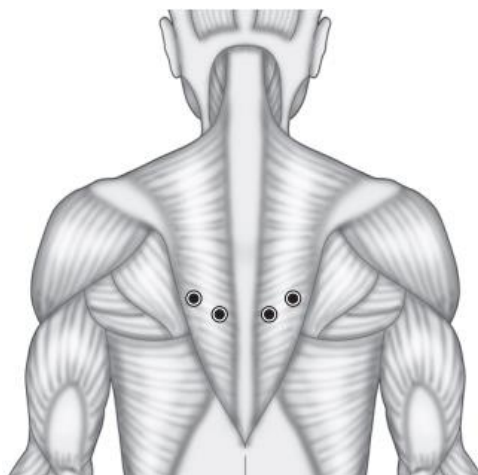
Upper Trapezius



Location: Place the sensor so they run parallel to the muscle fibres of the upper trapezius, along the ridge of the shoulder, slightly lateral to and one-half the distance between the cervical spine at C-7 and the acromion.

Muscle Test: Shoulder elevation/shrug, lateral bending of the head.

Lower Trapezius



Location: Palpate the interscapular region. Have the participant retract and depress the scapula and then flex the arm to at least 90 degrees. Palpate the inferior medial border of the scapula for the muscle mass that emerges. Place the sensor on the oblique angle, approximately 5 cm down from the spine of the scapula. The sensor is placed next to the medial edge of the scapular at a 55 degree oblique angle.

Muscle Test: Abduction of arms; retraction of the shoulder back and down at a 45 degree angle.

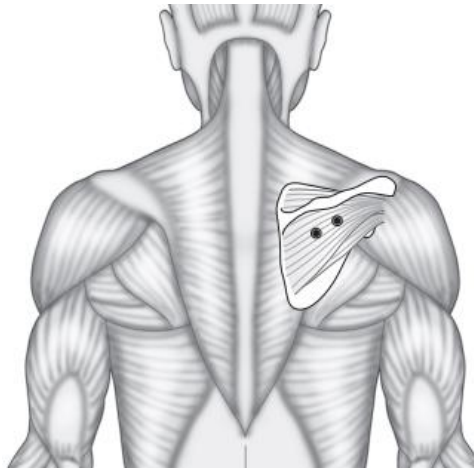
Serratus Anterior



Location: Have the participant flex the arm against resistance. Palpate this contraction in an area just anterior to the border of the latissimus dorsi muscle at the level of the inferior angle of the scapula. Place the sensor horizontally just below the axillary area, at the level of the inferior angle of the scapula, and just medial of the latissimus dorsi. It is important that the electrodes are anterior to the latissimus dorsi muscle.

Muscle Test: Forward flexion of the arms, protraction of the shoulder, push-ups.

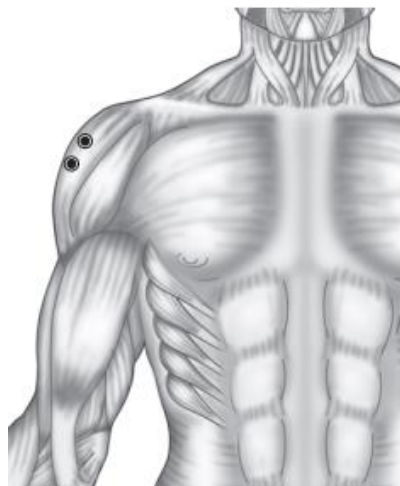
Infraspinatus



Location: Palpate the spine of the scapula. The sensor is placed parallel to and approximately 4 cm below the spine of the scapula, on the lateral aspect, over the infrascapular fossa of the scapula. Avoid placement over the posterior deltoid.

Muscle Test: Elbow bent to 90 degrees with lateral (external) rotation of the bend arm out to the side; abduction of the arm.

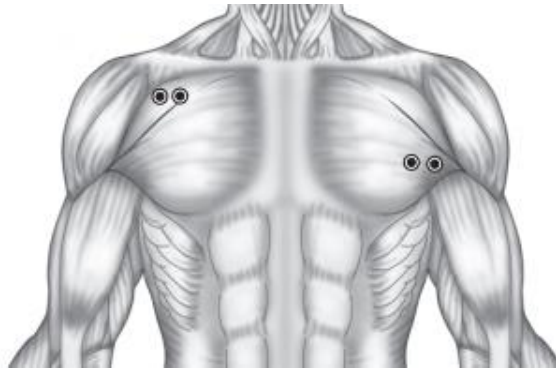
Middle Deltoid



Location: The sensor is placed on the lateral aspect of the upper arm and approximately 3 cm below the acromion, over the muscle mass so that the sensor runs parallel to the muscle fibres.

Muscle Test: Abduction of the arm.

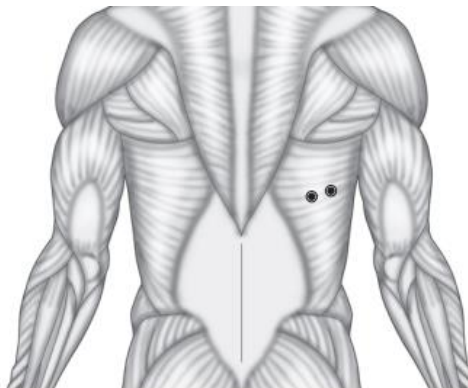
Pectoralis Major (Clavicular Head)



Location: Palpate the clavicle. Place the sensor on the chest wall at an oblique angle towards the clavicle, approximately 2 cm below the clavicle, just medial to the axillary fold

Muscle Test: Flexion of the arm, abduction of the arm above 90 degrees, medial rotation, horizontal adduction of the arm.

Latissimus Dorsi



Location: Palpate the scapula. The sensor is placed approximately 4 cm below the inferior tip of the scapula, half the distance between the spine of the lateral edge of the torso. It is orientated in a slightly oblique angle of approximately 25 degrees.

Muscle Test: Extend, adduct, or medially rotate the arm.

Appendix G: Main Study Recruitment Email

Dear all,

Would you like to have your shoulder function analysed and optimised?

I'm a postgraduate research student and I'm recruiting male participants to take part in a short study which will analyse shoulder function and assess the effectiveness of treatments strategies to optimise it.

This is an ideal opportunity for all athletes who use their shoulders to compete (Gaelic football, hurling, rugby, basketball, tennis, athletics field events, archery, etc.), gym goers who do weights, or if you're just interested to see if your shoulder is functioning optimally.

Candidates will be required to attend two sessions on separate days: an introductory and screening session that will last approx. 30 minutes, and the main testing session lasting approx. 90 minutes.

During the study candidates will undergo:

A shoulder evaluation to assess any underlying injuries, shoulder range of motion and strength

Visual shoulder analysis by two qualified practitioners

Surface electromyography (sEMG) shoulder analysis using wireless sEMG sensors

Treatment to optimise shoulder function

Note: Candidates must be males aged between 18-45 years and have a healthy shoulder at the time of testing.

After all tests are completed and analysed full feedback will be given to participants detailing their specific results. Advice on appropriate strategies to improve your shoulder function will then be provided.

If this is of interest to you please contact me by email or phone:

email: m.donohoe@research.ait.ie

phone: [087 654 8243](tel:0876548243)

Kind regards,

Michael Donohoe BSc ARTC

Postgraduate Research,
Bioscience Research Institute,
Athlone Institute of Technology,
Dublin Road,
Athlone,
Co. Westmeath,
Ireland.



HAVE YOUR SHOULDERS ANALYSED & OPTIMISED

A Postgraduate Research Student is looking for male participants to take part in a short study analysing shoulder function & appropriate treatments to optimise it.

If this is of interest to you please contact the research student:

Michael: m.donohoe@research.ait.ie



**ATTENTION
ALL**

Gaelic Footballers

Hurlers

Rugby Players

Basketballers

Field Athletes

Tennis Players

STUDY INVOLVES

Shoulder Evaluation

Visual Shoulder Analysis

Surface Electromyography (sEMG) Analysis

Treatment

Appendix I: Main Study Plain Language Statement



Volunteer Information Sheet

Supervisors: Dr. Niamh Ní Chéilleachair

Dr. Siobhán O'Connor

Dr. Giles Warrington

Prof. Neil Rowan

Investigator: Mr. Michael Donohoe

Purpose

The aims of this study is to investigate the effects of latent myofascial trigger points on muscle activation patterns of the scapula (shoulder blade) rotators and glenohumeral (shoulder joint) stabilisers, and to determine the most effective treatment for the restoration of “normal” muscle function.

What is required of you?

As part of this study you will be required to attend two separate sessions, first an introductory/screening session, followed by a testing session. After the details of the study have be explained you will be required to sign an informed consent form. Prior to inclusion in this study you will be required to undergo screening by completing a pre-participation questionnaire and a pre-participation physical examination. These are in order to ensure you meet the requirements necessary for participation in the study.

Introductory/Screening session

Once you have been given full details of the study you will be given the opportunity to ask any questions you may have. At this point you will be required to sign an informed consent form before the screening component of the session begins.

Pre-participation Screening

- You will be required to fill out a questionnaire related to the study.

- You will be required to go through a physical examination of the upper limb. This is to verify that you do not have any upper limb issues which may cause you to be excluded from the study.
- Your height and weight will be measured.
- You will be required to return on an agreed date for the testing session.

Testing session

The testing session is made up of four separate components: scapular dyskinesis screening, surface electromyography (sEMG) analysis, myofascial trigger point (TrP) examination, and treatment intervention. The scapular dyskinesis screening and sEMG analysis will be repeated after your specific treatment intervention.

- You will be required to wear appropriate clothes for the testing session; shorts and a t-shirt.

Scapular Dyskinesis Screening

This screening test will be used to assess the movement and control of your scapula (shoulder blade) during overhead movements. Your back will be recorded with a video camera as you perform an overhead movement in time to a metronome. The video camera will be adjusted to capture the back of your head, waist, and elbows only.

- You will be required to perform an overhead movement at a slow and controlled tempo in time to a metronome.

Surface Electromyography (sEMG) Analysis

Following the scapular dyskinesis screening your shoulder muscles will be analysed using sEMG sensors. This will require a number of wireless sensors to be stuck onto your skin with adhesive tape.

- You will be required to be fitted with wireless sEMG sensors on specific muscles involved in movement of your arms.
- You will not feel any sensation, other than the presence of the sensor on your skin, during the sEMG analysis.
- Following the application of the sEMG sensors you will be required to repeat overhead movements, similar to those performed during the scapular dyskinesis test.

Trigger Point Examination

Following the two pre-intervention tests you will then be examined for the presence of latent trigger points (LTrPs) within the shoulder muscles. LTrPs are small contractures within muscles. Upon identification of an LTrP a pain pressure reading will be taken of the suspected trigger point. This involves using a device that measures pressure. This will assess the sensitivity of the LTrP.

- You will be required to lie on an examination table where you will be examined for myofascial trigger points.
- During the examination you will be required to communicate with the examiner to identify areas of increased tenderness within your shoulder muscles.
- When recording the sensitivity of the LTrPs you will be required to identify the point at which the sensation you feel changes from a one of pressure to one of slight discomfort.

Intervention

Next you will be randomly assigned to one of three treatment groups, manual release, dry needling group or a control group.

- You will be required to lie on the examination table where you will be treated.
- The researcher will explain the treatment procedure to you before it begins. You will have another opportunity to ask the researcher any questions you may have at this point.
- During the treatment you will be required to maintain verbal communication with the researcher as they treat any LTrPs that were found during the examination process.

Post-intervention testing

After you receive your treatment you will repeat the scapular dyskinesis testing and the sEMG analysis.

Potential Risks and Benefits

Risks

During both sessions males will be required to remove their t-shirts. You may experience some distress, discomfort or embarrassment as a result of this. To minimise this all procedures will take place behind a curtained off area.

During the testing session you may feel discomfort during the trigger point examination and the treatment intervention. This will be minimised with your communication with the researcher. They will alter the treatment to your tolerances.

Should you receive the dry needling intervention there a chance of minor bleeding and/or bruising due to lesion of small vessels (capillaries). As it is only capillaries that are affected by this the quantity of blood is only a teardrop. This will be minimised by compression of the treated area.

Benefits

As part of the screening process you will have to go through a number of tests used to assess any abnormalities in the shoulder joint. The results of these test will be explained to you after your examination. Should you need any further assessment because of any issues which may have been discovered you will be given appropriate advice.

As part of the testing session your scapular control and the muscles involved will be assessed for any abnormalities. Upon completion of the testing the results of the testing will be explained to you. Should you have any abnormalities these issues will be explained and you will be advised how to best correct them.

Any trigger points found during the examination process will be treated using an appropriate intervention. After testing is complete you will be given advice on how to best reduce the occurrence of these trigger points.

Confidentiality

The results and information received from this study are regarded as confidential and will be used by the investigating team only. It will be stored in a secure filing cabinet and password protected computer which will only be accessible to the investigating team. Your data will be kept anonymous through your personal ID code. Your data will be destroyed 5 years after publication of this study.

Freedom of Withdrawal

Participation in the study is voluntary. Therefore you are free to withdraw from the study at any time without prejudice or reason. If you have any queries prior to consenting participation or during the study please ask any of the investigating team.

You can contact the researcher at any time should you have any questions.

Contact information:

Michael Donohoe
Department of NHS
Athlone Institute of Technology
Athlone

m.donohoe@research.ait.ie

087 654 8243

Appendix J: Main Study Informed Consent Form



Informed Consent Form

An investigation into the effectiveness of latent myofascial trigger point dry needling on muscle function and performance.

- I have read and understand all the information in the **Volunteer Information Sheet**.
- I understand what the project is about and what the results will be used for.
- I am fully aware of all testing procedures and they have been verbally explained to me in detail.
- I am aware of the potential **risks and benefits** associated with this study.
- I understand that any information about me will be kept confidential and my information will be coded with a subject ID.
- I understand that the results of the research study may be published but that my identity will not be revealed.
- I know that my participation in this study is voluntary and that I can withdraw from the study at any time without giving a reason.
- I understand that if I have any questions regarding any aspect of this research study I can contact any of the investigators involved with this study.

Volunteer's name: _____

Volunteer's signature: _____

Date: _____

Witness' Name: _____

Witness' Signature: _____

Date: _____

Investigator's signature: _____

Date: _____

Appendix K: Main Study Pre-participation Questionnaire



Pre-participation Questionnaire

Participant ID: _____

1 What is your gender?

- Male
Female

2 What is your date of birth?

DOB
DD/MM/YYYY

3 What is your ethnicity?

- White/Caucasian
Black/African American
Asian
Other (Please specify) _____

4 Have you had any pain in the back, neck, or upper limb in the past 7 days?

- Yes
No

5 i) Have you a history of any injuries to your neck or upper limb?

- Yes
No

ii) if yes, tick where relevant.

- Rotator cuff tear Labral tear Shoulder dislocation
Fractured scapula Fractured humerus Fractured clavicle
Other If other, please specify: _____

6 i) Have you a history of any nerve injuries in the cervical or shoulder area?

- Yes
No

ii) If yes, tick where relevant.

- Long thoracic Spinal accessory Cervical nerve root

7 Have you any allergies to adhesives, metals and/or a phobia of needles?

- Yes
No

Appendix L: Location of Latent Trigger Points



Participant ID: _____



Muscle	Side	LTrP No.	Pain Pressure Threshold			Referral Sensation	LTR
Middle Deltoid							
Upper Trapezius							
Lower Trapezius							
Infraspinatus							
Serratus Anterior							

Appendix M: Visual Analogue Scale (VAS)

