The immediate effects of thoracic spine grade III mobilisation on the muscle activity of the middle and lower trapezius muscle

By

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Dissertation submitted in partial compliance with the requirements for the Master’s Degree in Technology: Chiropractic Durban University of Technology

I, Shinay Elizabeth Smit, do declare that this dissertation is representative of my own work in both conception and execution (except where acknowledgements indicate to the contrary)

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M. Tech: Chiropractic
"Families are the compass that guides us. They are the inspiration to reach great heights, and our comfort when we occasionally falter."

To my parents, Van De Sandt De Villiers and Sanette Smit, and my sister, Amy Smit, I dedicate this dissertation to you.
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Father God, for giving me the passion and strength to follow my dreams – “Commit to the Lord whatever you do, and He will establish your plans.” Proverbs 16:3

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ABSTRACT

Background: Thoracic spine dysfunction often presents with regional hypomobility which is often caused by abnormal muscle activity in the overlying area. Such as the trapezius muscle. Joint dysfunction is often treated by manual therapies such as joint mobilisation. Previous studies have established that joint mobilisation improves joint mobility, achieves hypoalgesia, improves stability, range of motion and proprioception. Although documented in the literature, the direct benefits of joint mobilisation on muscle activity are not well understood. Therefore, an investigation into the effects of mobilisation on muscle activity was explored.

Aim: This study aim was to determine the immediate effects of thoracic spine grade III mobilisation on the muscle activity of the middle and lower trapezius muscles.

Methods: This was a quantitative, experimental, study with a pre-test post-test design. Surface electromyography was used to measure the muscle activity of the middle and lower trapezius muscle. A sample size of 48 asymptomatic participants were recruited and randomly divided into the intervention or control group. The intervention group received thoracic grade III mobilisation and the control group remained prone between the pre-test and post-test readings. Within group comparisons was achieved using paired T-tests. Within group and between group comparisons of the change between pre and post intervention was achieved using repeated ANOVA testing. A p-value below 0.05 was considered significant.

Results: Spinal mobilisation had no effect on muscle activity. Despite the lack of statistical evidence, there was a positive trend in the effects of thoracic spine mobilisation with a borderline treatment effect in the left middle trapezius muscle ($p = 0.063$). There was an overall decrease in muscle activity in the intervention group.

Conclusion: The results showed that mobilisation did not produce a noteworthy change in muscle activity of middle and lower trapezius muscles between the intervention and control groups and the null hypothesis was not rejected.

Key Indexing Terms: Muscle activity, neurophysiology, spinal mobilisation, trapezius muscle, thoracic spine.
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<tr>
<td>Ach</td>
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<td>adenosine triphosphate</td>
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<td>CDC</td>
<td>Chiropractic Day Clinic</td>
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<td>cm</td>
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<td>CN</td>
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<td>CNS</td>
<td>central nervous system</td>
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<td>DUT</td>
<td>Durban University of Technology</td>
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<td>electromyography</td>
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<td>GTO</td>
<td>golgi tendon organ</td>
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<td>IREC</td>
<td>Institutional Research Ethics Committee</td>
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<td>intervertebral disc</td>
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<td>LLT</td>
<td>left lower trapezius</td>
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<td>LMT</td>
<td>left middle trapezius</td>
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<td>mm</td>
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<td>MSK</td>
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<td>MVIC</td>
<td>maximum voluntary isometric contraction</td>
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<td>n</td>
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<td>PNS</td>
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<td>RLT</td>
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<td>spinal manipulation therapy</td>
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DEFINITIONS

Asymptomatic:
A patient without symptoms (Venes 2017).

Cavitation:
The formation of vapour and gas bubbles within joint fluid, through the local reduction of pressure typically as a result of joint mobilisation and manipulation (Bergmann and Peterson 2011).

Joint Dysfunction / Fixation:
These terms are used interchangeably throughout this study. It refers to the state in which a joint has become temporarily immobilised in a position, which it may normally inhabit during any phase of physiologic movement. It is the restriction of an articulation in a position of movement when the joint is at rest or in a position of rest when the joint is in movement; irrespective of the presence of pain (Bergmann and Peterson 2011).

Joint Facilitation:
Reduction in the stimulus threshold in a particular spinal cord segment and its respective joint. When becoming highly excitable, a smaller stimulus will trigger excessive reflex-based firing within the segment leading to joint dysfunction and muscle hypertonicity (Korr 1975).

Motion Palpation:
This is a palpatory diagnosis of passive and active segmental joint range of motion (Bergmann and Peterson 2011).
Muscle Activity:

The random, staccotic firing of action potentials of groups of muscle fibres (Crisswell 2010).

Spinal Mobilisation:

A form of, non-thrust, manual therapy that entails rhythmic, gentle and passive movements of controlled force and amplitude on joints against a restrictive elastic barrier within the patient’s physiological range of motion (Bergmann and Peterson 2011).

Grade III Maitland Mobilisation:

A grade III Maitland mobilisation technique is used in this study – it is a mobilisation of high amplitude and low velocity at the end of the patient’s physiological range of motion and minimally engages the elastic barrier (Ward, Hebron and Petty 2017).

Surface Electromyography:

Surface electromyography refers to the use of surface electrodes for recording electrical action potentials from the underlying musculature. It is used in the analysis of movement, posture and emotional expression; inspecting for any evidence of antalgic postures, antalgic postures, and abnormal muscle recruitment patterns (Crisswell 2010).
CHAPTER 1: INTRODUCTION

This chapter provides a background to the study by introducing the research problem and a study rationale in order to emphasise how this study will contribute to the body of knowledge. The aims and objectives are illustrated as well as the flow of the dissertation.

1.1 Introduction

The thoracic region refers to a region of the spine that extends from the vertebral levels T1 – T12. This spinal segment is responsible for protecting organs within the thorax, allowing for movements of the back as well as movements of the scapulae. This spinal region is a crucial aspect of the body’s kinematic chain due to the large number of muscles that attach in the thoracic spine. One such muscle includes the trapezius muscle, which is the broadest, most superficial, and triangular muscle of the upper back. It spans from the most superior aspect of the cervical spine to the shoulder joint complex as well as the most inferior aspect of the thoracic spine (Cramer and Darby 2014). Hence, this muscle is a fundamental muscle that acts as a biomechanical link between the different kinematic chains of the human body (Ignasiak, Ferguson and Arjmand 2016).

The thoracic spine is the middle segment of the spinal column and connects the cervical- and lumbar regions to one another. Relative to the other spinal regions, which rely on adjacent musculature for stability, the thoracic spine is more rigid due to the additional bony and ligamentous structures of the rib cage (Cramer and Darby 2014). Biomechanically, the thoracic spine is responsible for providing stability in the sagittal plane of the spine, optimising force transmission from the upper body to the lumbar spine, supporting the trunk muscles by providing a strong framework for attachment as well as providing adequate flexibility for three-dimensional movements (Ignasiak, Dendorfer and Ferguson 2015).
Thoracic spine dysfunction occurs when there are deviations from normal alignment in the thoracic spine resulting in reduced motion and abnormal muscle recruitment in this region (Joshi, Balthillaya and Neelapala 2019). Thoracic spine dysfunction, consequentially, alters the normal length-tension relationships of the postural muscles that attach in this region, leading to dysfunction of these muscles. Biomechanically, the key postural muscles of the thoracic spine have attachments spanning across the other spinal regions. Hence, thoracic spine dysfunction will alter mechanical loading in other spinal regions and vice versa (Lee and Lee 2017). Ultimately, if there is a change in any segmental chain, whether in the cervical spine or lumbar spine, it creates hypomobility in the thoracic spine and, therefore, alters the overall biomechanics (Deepa, Dabholkar and Yardi 2014).

Although joint dysfunction, otherwise known as a fixation, is not defined by the presence of pain, dysfunction of the musculoskeletal (MSK) components will eventually lead to thoracic spine pain (Haavik and Murphy 2012). Thoracic spine pain refers to any pain that occurs between the cervicothoracic junction (C7/T1) and the thoracolumbar junction (T12/L1) (Heneghan and Rushton 2015). It occurs globally, in isolation, in 34.7% of cases in comparison to being accompanied by pain in other spinal regions which occur in 76.7% of cases (Roquelaure et al. 2014). This pain is a nonspecific pain which is why the thoracic spine is often disregarded as a causative factor of a patient’s chief complaint (Pecos-Martín et al. 2015). A study by Reed and Pickar (2015) demonstrated that intervertebral fixations resulted in a decrease in muscle spindle response of the paraspinal muscles resulting in abnormal muscle activity.

The most common pain associated with this region is MSK pain (Pan et al. 2018). For this reason, manual therapy is a very effective, non-invasive, approach to treating thoracic spine dysfunction and associated pain. One such manual therapy includes spinal mobilisation due to its beneficial clinical effects. A study by Yang et al. (2015) elicited that thoracic spine mobilisation improved thoracic spinal mobility which in turn
assisted in lumbar stability. More so, a clinical trial by Cho, Lee and Lee (2017) further proved that spinal mobilisation improves joint proprioception and joint alignment.

Many studies have evaluated the effects of spinal manipulation therapy (SMT) on muscle activity and have been proven that SMT is a successful manual therapy tool, but there is a paucity in literature that evaluates the immediate effects of spinal mobilisation, specifically, on muscle activity relative to other manual therapies. A study conducted by Lee and Lee (2017) demonstrated that spinal mobilisation decreased the muscle tone of the trapezius muscles through analysing range of motion (ROM), posture and pain threshold but calls for further investigations specifically on the effects on neuromuscular activity. Understanding the immediate neurophysiological effects of spinal mobilisation on the muscle activity of muscles in the thoracic spine will contribute to the paradigm shift in how therapists consider the technique and application of spinal mobilisation; this manual therapy tool will be used in cases where SMT is contraindicated and still achieve similar treatment outcomes. The benefits of mobilisation on muscle activity will be better understood and treatment outcomes will be more beneficial for practitioners as well.

1.2 Research Problem

There are numerous studies proving the clinical benefits of SMT which include decreased muscle activity, improved joint mobility, proprioception decreased pain, but limited studies are available on the clinical benefits when joint mobilisation is used as a treatment modality (Pecos-Martín et al. 2015). Current literature focuses on the effects of spinal mobilisation on ROM, pain threshold and joint alignment but there is a paucity in literature specifically focusing on the effects of spinal mobilisation on muscle activity.

More so, there are limited studies that have focused on manual therapy treatment protocols of the thoracic region because this region is often overlooked. Most often, patients experience neck pain or lower back pain as their primary complaint and these areas are focused on when the source of the dysfunction, in fact, originates in the
thoracic spine (Heneghan and Rushton 2015). Of the current studies that focus on the thoracic spine, they are primarily targeted at thoracic SMT and its effects on muscle activity (Masaracchio et al. 2019).

Furthermore, the thoracic region is an area with widespread muscular attachments. The trapezius muscle is one of the biggest and most superficial muscles of the thoracic spine – spanning from the top of the cervical spine to both shoulders as well as the bottom of the thoracic spine (Moore, Dalley and Agur 2014). Therefore, evaluating the effects of thoracic spine mobilisation on the muscle activity of the trapezius will target both the neurophysiological and biomechanical aspects of thoracic spine dysfunction (Mannen et al. 2015). Mobilisation is a modality that is frequently used due to its major clinical benefits thereby warranting a substantial need to establish the direct effects of this modality on the neuromuscular activity of muscles in the thoracic spine (Salom-Moreno et al. 2014).

1.3 Study Rationale

Thoracic spine dysfunction and pain is often caused by skeletal muscles (Ortega-Santiago et al. 2019). This is attributed to the fact that injury to the thoracic spine alters the normal muscle recruitment patterns; they become hyperactive (Korr 1975; Abboud, Nougarou and Descarreaux 2016). The musculature bulk associated with thoracic spine dysfunction are the trapezius, levator scapulae, rhomboids and erector spinae. Over activity of these muscles cause hypomobility in the thoracic spine and the cervical and lumbar spine become hypermobile as a compensatory mechanism (Muscolino 2017). The abnormal biomechanics of the entire spine consequently induces pain and muscles fatigue sooner – making them more susceptible to injury (Abboud, Nougarou and Descarreaux 2016).

The thoracic spine is often overlooked during spinal assessments because pathologies related to the thoracic spine are, most often, non-specific and thought to be of another origin (Muscolino 2017; Pan et al. 2018). In 40.7% of spinal assessments, thoracic spine dysfunction co-exists with clinical complaints in the
cervical and lumbar regions and these patients experience no pain in the thoracic spine, hence, the area becomes neglected (Heneghan et al. 2015). A systematic review by Pan et al. (2018) emphasised that it is essential to address the abnormal biomechanics of an asymptomatic, dysfunctional, thoracic spine when treating a patient’s primary complaint. Pecos-Martín et al. (2015) further emphasised that conducting a clinical trial on asymptomatic individuals will further assist in achieving objective results as the avoidance behaviour associated with pain will be eliminated.

Joint mobilisation is an effective form of manual therapy for individuals that are symptomatic and asymptomatic for pain; mobilisation improves joint mobility and achieves hypoalgesia (Mehyar et al. 2017). It further provokes improved stability, postural sway, ROM and joint proprioception (Weerasekara et al. 2017). However, there are limited studies that investigate the actual neurophysiological effects of spinal mobilisations.

A systematic review by Hegedus et al. (2011) focused on the immediate neurophysiological effects of a single session of spinal mobilisation and it revealed that mobilisation elicited: increased pain pressure threshold and changes in skin conductance, skin temperature and neuro-tensioning. These effects were local and remote to the site of the mobilisation; implying that spinal mobilisation does in fact have a clinical and neurophysiological effect, but its effects, solely, on muscle activity remain unclear. Studies by Pecos-Martín et al. (2017) revealed that the neurophysiological effects of SMT have been investigated but SMT is often contraindicated and further establishing the neurophysiological effects of spinal mobilisation will allow this manual therapy to serve as an effective alternative treatment approach in such cases.

Understanding the immediate effects of spinal mobilisation on the muscle activity of muscles in the thoracic spine will contribute to the evolving body of knowledge. Although SMT is widely investigated, it is beneficial to understand the effects of spinal mobilisation in cases where SMT is contraindicated. Treatment protocols for manual
therapists would be more valuable as compensatory mechanisms and pain in the cervical and lumbar segments would be resolved more efficiently by normalising the biomechanics in the thoracic spine.

1.4 Aims and Objectives

1.4.1 Aim

The aim of this study was to determine the immediate effects of thoracic spine grade III mobilisation on the muscle activity of the middle and lower trapezius muscles.

1.4.2 Objectives

Objective One:
To determine the immediate effects of thoracic spine grade III mobilisation on the muscle activity of the middle and lower trapezius muscles.

Objective Two:
To determine the immediate effects of a control procedure on the muscle activity of the middle and lower trapezius muscles.

Objective Three:
To compare and correlate the electromyographic data between the intervention and control groups.

1.5 Hypotheses

1.5.1 Null Hypothesis

There will be no difference in muscle activity between the intervention and control groups in terms of the surface electromyographic readings pre and post intervention.

1.5.2 Alternate Hypothesis

There will be a statistically significant differences between the intervention and control groups in terms of the surface electromyographic readings pre and post intervention.
1.6 Flow of Dissertation

This chapter has introduced the research problem and its setting as well as outlining the aims, objectives, and hypotheses.

Chapter two entails the literature review which highlights the literature enclosing the research problem in terms of the neurophysiology related to spinal mobilisations and its clinical benefits. In addition to this, there is an overview of thoracic spine dysfunction and its consequent abnormal biomechanics – specifically relating to the trapezius muscles.

Chapter three outlines and explains the methodology of the study, measurement tools and interventions that were used to achieve the aims and objectives of this study.

Chapter four entails the results recorded in this study.

Chapter five includes analysis and discussion of the results in relation to the current literature in context of the research problem.

Chapter six is the final chapter of this study which is comprised of the conclusion as well as limitations and recommendations for further research.
CHAPTER 2: LITERATURE REVIEW

2.1 Introduction

This chapter describes the anatomy of the thoracic spine and its relevant articulations and an outline of the physiology of both the nervous system and skeletal muscle as this is a study investigating the neurophysiological effects of joint mobilisation on muscle activity. Hence, the importance of establishing a thorough understanding of the relevant anatomy and systems. In addition to this, the association between joint dysfunction and muscle activity will be discussed in order to relate to the theories around the clinical effects of grade III spinal joint mobilisation. A review of literature concerning the research question shall also be discussed.

All relevant literature was accessed online using appropriate key indexing terms: spinal mobilisation therapy, muscle activity, thoracic spine, trapezius muscle, and neurophysiology. Literature was only used if the sources were reputable journal articles or published textbooks. In addition to this, literature older than ten years was avoided as much as possible to ensure that information was relevant and up-to-date.

2.2 Overview of the Thoracic Spine

The thoracic spine is the second segment of the vertebral column and forms a transitional area between the base of the neck, superiorly, and the lumbar spine, inferiorly. Anatomically, this spinal region extends from vertebral levels T1 to T12 respectively. This spinal segment is a complex area of the spine as it not only forms a framework to support the back, but also has the responsibility of protecting the organs within the thorax along with facilitating movements of the scapulae (Moore, Dalley and Agur 2014). Due to the chief responsibility of protecting the viscera within the thorax, segmental spinal mobility is a second priority indicating that the thoracic spine is the least mobile segment of the vertebral column (Gkasdaris et al. 2016).
2.2.1 Thoracic Vertebrae

There are twelve thoracic vertebrae that form the thoracic spine. Collectively, these vertebrae form a kyphotic curve in this spinal region. The main feature of thoracic vertebrae that distinguishes them from other vertebrae is the presence of costal facets which provide an attachment site for the ribs (Waxenbaum and Futterman 2018). Thoracic vertebrae are divided into two groups: atypical and typical thoracic vertebrae. Vertebrae T5 – T8 are considered to be the most typical because they possess features that are mutual with one another (Moore, Dalley and Agur 2014).

Features of typical thoracic vertebrae include:

- The presence of a vertebral body which is heart-shaped and increases in size as the vertebral level descends down the spine. In addition to this, four smooth, posterolateral costal facets are found bilaterally on the superior and inferior aspect of each vertebral body. These are known as demi-facets. The superior demi-facet is positioned to articulate with the head of the same rib that articulates with the inferior demi-facet of the vertebra above.
- A vertebral arch which unites to form a spinous process. Thoracic spinous processes are sloped posteroinferiorly to overlap the vertebra below. These processes are long in comparison to spinous processes from other spinal segments.
- Bilateral pedicles which project symmetrically to form the transverse processes. The transverse processes are broad and symmetrical in order to serve as a secure site for muscle attachment but decrease in size as they descend down the vertebral column. Costal facets are present on the end of each transverse process to allow for articulation with the tubercle of its respective rib. The articular processes are vertical and form an arc, in the centre of the intervertebral discs (IVD), with the articular facets which are coronally orientated.
The atypical thoracic vertebrae include T1 – T4 as well as T9 – T12 (Moore, Dalley and Agur 2014):

- **T1 – T4** share selected features of cervical vertebrae. However, T1 is the most unique due to the fact that the superior costal facets are complete and articulate with the first rib in isolation. Inferiorly, T1 possesses typical demi-facets present which articulate with the second rib. The spinous process has similar features to the seventh cervical vertebra – it is long and virtually horizontal.

- **T9 – T12** vertebrae share selected features of lumbar vertebrae. T11 and T12 are, specifically, considered to be atypical thoracic vertebra because they do not have any costal facets present on their transverse processes – they only have one pair of costal facets which articulate with their respective ribs. In rare cases, T10 will bear a resemblance to these vertebrae and will be considered as atypical as well.

- **T12** predominantly signifies the transitional area between the thoracic and lumbar regions of the spine. Therefore, it possesses characteristics of both regions. Its thoracic features include the presence of costal facets and superior articular facets that face posterolaterally in order to allow for rotation to occur. However, it possesses features of lumbar vertebrae inferiorly as the inferior articular processes are positioned to only allow for flexion and extension. In addition to this, there are mammillary processes present on the posterior aspect of the superior articular
processes in order to serve as an attachment site for multifidus and intertransversarii muscles.

2.2.2 Joints of the Thoracic Spine

2.2.2.1 Facet Joints

Facet joints (otherwise known as zygapophyseal joints) are plane synovial joints where the superior articular processes articulate with the inferior articular processes of the vertebra above (Moore, Dalley and Agur 2014). In the thoracic spine, they are angulated 60 degrees to the transverse plane and 20 degrees to the frontal plane and allow for gliding movements to occur between the corresponding articular processes (Waxenbaum and Futterman 2018). They are innervated by the medial branches of the posterior rami of the spinal nerves that come from the spinal level above and below the facet joint. There is an abundant supply of sensory innervation which is why dysfunction in these joints are more likely to produce pain (Crossman and Neary 2015). In addition to this, there are also mechanoreceptors situated within the thoracic facet joints which assist in keeping the thoracic vertebrae aligned.

![Figure 2.2 Facet joints (Moore, Dalley and Agur)](image)

Each facet joint is surrounded by a thin capsule which is filled with synovial fluid in order to allow for smooth articulation. However, the movement that occurs in the thoracic spine is minimal in comparison to other regions of the spine due to the positioning of the ribs and spinous processes; in addition to the orientation of the facet joints which collectively limit the amount of flexion and extension in the thoracic spine to a great extent. The joint capsule of the facet joint unites the surfaces of the articular processes between adjacent vertebrae in order to form a compliant connection between all thoracic vertebrae and IVD’s which also assists in limiting hyper-extension.
and flexion which in turn protects the spinal cord from mechanical damage (Moore, Dalley and Agur 2014).

2.2.2.2 Costotransverse Joints

There are two components that allow for articulation between a typical rib and the vertebral column to occur posteriorly. These include the costotransverse joints and the joints that are related to the head of the. Rib heads articulate with the superior costal facets of the corresponding vertebra, the inferior costal facet of the vertebra above as well as the IVD uniting the two adjacent vertebrae. There is an intra-articular ligament at the head of the rib which joins the rib crest of the rib head to the IVD and divides the joint space into two synovial cavities. On the anterior margin of the rib head, the joint capsule forms a radiate ligament which spreads out to the sides of the respective two adjacent vertebrae and their uniting IVD. Due to this close relationship between the rib heads and the vertebral bodies, the demi-facets only allow for a minimal amount of pivoting of the rib heads to occur. Nonetheless, the smallest amount of movement in this region will produce a sizable amount of movement at the anterior aspect of the rib.

![Figure 2.3 Articulations of the costovertebral joints](Moore, Dalley and Agur 2014)

Movements that occur in the costotransverse region are restricted by the costotransverse ligaments that attach laterally to the vertebral arches of the vertebrae. These ligaments have thin joint capsules and also provide strength to the joint
complex. There is an anterior costotransverse ligament that passes from the neck of the rib to the transverse process which strengthens the costotransverse joint anteriorly. In addition to this, there is a lateral costotransverse ligament that connects the tubercle of the rib to the tip of the transverse process which supports the posterior aspect of the joint. The superior costotransverse ligament passes from the neck of the rib to the superior aspect of the transverse process. It can be divided into a strong, anterior costotransverse ligament which limits the amount of gliding that occurs as well as a weak, posterior costotransverse ligament. Ribs 1 – 6 have convex tubercles and fit into the concavities of the transverse processes in order for rotation to occur predominantly in a transverse axis that crosses the intra-articular ligament and the head and neck of the rib. In contrast, the tubercles of ribs 7 – 10 are flat in order to allow for gliding within the costotransverse joint.

![Figure 2.4 Articulations of the costotransverse joints (Moore, Dalley and Agur 2014)](image)

**2.2.3 Muscle Overview**

Skeletal muscle is an important component of the MSK system – particularly in the vertebral column. According to Cramer and Darby (2014), musculature of the spine is crucial for postural control, spinal movements and spinal stabilisation through their contractions which results in a rigid support system around the spine. However, the most crucial role that thoracic muscles have is their involvement in respiration. Any muscle imbalances and consequential abnormal length-tension relationships will perpetuate abnormal breathing patterns (Tortora and Derrickson 2014). There are many muscles found in the thoracic region and these include the supraspinatus, infraspinatus, teres minor and subscapularis (i.e. rotator cuff muscles), the trapezius, rhomboids, levator scapulae and serratus anterior (i.e. the scapular stabilisers) as well
as the erector spinae, latissimus dorsi and quadratus lumborum (Moore, Dalley and Agur 2014).

The principal muscles involved in movements of the thoracic spine are the levator scapulae, trapezius, rhomboid muscles as well as the erector spinae muscles (Yang et al. 2015). The upper thoracic spine also contributes to the movements of the neck through action of the levator scapulae and trapezius muscles. Lastly, the group of muscles that are responsible for producing scapular movements include the trapezius, levator scapulae, rhomboid, serratus anterior and pectoralis minor muscles; collectively referred to as the scapulothoracic muscles (Castelein et al. 2015). Hence, the trapezius muscle plays a crucial role in thoracic spine and its mobility. The trapezius muscle is responsible for dynamic movements which include lateral flexion and rotation of the cervical spine, shoulder elevation and depression as well as internal rotation of the upper limb. However, this muscle is still primarily a postural muscle (Moore, Dalley and Agur 2014).

2.2.3.1 The Trapezius Muscle

The trapezius muscle is the most superficial muscle of the upper back. It is one of the broadest, triangular, muscles of the upper back and trunk. The trapezius muscle is relatively flat and occurs bilaterally on each side of the spine. The fibres are long and extend over a great surface area of the cervical and thoracic spine which is why it is considered to be the most superior back muscle (Cramer and Darby 2014). The muscle has many attachments due to the large amount of actions, and consequential dynamic movements, that it is responsible for. The trapezius is most commonly divided into three divisions, based on the directions of the fibres. These divisions are known as the upper fibres which have a descending orientation, middle fibres which are virtually horizontal and the lower fibres which are in an ascending direction (Moore, Dalley and Agur 2014).

Literature from Simons et al. (1999) outline the anatomy and functions of the different divisions. The upper fibres attach, proximally, to the medial third of the superior nuchal
line, external occipital protuberance and the ligamentum nuchae. The fibres attach distally to the posterior aspect of the medial third of the clavicle by converging anterolaterally. The upper fibres are, unilaterally, responsible for ipsilateral lateral flexion and extension and extreme contralateral rotation of the head and neck. Bilaterally, they are responsible for extension of the head and neck. In addition to this, these fibres are also responsible for elevation of the scapulae, retraction of the clavicles and elevation and rotation of the glenoid fossa.

The middle fibres attach to the spinous processes and interspinous ligaments of C6 – T3 vertebrae proximally. They converge laterally to their distal attachment which is the medial border of the acromion and the superior lip of the spine of the scapula. The primary function of these fibres is scapular retraction.

![Image](image_url)

**Figure 2.5 The trapezius muscle (Simons 1999)**

Proximally, the lower fibres attach at the spinous processes and interspinous ligaments of T4 – T12 vertebrae. They converge laterally to attach, distally, to the tubercle at the medial end of the spine of the scapula. The lower fibres are responsible for scapular retraction and depression as well as superior rotation of the glenoid fossa.
2.2.3.2 Nerve Supply Relevant to the Trapezius Muscle

The trapezius muscle receives its somatosensory and proprioceptive innervation from cervical nerve roots C2 – C4 of the cervical plexus (Gavid et al. 2016). The motor innervation comes from cranial nerve (CN) XI, also known as the spinal accessory nerve. According to Moore, Dalley and Agur (2014), this CN ascends into the cranial cavity via the foramen magnum. It emerges from the nucleus of the spinal accessory nerve, a column of ventral horn motor neurons, as a series of nerve rootlets in the superior five or six cervical divisions of the spinal cord. It, briefly, joins with the vagus nerve as they pass through the jugular foramen to exit the cranial cavity. They then separate as the spinal accessory nerve descends along the internal carotid artery where it penetrates and innervates the sternocleidomastoid muscle. Following this, the muscle emerges near the centre of the posterior border of the sternocleidomastoid muscle and crosses the posterior cervical region, the C2 – C4 spinal nerve roots join the spinal accessory nerve at this point. It continues to pass deep to the superior border of the trapezius muscle – where it resumes to descend along the deep surface of the trapezius muscle, providing multiple motor branches to the muscle, respectively.

Figure 2.6 Distribution of the spinal accessory nerve (Moore, Dalley and Agur 2014)
2.3 Overview of the Nervous System

The nervous system is the most crucial organ system in the body due to its responsibility of perceiving and processing details from external and internal environments in order to determine how the body should respond appropriately (Crossman and Neary 2015). Hence, it is the primary system in the human body that relates SMT and the body’s response in terms of how it will react to the change of the environment internally and externally subsequent to joint manipulations. Naish, Revest and Syndercombe Court (2015) state that the nervous system is anatomically divided into the central nervous system (CNS) and the peripheral nervous system (PNS).

2.3.1 The Peripheral Nervous System

The PNS is made up of nerves outside the CNS and have the function of directing nerve impulses to and from the CNS. It is further divided into the autonomic nervous system which is innervates smooth muscle, specialised effector cells and glands and the somatic nervous system which entails the sensory and motor pathways of skin, joints and skeletal muscle (Naish, Revest and Syndercombe Court 2015). The somatic nervous system is bilaterally symmetrical because it is made up of twelve pairs of CN’s and thirty-one pairs of spinal nerves. The CN’s primarily innervate structures in the head to transmit special senses which include hearing, smell, vision and taste. However, CN X (vagus nerve) conveys information from organs in the abdomen and thorax and CN XI provides motor innervation to the trapezius and sternocleidomastoid muscles (Cramer and Darby 2014).

Each spinal nerve emerges from the spinal cord segmentally where they travel through the intervertebral foramen at their respective level and each spinal nerve is named based on the region and spinal level at which they emerge. These nerves are responsible for connecting the CNS to the sensory receptors found throughout the body. These receptors are found in joints, tendons, skin, viscera, muscles and all effector organs. A spinal nerve is made up of a dorsal and ventral root which carry
sensory and motor fibres respectively; these can be somatic or autonomic (Mai and Paxinos 2012; Moore, Dalley and Agur 2014).

2.3.2 Peripheral Nerves

Neurons are the structural units that make up peripheral nerve fibres. According to (Cramer and Darby 2014), neurons function by generating and transmitting action potentials which travel rapidly from one neuron to the next by means of synapses; the specialised junctions between two interfacing neurons. These action potentials are essentially electrical signals that are formed when the cell membrane of a neuron depolarises. This occurs because there are ion channels within the cell membrane which can be opened in two ways. The cell membrane can be opened when neurotransmitters bind to a receptor in, or near, the ion channel or when there is a change in voltage across the cell membrane. When the ion channels open, there is an influx of sodium which is what causes the membrane to depolarise (Mai and Paxinos 2012). There are three different types of neurons that can make up a peripheral nerve fibre: a sensory neuron, motor neuron or interneuron. 

Sensory receptors can be categorised based on the different types of stimuli that they detect.

Tortora and Derrickson (2014) classify sensory receptors in the following way:

- **Mechanoreceptors** – these receptors respond to mechanical stimuli that result in the deformation of cells which include stretching, pressure or bending. Therefore, they provide sensations of pressure, touch, hearing, equilibrium, proprioception and vibration.
- **Thermoreceptors** – responsible for detecting temperature changes.
- **Nociceptors** – these receptors respond to chemical and/or physical damage. Hence, they are responsible for detecting painful stimuli.
- **Photoreceptors** – responsible for detecting light that hits the retina of the eye.
- **Chemoreceptors** – respond to chemical changes in the mouth, nose and body fluids.
• Osmoreceptors – these receptors respond to changes in the osmotic pressure of body fluids.

**2.3.2.1 Somatic Sensations**

For the purposes of this study, somatic sensations will be discussed. Somatic sensations occur in response to the stimulation of sensory receptors that are rooted in the skin or subcutaneous layer throughout the human body. Somatic sensory receptors have an asymmetrical distribution throughout the body; some regions in the body will have more receptors present in comparison to others (Tortora and Derrickson 2014; Crossman and Neary 2015).

Tortora and Derrickson (2014) demonstrates that there are four mechanisms whereby somatic sensations occur and these are tactile, thermal, pain and proprioceptive. Tactile sensations of pressure, touch and vibration arise from the stimulation of mechanoreceptors that are attached to large, myelinated A fibers. Tactile receptors found in the skin and subcutaneous layer include Meissner corpuscles, hair root plexuses, Merkel discs, Ruffini corpuscles, Pacinian corpuscles and free nerve endings. Thermal sensations are mediated by two types of thermal receptors. Cold receptors (10 – 40 degrees Celsius) are found in the stratum basale of the epidermis and are mainly attached to myelinated A fibers. Warm receptors (32 – 48 degrees Celsius) are found in the dermis and are attached to unmyelinated C fibres.

Nociceptors are responsible for pain sensations; they are found in all tissues of the body except for the brain. Any overstimulation of thermoreceptors, mechanoreceptors or chemoreceptors have the potential to activate nociceptors. There are two types of pain. Fast pain (sharp pain) occurs within 0.1 second after the painful stimulus and is propagated via myelinated A fibers. Fast pain is limited to the superficial body tissues. Slow pain (burning pain) occurs more than one second after the painful stimulus and this type of pain is propagated by unmyelinated C fibers. Slow pain can occur superficially and in deeper tissues of the body.
Proprioceptive sensations give the body the ability to perceive location, movements and actions of the body parts without looking at them. This allows for balance and co-ordinated movements to occur. These sensations arise from proprioceptors which are found in tendons, ligaments, muscles and hair cells of the inner ear. In addition to this, proprioceptors also allow for the body to perceive the weight of an object in order to regulate the muscular effort required to perform different tasks. These four modalities are summarised in table 2.1 below.

Table 2.1 Summary of sensory receptors responsible for somatic sensations

<table>
<thead>
<tr>
<th>Receptor Type</th>
<th>Location</th>
<th>Sensation</th>
<th>Adaptation Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>TACTILE RECEPTORS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meissner Corpuscles</td>
<td>Dermal papillae of hairless skin</td>
<td>• Touch • Pressure • Slow vibrations</td>
<td>Rapid</td>
</tr>
<tr>
<td>Hair Root Plexuses</td>
<td>Hair follicles in the skin</td>
<td>• Touch</td>
<td>Rapid</td>
</tr>
<tr>
<td>Merkel Discs</td>
<td>Epidermis</td>
<td>• Touch • Pressure</td>
<td>Slow</td>
</tr>
<tr>
<td>Ruffini Corpuscles</td>
<td>Dermis and in ligaments and tendons</td>
<td>• Stretching of the skin</td>
<td>Slow</td>
</tr>
<tr>
<td>Pacinian Corpuscles</td>
<td>Dermis and subcutaneous layer, submucosal tissues, joints, periosteum and some viscera</td>
<td>• Pressure • Fast vibrations</td>
<td>Rapid</td>
</tr>
<tr>
<td><strong>THERMORECEPTORS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hot and Cold Receptors</td>
<td>Skin and mucous membranes of the mouth, vagina and anus</td>
<td>• Heat • Cold</td>
<td>Initially rapid, then slow</td>
</tr>
<tr>
<td><strong>PAIN RECEPTORS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nociceptors</td>
<td>All body tissues except the brain</td>
<td>• Pain</td>
<td>Slow</td>
</tr>
<tr>
<td><strong>PROPRIOCEPTORS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muscle Spindles</td>
<td>Intrafusal muscle fibres of most skeletal muscle</td>
<td>• Muscle length</td>
<td>Slow</td>
</tr>
<tr>
<td>Golgi Tendon Organs</td>
<td>Tendons and ligaments</td>
<td>• Muscle tension</td>
<td>Slow</td>
</tr>
</tbody>
</table>
Joint Kinesthetic Receptors | Pacinian corpuscles, Ruffini corpuscles, golgi tendon organs and free nerve endings | Joint position | Movement | Rapid

(Adapted from Cramer and Darby 2014; Tortora and Derrickson 2014).

2.4 Physiology of Skeletal Muscle

2.4.1 Introduction

Skeletal muscles have a large amount of functions in the human body and make up approximately 45% of the human body mass. Anatomically, skeletal muscles are the kinematic links that connect the entire MSK system because they attach to different bones via tendons. Skeletal muscle is a pliable body tissue that continuously modifies to use, ageing, malignancies and disease (Jefferies et al. 2017).

Skeletal muscle is best-known for its contractibility and ability to create movement. In addition to this, skeletal muscle is responsible for the ceasing of movement – for example resisting gravity in order to maintain the body's posture and maintaining joint stability. This is achieved by the constant adaptation of skeletal muscle in order to maintain optimal length-tension relationships between adjacent skeletal structures. These tissues are found throughout the body at sphincters in order to control swallowing, urination and defecation. These muscles also form a barrier against external trauma in the abdominal and pelvic regions by protecting and supporting the weight of the internal organs (Marieb 2015). Skeletal muscle is thermogenic which means that it also contributes to the maintenance of homeostasis. This is achieved by the breakdown of Adenosine Triphosphate (ATP) which generates heat; this is particularly evident during exercise when sustained muscle contractions increases body temperatures as well shivering in cold conditions (Jefferies et al. 2017).

2.4.2 Morphology

Skeletal muscle fibres have a sarcomeric arrangement of proteins which is why these muscles are also known as striated muscles – they present with a striated appearance microscopically. These fibres are multinucleated and vary in biochemical and
physiological properties between different muscles. These are the two histological features that distinguish skeletal muscle from cardiac and visceral muscle. Motor neurons which are usually found towards the centre of the muscle have branched axons that innervate different fibres but each muscle fibre only receives innervation from one motor neuron and acts as a single unit (Costanzo 2014; Jefferies et al. 2017).

According to Hall and Guyton (2011), skeletal muscles are composed of many fibres that are further made up of smaller subunits. In most cases, each fibre extends the entire length of the muscle. There is a thin membrane that encloses each muscle fibre and it is known as the sarcolemma. The sarcolemma is composed of a plasma membrane and an outer thin layer of polysaccharides which contain thin collagen fibrils. This is where action potentials are conducted. At the end of each muscle fibre, the sarcolemma fuses with a tendon fibre which forms bundles and collectively becomes known as the muscle tendon – this is where muscles will attach to bone.
As illustrated in figure 2.8, each muscle fibre includes several myofibrils which are composed of, thin, actin and, thick, myosin filaments. Myofibrils are surrounded by a smooth endoplasmic reticulum which is known as the sarcoplasmic reticulum. These filaments are large, rod-like, polymerised protein molecules that give skeletal muscle its contractility characteristic. The sarcolemma has numerous transverse tubules which are invaginations that penetrate the cytoplasm to surround each myofibril. Terminal cisternae are found neighbouring the transverse tubules and are responsible for storing calcium ions that are used during muscle contraction (Hall and Guyton 2011; Tortora and Derrickson 2014).
On a microscopic level, it is noted that actin and myosin filaments partially interdigitate which results in the appearance of alternating light and dark bands. The light bands (I-bands) only contain actin filaments and are isotropic to polarised light. The dark bands (A-bands) contain myosin filaments and the portion of actin filaments that overlap with the myosin filaments; these bands are anisotropic to polarised light.

![Diagram of filaments within a sarcomere](image)

**Figure 2.8 Arrangement of filaments within a sarcomere (Tortora and Derrickon)**

Cross-bridges present on the ends of myosin filaments and the interaction between adjacent cross-bridges will result in muscle contraction (Hall and Guyton 2011).

Ultimately, a sarcomere is the functional unit of a muscle and refers to the portion of a muscle fibre found between two successive Z-discs. Z-discs are found at the end of actin filaments and transversely connect adjacent myofibrils across the entire muscle fibre. These discs are comprised of filamentous proteins that differ chemically from both actin and myosin. H-bands are found in the middle of the A-bands and only contains myosin filaments. In the middle of the H-band there is an M-band present which is a dense region that denotes the middle of the sarcomere.
According to Tortora and Derrickson (2014), myofibrils are composed of three different types of proteins which include:

- **Contractile proteins** – responsible for force generation during muscle contraction.
- **Regulatory proteins** – these proteins initiate and inhibit the contraction processes.
- **Structural proteins** – responsible for keeping myofibrils aligned, providing elasticity and extensibility properties to the myofibrils and acting as a network between myofibrils, sarcolemma and extracellular matrix.

Actin and myosin are the two contractile proteins. Myosin is found in the thick filaments and operates as a motor protein in skeletal, smooth and cardiac muscle. It generates force by converting ATP, which is a form of chemical energy, into mechanical energy. Each filament has the appearance of two golf clubs that are twisted together – the tails point towards the M-band and lie parallel to one another. The myosin heads spiral outwards. Actin molecules combine to form a thin filament and are attached to the Z-discs. Each actin molecule has a myosin-binding site in order for the myosin heads to attach here.

There are two regulatory proteins found in the thin filaments: tropomyosin and troponin. Tropomyosin is responsible for inhibiting muscle contraction by blocking the myosin-binding sites during muscle relaxation. Strands of tropomyosin are anchored to troponin molecules. When troponin binds with calcium ions, it changes shape and pulls tropomyosin molecules off the myosin binding sites which, successively, initiates muscle contraction.

The principal structural proteins found in skeletal muscle include titin, α-actinin, myomesin, nebulin and dystrophin. Titin is 50 times larger than an average-sized protein. This molecule spans from a Z-disc to an M-line (i.e. half of a sarcomere) in order to stabilise the position of the thick filament, prevent overextension of the sarcomere and to maintain the central location of the A-band. Titin is very elastic and accounts for most of the elasticity and extensibility characteristics in muscle fibres. α-
actinin molecules are found in the dense component of the Z-discs and these proteins bind to the molecules of the thin filament as well as titin. Myomesin is what the M-band is composed of – these proteins bind to titin and link adjacent thick filaments. Nebulin wraps around the entire length of each filament and it is a long, non-elastic protein. The primary functions of nebulin include anchoring the thin filaments to the Z-discs as well as regulating the length of the thin filaments during growth and development. Dystrophin connects the thin filaments of the sarcomere to the membrane proteins of the sarcolemma, which are sequentially attached to the proteins found in the connective tissue extracellular matrix. The main functions of dystrophin are transmitting the tension that is generated by the sarcomeres to the tendons as well as strengthening the sarcolemma.

2.4.3 Motor Unit

Jefferies et al. (2017) states that a functional motor unit is comprised of an alpha motor neurons anterior horn cell, axon and the muscle fibre that it innervates. A motor unit is the smallest element that is regulated by the CNS as all muscle contractions are voluntary. The neuromuscular junction is the specialised region of a muscle fibre where the axon interacts with the muscle fibre. At the neuromuscular junction action potentials depolarise the muscle fibre’s plasma membrane which provokes a release of calcium, leading to muscle contraction and, in turn, movement. The number and location of motor units in a muscle varies based on the size and function of the muscle (Moore, Dalley and Agur 2014; Tortora and Derrickson 2014).

2.4.4 Skeletal Muscle Contraction

The Sliding Filament Mechanism is often used to demonstrate the microscopic processes that take place (Tortora and Derrickson 2014). As depicted in Figure 2.9 below, this mechanism takes place due to myosin heads that attach along the thin filaments at both ends of the sarcomere, subsequently tugging the thin filaments towards the M-line.
This pull towards the midline causes the thin filaments to slide towards the middle of a sarcomere, resulting in the Z-discs coming closer to one another in order for the sarcomere to shorten. It is important to note that the actual filaments do not change in length – it is the sarcomeres which shorten the entire muscle fibre which sequentially shortens the full muscle.

Nonetheless, skeletal muscle contractions are initiated at a molecular level when an action potential arrives at the axon terminal within the neuromuscular junction and depolarises the plasma membrane of the muscle (Hall and Guyton 2011; Tortora and Derrickson 2014). This depolarisation process opens the calcium channels to allow the calcium in the extracellular fluid to enter the voltage-gated channels and diffuse into the axon terminal. Once in the axon terminal, the calcium binds to proteins that allow the membranes of the vesicles, containing Acetylcholine (Ach), to bind with the neuronal plasma membrane. In doing so, Ach is dispersed into the extracellular cleft where it, in turn, diffuses into the motor end plate to bind to the ionotropic receptors which opens ion channels in each respective receptor protein and, consequently, opens the channel for sodium and potassium ions to pass through. The difference in electrochemical gradients leads to an influx of sodium ions which produces a positive charge within the muscle fibre. This initiates depolarisation of the motor end plate,
known as an endplate potential. As a result, a muscle fibre action potential transmits along the sarcolemma into the T-tubules, causing the sarcoplasmic reticulum to release its stored calcium into the sarcoplasm.

Figure 2.10 Calcium release and uptake during skeletal muscle contraction and relaxation (Tortora and Derrickson 2014)

The calcium binds to troponin and this changes the shape of tropomyosin. In doing so, the inhibitory control of tropomyosin is reduced, and it moves away from the myosin-binding site in each of the actin molecules. The energised myosin cross-bridge binds to a thin actin filament and moves in an arc – causing the overlapping thick and thin filaments in each sarcomere to pass one another. This motion of many cross-bridges forces the thin filaments which are attached to the Z-line to move towards the centre of the sarcomere, causing it to shorten. The I and H bands shorten, but the A-bands do not change, and this is known as a power stroke. If the muscle fibre remains activated, each cross-bridge repeats its swivelling motion which results in the Sliding Filament Mechanism depicted in Figure 2.9 above.

As illustrated in Figure 2.11 above, the contraction cycle continues as ATP binds to myosin and results in detachment of the cross-bridges. The myosin-bound ATP is hydrolysed which, as a result, restructures the energised state of the myosin and returns it to its original position. Movement of the cross-bridges and hydrolysis of the ATP do not occur simultaneously. The cross-bridge can only reattach to a new actin molecule if there is still calcium present in the sarcomere – in this case, the cross-
bridge cycle will be repeated (Cramer and Darby 2014; Tortora and Derrickson 2014). Relaxation of skeletal muscle is initiated when calcium ions begin to return to the sarcoplasmic reticulum. The calcium that was bound to the troponin is removed and the grip of the tropomyosin restores as a result.

![Figure 2.11 The cross-bridge cycle of skeletal muscle contraction (Tortora and Derrickson 2014)](image)

2.4.5 Role of Muscle Spindles and Golgi Tendon Organs

Muscle spindles and Golgi Tendon Organs (GTO) are sensory receptors that are responsible for intrinsic muscle control and they, almost totally, operate involuntarily. These receptors convey information to the spinal cord, cerebellum, and the cerebral cortex to control muscle contraction (Hall and Guyton 2011; Marieb 2015).
2.4.5.1 Muscle Spindles

Muscle spindles are fusiform proprioceptors that are distributed along the extrafusal skeletal muscle fibres. Each spindle consists of a bundle of intrafusal fibres and is enclosed within a connective tissue sheath (Marieb 2015). The connective tissue sheath secures the muscle spindles to the perimysium and endomysium of the extrafusal muscle fibres. These sensory receptors monitor any stretch that is placed on skeletal muscle and initiate a reflex to resist the stretch, thus, preventing overstretching of the muscles and muscle fibre damage consequently (Hall and Guyton 2011; Crossman and Neary 2015; Marieb 2015).

The intrafusal muscle fibres within the muscle spindles are modified skeletal muscle fibres – there are few to no actin and myosin filaments in the midsection of the fibre between the two ends, making their primary function that of sensory perception. There are two types of intrafusal muscle fibres: annulospiral endings and flower-spray endings. Annulospiral endings are nuclear chain fibres associated with type Ia sensory fibres and react to the nature in which a muscle is stretched and are wrapped around
the midportion of the intrafusal muscle fibres. Flower-spray endings, otherwise known as secondary endings, are nuclear bag fibres associated with sensory type II fibres that form a bundle of thin, radiating, branches in the midportion of the intrafusal muscle fibre. These nerve endings react to the quantity and speed of a stretch placed on the muscle. Along with the sensory receptors, the contractile distal ends of the intrafusal fibres are innervated by gamma motor neurons. Hence, muscle spindles are able respond to a change in length of the entire muscle and when the end portions of the intrafusal muscle fibres contract as these stretch the midportion of the entire muscle spindle (Hall and Guyton 2011; Tortora and Derrickson 2014).

Ultimately, when a muscle is stretched, the stretch reflex ensues to avoid overstretching of the muscle. This reflex occurs when the muscle spindle becomes elongated, this places a stretch on the intrafusal muscle fibres. The stretching at the central portion of the intrafusal fibres increases the firing rate of the type Ia sensory fibres which conveys an action potential to the spinal cord. When this information reaches the spinal cord, the sensory neuron within the spinal cord synapses with an alpha motor neuron which stimulates the extrafusal muscle fibres – causing a reflex contraction and subsequent shortening of the respective muscle (Tortora and Derrickson 2014). Reciprocal inhibition is a reflex mechanism that needs to occur simultaneously with the stretch reflex in order for it to be effective in preventing muscle tissue injury (Marieb 2015). This is achieved by an axon which branches off the muscle spindle – known as an axon collateral. The axon collateral synapses with an inhibitory interneuron in the spinal cord. Following this, the interneuron, synapses with, and inhibits, the motor neuron that customarily innervates the antagonistic muscles. Subsequently, when the stretch reflex occurs and the stretched muscle contracts, the antagonistic muscles opposing the contraction are stimulated to relax. These axon collaterals also transmit nerve impulses to the brain along ascending spinal pathways to alert the brain of the reflexes that have occurred and to co-ordinate skeletal muscle movements accordingly (Tortora and Derrickson 2014).
The gamma loop refers to the course that involves the gamma motor neurons, alpha motor neurons, Ia sensory fibres, intrafusal muscle fibres and extrafusal muscle fibres and it is essential for the preservation of stretch reflexes and refined adjustments in muscle activity (Korr 1975; Tortora and Derrickson 2014). When a muscle actively contracts against a load, an action potential is transmitted from the CNS to the alpha motor neurons – initiating muscle contraction of the extrafusal muscle fibres. Concurrently, impulses are sent to the gamma motor neurons of the intrafusal muscle fibres, and they contract as well. Hence, the intrafusal muscle fibres shorten whenever the extrafusal muscle fibres do as their gamma motor neurons are activated simultaneously. Therefore, the centre regions of the intrafusal fibres are nearly always under tension because contraction of the intrafusal fibres stretches the central region and increases the firing rate of the type Ia sensory fibres. This mechanism is known as alpha-gamma coactivation and is what gives muscle spindles their ability to indicate changes in muscle length when muscle contractions occur (Korr 1975; Hall and Guyton 2011; Tortora and Derrickson 2014; Crossman and Neary 2015).

2.4.5.2 Golgi Tendon Organs

Golgi tendon organs (GTO) are mechanoreceptors located at the junctions between skeletal muscle and their tendons – near its insertion point. These receptors are comprised of small bundles of collagen fibres enclosed within a connective tissue capsule – sensory nerve endings penetrate this capsule coil between and around these collagen fibres (Marieb 2015). The GTO initiates tendon reflexes to prevent excessive tension within the tendons and their respective muscles, protecting them against damage (Cramer and Darby 2014; Tortora and Derrickson 2014). Similar to the muscle spindles, GTO’s have both a static a dynamic response when there is a change in the length of the muscle – the rapid response when there is a sudden change in muscle tension, yet subsiding within a fraction of a second to a steady-state proportional to the muscle tension (Hall and Guyton 2011).

The tendon reflex occurs as a feedback mechanism and is responsible for controlling muscle tension by initiating muscle relaxation before the muscle contraction exceeds
its physiological barrier and a tendon is torn. The tendon reflex ensues when contraction or stretching of a muscle increases the tension within the tendon – this stretches and stimulates the GTO. In response to this, the GTO transmits nerve impulses to the spinal cord via type Ib sensory nerve fibres. These impulses synapse with interneurons within the spinal cord and the interneurons, sequentially, inhibit the alpha motor neurons innervating the respective muscle (Hall and Guyton 2011; Tortora and Derrickson 2014).

Once again, reciprocal inhibition needs to occur, it is essential for the co-ordination of body movements and avoids inconsistency between opposing muscles (Marieb 2015). While the tendon reflex occurs, the type I b sensory neurons simultaneously synapse with an excitatory interneuron within the spinal cord which, in turn, innervates the gamma motor neurons of the antagonistic muscles to contract. Therefore, while the tendon reflex initiates relaxation of a muscle, reciprocal inhibition will trigger the contraction of the antagonistic muscles (Tortora and Derrickson 2014).

2.5 Surface Electromyography and Muscle Activity

2.5.1 Surface Electromyography

According to Chowdhury et al. (2013), surface electromyography (sEMG) refers to the collective electrical signal from muscles, that is controlled by the nervous system and produced during muscle contraction. An sEMG signal represents the electrical activity of a muscle’s motor units and their action potentials within the area where the electrodes are placed. It is a non-invasive, experimental approach to understanding the human body’s responses under normal and pathological conditions by recording and analysing the electrical signals associated with muscle activity (Lehman 2012).

Surface electromyography (sEMG) is an objective way to evaluate and understand the intentional and reactive motor behaviours of skeletal muscle. It is an effective way to communicate evidence concerned with muscle activations (e.g. the intensity of muscle contractions), the myoelectric exhibition of muscle fatigue as well as the pattern of recruitment of motor units (Cavalcanti Garcia and Vieira 2011). It is evident that joint
dysfunction and muscle dysfunction go hand in hand (Capobianco et al. 2018). Therefore, sEMG is an objective measurement tool used to assess the features of joint dysfunction.

Golabchi et al. (2019) used sEMG to evaluate aberrant muscle patterns during a functional examination. This was done in order to discover the primary causes of non-physiological postures and movement trajectories such as muscle hyperactivity, spasms or synergistic and/or antagonistic compensations amongst others. The sEMG recordings were collected from 62 participants, between the ages of 22 – 66 years, who performed functional evaluations in order to develop and characterise algorithms. These algorithms were then used in a case study evaluation of a patient with persistent back pain after a lumbar hardware removal. This was done to evaluate the suitability of the proposed technique. It was concluded that the accuracy of evaluating the severity of aberrant muscle patterns rely on electromyography (EMG) based measures. It revealed that it was suitable to stem clinically relevant information from EMG readings during functional assessments. Table 2.2 further demonstrates different circumstances where sEMG has been used effectively in experimental studies:

<table>
<thead>
<tr>
<th>Author</th>
<th>Sample Size</th>
<th>Study Design</th>
<th>Intervention and Aim</th>
<th>Outcome Measures</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abboud, Nougarou and Descarreaux (2016)</td>
<td>n = 23, asymptomatic with no history of LBP</td>
<td>RCT</td>
<td>1. 30-minute passive lower back creep deformation position.</td>
<td>EMG amplitude, median frequency and dispersion pre-test and post-test.</td>
<td>Fatigue-related changes in EMG median frequency observed.</td>
</tr>
<tr>
<td>Park et al. (2015)</td>
<td>n = 19, experiencing upper trapezius pain</td>
<td>RCT</td>
<td>1. Ipsilateral neck rotation (INR)</td>
<td>sEMG of upper-, middle- and lower trapezius muscles with and without INR and the Wilcoxon</td>
<td>INR reduced EMG readings in the upper trapezius and improved muscle imbalance</td>
</tr>
</tbody>
</table>
Based on the table above, it is evident that sEMG is the most appropriate tool to use when evaluating joint dysfunction in terms of altered muscle activity. The sEMG studies above were able to highlight the different clinical aspects that may be associated with altered muscle activity such as muscle fatigue and creep deformation, abnormal joint biomechanics and their respective kinematic chains, joint stability and muscle recruitment as well as altered postural positions – locally and remotely to the affected muscles. More so, the regularity of sEMG as a measurement tool particularly for the trapezius muscles are illustrated in the studies above due to the superficial anatomical position of the muscle.

When assessing muscle activity of the trapezius muscle with associated thoracic spine dysfunction, altered muscle activity of the middle and lower trapezius continues to correspond in the abovementioned studies. This reinforces the point made by
Muscolino (2017), that suboptimal functioning of the trapezius muscles can be a source of postural abnormalities, neck pain, shoulder pain, breathing discomfort and lower back pain. It evident that joint dysfunction and muscle dysfunction correspond with one another. Therefore, sEMG is the most effective measurement tool for assessing joint dysfunction in the thoracic spine as thoracic joint disfunction will, inherently, result in altered muscle activity in the trapezius muscle (Cavalcanti Garcia and Vieira 2011).

2.5.2 Muscle Activity

Muscle activity refers to the arbitrary, staccotic firing of action potentials of groups of muscle fibres (Crisswell 2010). There are many factors that can alter normal muscle activity such as MSK injuries resulting in joint dysfunction, motor disorders, electrolyte imbalances and level of fitness. Muscle activity is an important component that affects joint loading; abnormal muscle activity patterns will result in abnormal joint loading (Madadi-Shad et al. 2019). Therefore, abnormal muscle activity in the spine will result in abnormal loading of the spine and consequently biomechanical imbalances which, in turn, leads to joint dysfunction.

2.5.3 Muscle Activity in the Trapezius Muscle

The middle trapezius muscle is primarily responsible for adducting the scapula while the lower trapezius aids in this movement as well as depressing the scapula (Cramer and Darby 2014). Independent neural control of the different divisions of the trapezius muscle is, functionally, essential to maintain posture and controlled movements. Abnormal morphology and histological properties such as muscle hypertonicity or abnormal length-tension relationships of the trapezius muscle results in fatigue or pain which, in turn, will alter the afferent feedback loop and the muscle will then function on a suboptimal level (Madeleine et al. 2011). A study by Nicoletti, Spengler and Läubli (2014) focused on the Cinderella hypothesis which states that muscle fibres that undergo prolonged low-level activities, remain active the whole time. This study further emphasised that this hypothesis is often observed in the trapezius muscle – a lack of rest time overloads these fibres whether activity levels are low or high. There is a
strong correlation between sustained trapezius muscle activity and the development of neck pain or lower back pain as a compensatory mechanism (Nicoletti, Spengler and Läubli 2014). Surface electromyography (sEMG) is one of the best, non-invasive, ways to evaluate the muscle activity of the trapezius muscle as it is, anatomically, a superficial back muscle (Simons et al. 1999; Crisswell 2010).

2.6 Joint Dysfunction

A joint fixation or dysfunctional joint refers to the presence of functional alterations within a spinal motion segment that results in joint hypomobility in one or more directions (Bergmann and Peterson 2011). A study conducted by Kim et al. (2019) emphasises that a joint dysfunction is present when there are functional changes, limited ROM and positive findings of relevant orthopaedic tests; irrespective of the presence of pain. This implies that individuals with segmental joint dysfunction can be asymptomatic.

The fundamental characteristic of a joint dysfunction is intervertebral hypomobility (Henderson 2012). Fryer (2016) and Kim et al. (2019) both state that spinal pain is not a distinctive characteristic of joint dysfunction, and palpable signs of dysfunction are consistent in both symptomatic and asymptomatic patients. The clinical picture of a joint dysfunction includes, but is not limited to, the following components: palpable decreased intersegmental ROM, typified by abnormal joint play and hard end feel of a joint, hypertonicity of segmental paraspinal tissues, joint asymmetry of the relevant surrounding landmarks and tenderness to palpation of the affected joints (Bergmann and Peterson 2011; Fryer 2016).

It is evident that joint dysfunction results in altered joint biomechanics and consequential neuromuscular abnormalities which predisposes healthy individuals to pain and further dysfunction throughout the human body (Ebrahimi et al. 2018). Theoretically, abnormal joint biomechanics perpetuates abnormal biomechanical joint loading. This, in turn, results in abnormal firing of mechanoreceptors in paraspinal tissues which alters the muscle activity of these tissues – they become hypertonic.
(Capobianco et al. 2018; Madadi-Shad et al. 2019). Ultimately, joint dysfunction leads to increased muscle activity and, similarly, muscle hypertonicity predictably results in joint dysfunction. This concept is emphasised in a study conducted by Meyer et al. (2017) that exemplifies how joint dysfunction has the ability to distort afferent input to the CNS which, consequently, results in abnormal efferent outputs to the receptor organs. Eventually, the abnormal afferent input has the ability to lead to unfavourable neural changes in the CNS (Henderson 2012).

2.6.1 Joint Dysfunction and Thoracic Range of Motion

Assessing the ROM of both segmental joints and spinal regions is a fundamental component of a functional injury evaluation. It allows practitioners to identify appropriate interventions and provides feedback about the appropriateness of the intervention (Johnson et al. 2012; Buckup, Wiser and Buckup 2016).

The thoracic spine is the least mobile spinal region owing to the ribcage that attaches to thoracic vertebrae, however, inter-segmental angular movements occur within the thoracic spine. Morita et al. (2014) examined the functional ROM of the thoracic spine in the sagittal plane of 50 healthy patients. Preoperative myelography, multidetector-row, computed tomography scanning was performed at passive maximum flexion and extension of the thoracic spine in order to calculate the total and intersegmental kyphotic angles in addition to the ROM. The results evidenced that the thoracic spine showed a marked ROM in the sagittal plane.

Many aspects of thoracic spine ROM have been investigated and the common thread in literature reveals that the thoracic spine is most often overlooked as a source of pain attributable to the fact that thoracic spine dysfunction leads to non-specific pain that may or may not be experienced specifically in the thoracic spine (Heneghan and Rushton 2015).

The kinematic relationship between the cervical and thoracic spines was examined by Tsang, Szeto and Lee (2013) by evaluating the movement co-ordination between the
two spinal regions in 34 asymptomatic individuals. Electromagnetic motion sensors were placed anatomically onto the overlying skin of the head, T1, T6 and T12 spinous processes in order to measure the angular displacement of the cervical and thoracic spine during active neck movements. Findings revealed that the motion of the thoracic spine, particularly the upper thoracic region, has a significant contribution to neck mobility – implying that the thoracic spine should be considered when examining neck dysfunction.

The question of whether there is a noteworthy difference in the sagittal thoracic ROM in patients with and without shoulder impingement syndrome was focused on in a study by Theisen et al. (2010). Clinical and ultrasound topometric examinations were performed on 39 participants in the following postures: sitting up straight, sitting with maximal flexion and sitting with maximal extension. In addition to this, the disabilities of the arm, shoulder and hand and the constant scores were obtained through a self-assessment questionnaire as well as measurements of the lengthening and shortening dorsal projections of the in functional positions by tape with Ott’s sign. It was evident that the abnormal findings in the thoracic spine correlated with the group that was symptomatic to shoulder impingement syndrome.

A study conducted by Schinkel-Ivy and Drake (2019) investigated the interaction between thoracic movement and lumbar muscular co-contraction in 30 asymptomatic young adults. Participants were required to perform 10 repetitions of upright standing, maximum trunk ROM and thoracic movement tasks while keeping the lumbar spine in a relatively neutral position in order to measure the lumbar muscle activation. Lumbar co-contractions were calculated, compared between the different tasks and compared to the respective thoracic angles. The findings disclosed that there was a definitive interaction between the thoracic and lumbar spines which proposes that thoracic posture and ROM contributes to the lumbar spine mechanics and should be accounted for when examining the lumbar spine.
Based on the studies above, it is evident that joint dysfunction in the thoracic spine will decrease the range of motion in this spinal segment – altering overall thoracic spine mobility. Spinal mobilisation has proven to improve joint range of motion (Yang et al. 2015). Hence, the relevance of determining the effects of spinal mobilisation on muscle activity to contribute to the understanding of the mechanism responsible for the subsequential improved joint mobility.

### 2.6.2 Joint Dysfunction and Muscle Activity

As mentioned above, joint dysfunction further enables abnormal movement patterns in terms of muscle activation, muscle coordination, normal gait patterns and basic motor functions. This self-perpetuating cycle alters the integrity of joints which predisposes these MSK components to pain and degeneration (Capobianco et al. 2018). Muscular hypertonicity refers to an abnormally increased muscular resistance when a patient is consciously trying to maintain a relaxed state of muscle activity; there is an abnormal increase in resting muscle activity. This is attributed to an increased excitability of the muscle fibres. Evidently, hypertonic muscles demonstrate increased, involuntary, electromyographic activity that may or may not be painful (Bhimani, Gaugler and Skay 2017; Srivastava, Patten and Kautz 2019).

Korr (1975) uses the neurobiological concept of a facilitated segment, a reflex-based theory, to illustrate the effects of muscle hypertonicity on joint dysfunction. It is used to rationalise how a change in the kinesiopathology of a joint will alter its neuropathophysiology. The concept of a facilitated segment is still widely used as it best illustrates the neuromuscular dysfunction associated with abnormal joint biomechanics (Vernon 2010).

The facilitated segment is grounded on the principle that unfortified and ungainly movements cause segmental back muscles to shorten and stay hyperactive as a protective mechanism to guard the spinal segments. This approximation of the muscle fibres causes the central region of the muscle spindle to slacken which results in the inhibition of the muscle spindles. This stimulates the CNS to increase the gamma
motor neuron discharge to result in the sudden contraction of the intrafusal muscle fibres of the skeletal muscle. This is known as gamma gain; the alpha motor neuron activity causes the muscles to remain in a state of sustained contraction which, in turn, results in joint subluxation and dysfunction of the respective joint segment.

Muscles can produce and resist movement (Tortora and Derrickson 2014). Therefore, it is possible for muscle hypertonicity to result in joint restriction and dysfunction. For a motion segment to function optimally, there needs to be a balance between the agonists and the antagonists. Any muscle imbalance will perpetuate Korr’s (1975) model of a facilitated segment and, consequently, leave the segmental muscles in a state of involuntary hypertonicity (Korr 1975; Bergmann and Peterson 2011; Tortora and Derrickson 2014).

2.7 Spinal Mobilisation Therapy

Bergmann and Peterson (2011) describe spinal mobilisation as a form, non-thrust, manual therapy that entails rhythmic, gentle and passive movements of controlled force and amplitude on joints against a restrictive elastic barrier of the patient’s physiological ROM. A grade III Maitland mobilisation is more specifically described as a mobilisation of high amplitude and low velocity at the end of ROM and minimally engages the elastic barrier (Ward, Hebron and Petty 2017).

Joint mobilisations are graded into five different categories:

- Grade I – small amplitude movements are applied to the joint in the beginning ROM.
- Grade II – the application of large amplitude movements in the full ROM.
- Grade III – large amplitude movements are applied that reaches the end ROM.
- Grade IV – small amplitude movements at the end ROM.
- Grade V – high velocity, small amplitude thrust at the end ROM; joint manipulation.
Grades I and II joint mobilisations are clinically indicated to reduce pain and discomfort whereas grade III, and above, are clinically used to stretch the joint capsule and surrounding structures to increase segmental ROM (Hengeveld, Banks and Maitland 2014). Occasionally, joint mobilisation may be accompanied by a cavitation which is an audible pop or click (Kawchuk et al. 2015). However, this is not a unique feature of a joint mobilisation. Bergmann and Peterson (2011) states that any form of manual therapy that results in joint separation to overcome the fluid tension in the joint has the potential to produce a joint cavitation.

Joint mobilisation is a well-known manual therapy for restoring ROM and reducing pain in dysfunctional joints (Heiser, O'Brien and Schwartz 2013). It is widely used by different manual therapists, including chiropractors, osteopaths, and physiotherapists particularly in cases where joint manipulation therapy is contraindicated. This form of manual therapy aims to restore joint dysfunction by improving the ROM, breaking down adhesions, correcting the proprioception and dynamic joint stability and, subsequently, assisting in restoring neuromuscular control (Konin and Jessee 2012; Shih et al. 2018).

Thoracic spine mobilisation is often used to treat functional impairments remotely and in other spinal regions (Lee and Lee 2017). Although mobilisation is a well-known manual therapy tool, it is used less frequently than SMT due to the fact that the mechanisms responsible for eliciting the clinical benefits are not definitive (Clijsters, Fronzoni and Jenkins 2014; Cho, Lee and Lee 2017). Pecos-Martin et al. (2015) proposes that the clinical benefits are attributed to the effects that grade III mobilisations have on the nervous system – joint mobilisations stimulate the mechanoreceptors and proprioceptors to induce a spinal cord-mediated effect that restores the joint biomechanics and neuromuscular control. Enhancing the understanding of how these clinical benefits occur may contribute to the body of knowledge of spinal mobilisation therapy as a treatment modality, as evidence of its neurophysiological effects are scarce (Aguirrebeña, Newham and Critchley 2016).
Table 2.3 below summarises studies that have investigated the clinical effects of spinal mobilisation:

### Table 2.3 Spinal mobilisation therapy and its clinical effects

<table>
<thead>
<tr>
<th>Author</th>
<th>Sample Size</th>
<th>Study Design</th>
<th>Intervention</th>
<th>Outcome Measures</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lee and Lee (2017)</td>
<td>n = 33, with subacute stroke.</td>
<td>RCT</td>
<td>1. Segmental mobilisation T4 – T8.</td>
<td>Limits of stability (LOS), inspiratory function, and global rating of change (GRC).</td>
<td>Participants who received mid-thoracic spine mobilisation demonstrated effective short-term improvements in LOS and GRC.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2. Foam roller exercises.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cho, Lee and Lee (2017)</td>
<td>n = 32, with forward head posture (FHP).</td>
<td>RCT</td>
<td>1. Cervical mobilisation with stabilisation exercises.</td>
<td>Craniovertebral angle (CVA), cervical range of motion (CROM), national pain rating scale (NPRS), pressure pain threshold, neck disability index (NDI), and GRC.</td>
<td>The thoracic group demonstrated better overall short-term outcomes in CVA, cervical extension NPRS, NDI, and GRC.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2. Thoracic mobilisation with mobility exercises.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deepa, Dabholkar and Yardi</td>
<td>n = 30, with mechanical neck pain.</td>
<td>RCT</td>
<td>1. Maitland thoracic mobilisation with deep neck flexor (DNF) endurance exercises.</td>
<td>NPRS, NDI, and CROM.</td>
<td>DNF Endurance training with Maitland thoracic mobilisation group showed a significant improvement in CROM and NPRS only.</td>
</tr>
<tr>
<td>(2014)</td>
<td></td>
<td></td>
<td>2. DNF endurance exercises only.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
It is evident that studies support that thoracic mobilisation has a mechanical effect locally, and more specifically, a significant mechanical effect globally. The studies in Table 2.3 above demonstrate the clinical effectiveness of spinal mobilisation in terms of improving stability and joint dysfunction, restoring normal spinal postures, and reducing pain. Furthermore, there is a distinct trend that the clinical benefits of mobilisation are optimal when combined with other manual therapies such as mobility- or endurance exercises.

2.8 Spinal Mobilisation Therapy and Muscle Activity

Spinal manipulation therapy (SMT) is believed to inhibit the gamma gain phenomenon illustrated by Korr (1975) in order to relax the associated hypertonic muscles around the joint. Korr (1975) suggested that this was achieved by the SMT that forcefully stretches the muscle spindles at a constant velocity which, in turn, stretches the intrafusal muscle fibres and results in a bombardment of afferent impulses to the CNS in order to inhibit the gamma motor neuron discharge. In addition to this, the stretch of the joint capsule stretches the GTO which results in further gamma and alpha motor neuron inhibition in order to restore normal resting tone of surrounding muscles (Korr 1975; Krekoukias, Petty and Cheek 2007; Lehman 2012; Pecos-Martin et al. 2015).

Various studies have demonstrated this theory of Korr (1975) and, more recently, a handful of studies have endeavoured to apply this concept to spinal mobilisation as well. Table 2.4 below highlights examples of studies that have focused on the effects of spinal mobilisation on muscle activity:

<table>
<thead>
<tr>
<th>Author</th>
<th>Sample Size</th>
<th>Study Design</th>
<th>Intervention</th>
<th>Outcome Measures</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lascurain-Aguirrebenka et al. (2021)</td>
<td>n = 40, with non-specific neck pain.</td>
<td>RCT</td>
<td>1. Cervical mobilisation.</td>
<td>GRC and sEMG of SCM, scalene, upper trapezius, and erector spinae during neck flexion, extension, lateral</td>
<td>Cervical mobilisations caused increased neck sEMG, mostly due to increased</td>
</tr>
<tr>
<td>Study</td>
<td>Participants</td>
<td>Design</td>
<td>Interventions</td>
<td>sEMG Measurements</td>
<td>Outcomes</td>
</tr>
<tr>
<td>------------------------------</td>
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<td>--------------------------------------------------------------------------------</td>
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<td>-------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>
| Patterson, Dickerson and Ribeiro (2020) | n = 22       | RCT    | 1. Control.  
2. Grade IV inferior shoulder mobilisation. | sEMG of anterior, middle and posterior deltid, supraspinatus, infraspinatus, upper and lower trapezius, serratus anterior and latissimus dorsi. | Joint mobilisation reduced the muscle activity in the infraspinatus, middle and posterior deltid and the serratus anterior. |
2. Exercise Training.  
3. Exercise Training with joint mobilisation. | sEMG of the peroneus longus, tibialis anterior and soleus and reaching distance of the Y balance test, ROM, Cumberland ankle instability tool and NPRS. | Joint mobilisation improved self-reported ankle instability severity, dorsiflexion mobility and balance performance. Effects on muscle activity remain uncertain. |
| Kamel, Raoof and Tantawy (2016) | n = 45, females with chronic LBP at least 3 months post-partum | RCT    | 1. PA Lumbar Mobilisation with Traditional Treatment.  
2. Placebo with Traditional Treatment.  
3. Traditional Treatment only. | Pain intensity, functional disabilities, and sEMG of erector spinae (ES) muscles pre-test and post-test. | PA lumbar mobilisation resulted in a significant decrease in ES muscle activity as well as improved functional ability compared to other groups. |
2. Grade IV Central Lumbar Mobilisation at L4.  
3. Placebo. | Ultrasound images of the lumbar multifidus (LM) muscles and sEMG of ES muscles pre- and post-test. | The difference in LM contraction was small and may not have clinical significance. Lumbar mobilization did not change the activity of ES in healthy people. |
Pecos-Martín et al. (2015)  

n = 34, with non-specific thoracic pain.  

RCT  

1. Grade III central PA mobilisation at T7.  
2. Placebo (less than grade I mobilisation at T7).  

sEMG of the thoracic erector spinae muscles pre- and post-test, pain intensity, and pain pressure threshold.  

Grade III central mobilisation over the most symptomatic thoracic segment reduced thoracic erector spinae muscle activity.

Krekoukias, Petty and Cheek (2007)  

n = 36, asymptomatic.  

RCT  

1. Central PA mobilisation to L3 for 3 minutes.  
2. Placebo.  
3. Control.  

sEMG of the lumbar erector spinae muscles pre- and post-test.  

There was a decrease in erector spinae muscle activity of the mobilisation group compared to the placebo and control groups.

The studies above demonstrated that spinal mobilisation does have the ability to reduce the muscle activity – particularly the erector spinae muscles. There is a paucity in literature focusing on the effects of spinal mobilisation on the muscle activity in the trapezius muscle specifically. There has been a recent necessity for specifying the neurophysiological effects of spinal manual therapies due to the paradigm shift from the strict biomechanical model. Ultimately, the principle is that spinal mobilisation stimulates the mechanoreceptors and proprioceptors of a joint modifying the sensory impulses to the CNS which inhibits the firing of the motor neuron pools and alters spinal reflex pathways, in turn reducing pain and muscle hypertonicity (Lehman 2012).

Various recent studies have supported the paradigm shift away from the strict biomechanical model; however, few studies have focused on the neurophysiological affects associated with spinal mobilisation therapy alone (Pecos-Martín et al. 2015; Gliedt et al. 2017). Hegedus et al. (2011) conducted a systematic review to examine the neurophysiological effects of spinal mobilisation therapy. The results of the review illustrated that spinal mobilisation also elicits immediate neurophysiological effects however this study emphasised that spinal mobilisation is still understudied and calls for continued randomised controlled trials with spinal mobilisation as the primary intervention.
2.9 Summary of the Literature

Several studies demonstrate that individuals with thoracic spine dysfunction have altered joint biomechanics and muscle imbalances, particularly of the trapezius muscle, which results in pain locally and in other joints of the kinematic chain: namely, the shoulder, cervical spine and the lumbar spine. Therefore, sEMG is an appropriate measurement tool for measuring the relationship between joint dysfunction and muscle activity (Golabchi et al. 2019).

Many individuals who experience spinal pain seek chiropractic treatment and SMT is the primary treatment modality that chiropractors use (Bergmann and Peterson 2011; Clijsters, Fronzoni and Jenkins 2014). However, spinal mobilisation therapy is a tool that is also frequently used, particularly in cases where SMT is contraindicated (Lehman 2012). Spinal mobilisation is a mechanical intervention that is used to increase joint ROM, reduce pain and normalise muscle imbalances – particularly muscle hypertonicity (Krekoukias, Petty and Cheek 2007; Pecos-Martín et al. 2015). Studies have examined this intervention in terms of its measurable clinical effects (Hegedus et al. 2011). However, there is a paucity in literature regarding the neurophysiological effects of thoracic spine mobilisation. More so, there is a scarcity in studies focusing on the neurophysiological effects of thoracic mobilisation on the trapezius muscle specifically.

Understanding the immediate neurophysiological effects may contribute to the body of knowledge of an evidence-based approach to consider the appropriate technique and application of spinal mobilisation. In doing so, the benefits of spinal mobilisation, in terms of its effects on muscle activity, will be better understood. Thus, this study aims to determine the immediate effect of thoracic spine grade III mobilisation on the muscle activity of the trapezius muscle in asymptomatic individuals.
CHAPTER 3: METHODOLOGY

3.1 Introduction

This chapter explains the design of the study, the sample population, how participants will be recruited as well as the inclusion and exclusion criteria for the participants. Group allocation and randomisation will also be discussed. In addition to this, the chapter also summarises the research procedure and protocol along with the measurement tool that is used in this study. Interventions, statistical data analysis and ethical considerations will also be outlined.

3.2 Study Design

This study was quantitative and utilised an experimental, pre-test and post-test design. This design was chosen as it is the most appropriate way to evaluate and compare the effects of the clinical intervention with a control group in addition to being the most efficient way to observe and quantify any changes caused by the experimental intervention (Reed Johnson et al. 2013).

3.3 Study Location

The study was conducted at the Chiropractic Day Clinic (CDC) at the Durban University of Technology (DUT) in South Africa. The CDC is a teaching clinic that offers chiropractic treatment to the public by students completing their Master’s degree in Technology in Chiropractic. Thereby, the study was conducted under the supervision and guidance of the research supervisor and qualified chiropractors who serve as clinical instructors.

Gatekeeper permission was obtained from the DUT Research Director (Appendix A) to utilise the DUT campus to conduct the research. In addition to this, gatekeeper permission was obtained from the DUT Clinic Director (Appendix B) to utilise the clinic for research purposes.
Furthermore, the study did not commence until the DUT Institutional Research Ethics Committee (IREC) granted full ethical approval to perform the study (Appendix C). The clinical trial was registered on the PAN Africa clinical trials registry (Appendix D).

3.4 Recruitment and Sampling Strategies

3.4.1 Participant Recruitment

Individuals were primarily recruited by means of word-of-mouth. Advertisements (Appendix E) were also placed on Ritson, Steve Biko and ML Sultan campuses of DUT and at local gyms and supermarkets. Permission obtained to place these advertisements (Appendix F) was obtained first. All the participants showed interest telephonically via WhatsApp. Participants were contacted, telephonically, by the researcher at their soonest convenience to perform a verbal pre-screening consultation in order to determine whether they were eligible for the study.

Once the pre-screening consultation was completed and the participant revealed to be eligible for the study, an appointment was made for the researcher to consult the individual at the CDC of DUT.

<table>
<thead>
<tr>
<th>Questions to ask potential participants</th>
<th>Expected Answers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Please may I ask you some questions?</td>
<td>Yes.</td>
</tr>
<tr>
<td>Are you between the ages 18 – 45?</td>
<td>Yes.</td>
</tr>
<tr>
<td>Have you experienced middle back pain in the last six weeks?</td>
<td>No.</td>
</tr>
<tr>
<td>Have you been receiving any manual therapy in the last month? This includes physiotherapy, chiropractic treatment or any other treatment that involves soft tissue manipulation.</td>
<td>No.</td>
</tr>
<tr>
<td>Are you currently using any anti-inflammatories, muscle relaxants, heart medication or other painkillers?</td>
<td>No.</td>
</tr>
<tr>
<td>Have you recently had any major injury or trauma to your spine?</td>
<td>No.</td>
</tr>
</tbody>
</table>
3.4.2 Sampling Strategy

3.4.2.1 Sample Size

The study sample size (n) was determined using power analysis calculations and 48 participants allowed for a power analysis of 81% (Esterhuizen, 2021). Of these participants, 24 were a part of the intervention group and the remaining 24 were in the control group.

3.4.2.2 Sample Allocation

Once participants met the inclusion criteria and were accepted into the study, they were randomly allocated into either Group A or Group B. Allocation took place by means of a randomisation table which was constructed by the statistician to avoid participant bias (Esterhuizen 2021). Participants arrived at the CDC and reported to the receptionist on duty who had the randomisation table with her. She was required to use the table by finding the number that corresponded to what number the participant was (e.g. participant number 24) and allocated the participant in to the respective group and informed the researcher accordingly.

A randomisation table was used in order to prevent selection bias and accidental bias. More so, randomisation adds value to scientific findings by producing comparable groups and further preventing bias when interventions are chosen for each participant. In doing so, the probability theory can be applied to articulate the possibility of chance as a source for the differences in different outcomes (Suresh 2011).

3.5 Population of the Study

Participants of this study could be male or female. However, all participants had to be asymptomatic, between the ages of 18-45 and meet the inclusion and exclusion criteria.
3.5.1 Inclusion Criteria

- The participants had to present with fixations in the region of the thoracic spine (Nyberg and Russell Smith 2013). Fixations are joints that have a decreased ROM and the presence of joint fixations were determined by means of active and passive motion palpation.
- The participants had to be between the ages of 18 – 45 years in order to eliminate the requisite for consent from a parent or legal guardian. In addition to this, individuals who are 45 years old and above have an increased likelihood of presenting with pathologies such as degeneration and, consequently, may alter the outcomes of the study (Walker et al. 2014).

3.5.2 Exclusion Criteria

- Participants who were experiencing any pain in the region of the thoracic spine (Minarini, Ford and Esteves 2018).
- Individuals who have taken any drugs that alter skeletal muscles four weeks prior to the consult were excluded because it may skew results recorded by the sEMG instruments. For example, Norflex (Kruk and Pycock 1991).
- Participants who received manual therapy (i.e. manipulation, mobilisation or any soft tissue treatment) on the thoracic spine four weeks prior to the consult were excluded. This was done to avoid the effects of previous interventions from altering results recorded during the study.
- Individuals who had diagnosed clinical contraindications to spinal joint mobilisation, identified during a case history and/or physical exam, were excluded from the study and referred to an appropriate medical discipline to assist with the condition. These contraindications included acute inflammation, Pott’s Disease, malignancy, hypermobility, neurological deficits, fractures, osteoporosis, rheumatoid arthritis during the acute phase, Paget’s Disease, Ankylosing Spondylitis during the inflammatory phase, Sheuermann’s Disease during phase 1 (Duarte et al. 2014), vertebral artery insufficiency, scoliosis, history of surgery and trauma and spinal cord insufficiency (Konin and Jessee 2012).
• Individuals with contraindications to sEMG at the region of electrode placement were excluded as well. These contraindications included skin rashes, open wounds or any skin irritations (Gallina et al. 2017).

• Individuals with a skinfold thickness that was higher than 19mm were excluded. The presence of adipose tissue would have altered the ability to take accurate muscle activity readings (Baniqued et al. 2016).

• When participants cavitated while receiving the spinal mobilisation, they were not excluded from the study. Bergmann and Peterson (2011) established that any form of manual therapy that causes joint separation to overcome the fluid tension in the synovial fluid has the potential to produce a joint cavitation. Hence, manipulation and mobilisation are now distinguished based on the velocity of the application and not the presence or absence of a joint cavitation – joint cavitation did not skew the data.

3.6 Measurement Tools

3.6.1 Surface Electromyography

Based on previous studies and literature, sEMG was the most appropriate tool to objectively measure the changes in muscle activity and, indirectly, joint dysfunction – the validity and reliability of sEMG was established in chapter two (Crisswell 2010; Cavalcanti Garcia and Vieira 2011; Capobianco et al. 2018). Hence, sEMG was used to measure the electrical activity of muscles maximum voluntary isometric contraction (MVIC) for ten seconds, and relaxation (for three minutes). It is an effective tool in quantifying the effects of an intervention on the neuromuscular activity of respective muscles. The electrodes are small pads that were placed on the surface of the participants skin and were connected to the EMG machine. These electrodes were responsible for detecting the electrical activity of the muscle below them. The researcher did the electrode placement on every participant to ensure consistency and avoid skewing of data.

According to De Luca et al. (2010), cross talk between adjacent muscles can be minimised by using a small surface area of muscles and small electrodes. In addition
to this, correct electrode placement is crucial in the process of recording accurate EMG readings (Crisswell 2010). De Luca et al. (2010) further states that the electrode needs to be placed between a motor point and a muscle’s tendon (but not exactly on top of the tendon as this decreases the amplitude of the EMG and increases the risk of cross talk). The electrodes also need to be parallel to the muscle fibres, along the longitudinal midline. Hence, great care and precision was taken when the electrodes were placed onto the participant. Furthermore, a root mean square (RMS) was used to normalise the data (Halaki and Ginn 2012). Surface electromyography (sEMG) is also very sensitive to sound (Chowdhury et al. 2013). This was managed by keeping the door shut at all times during the procedure and ensuring that there was silence during the maximum voluntary contractions.

As seen in Figure 3.1 – the electrodes, for the middle trapezius fibres, were placed (2cm apart horizontally) at a point midway between a line that joined the medial border of the scapula and thoracic vertebra T3 on each side (Crisswell 2010).

![Figure 3.1 Electrode placement for middle trapezius muscles (Crisswell 2010)](image)

As seen in Figure 3.2 - the electrodes, for the lower trapezius fibres, were placed (2cm apart) at 55-degree oblique angle at a point midway between the spine and the medial border of the scapula, 5cm below the spine of the scapula (Crisswell 2010).
3.7 Interventions

3.7.1 Intervention Group

The intervention group received a Grade III Maitland mobilisation; this is a mobilisation of high amplitude, within the restricted ROM and only slightly engages the elastic barrier (Ward, Hebron and Petty 2017). The mobilisation was applied by the researcher only and nobody else. This was to ensure that every participant received a mobilisation of the same velocity and amplitude each time and avoid the data from being skewed. The mobilisation was performed at vertebral levels T2 – T5 because this is where the upper and lower trapezius fibres mainly attach. This was done for the purpose of standardisation. Studies have revealed that clinical effects of spinal mobilisation are systemic; whether they are performed at dysfunctional segments or not (Pecos-Martín et al. 2015). The mobilisation was performed in the following way:

The patient was placed in the prone-lying position with the head piece slightly lowered and the pelvis firm on the chiropractic bed. The pads of the doctor’s thumbs were then placed on the spinous process, pointing transversely across the vertebral column. The remaining fingers spread out over the posterior chest wall and the mobilisation was finally performed in a posterior to anterior direction. The purpose of this form of contact was to achieve the best control of the mobilisation movement and to attain minimal discomfort. It is important to note that the pressure passing through the thumbs were not as a result of the intrinsic muscles of the hand, but instead from the doctor’s body weight. This was variable dependant on the degree of the fixation and patient tolerance (Cho, Lee and Lee 2017). All fixated levels were mobilised.
The mobilisation be described in the following way:
Doctor position: Fence stance position
Patient position: Prone
Contact point on patient: Spinous processes of thoracic vertebrae
Contact point on doctor: Thenar eminence
Vector of the movement: Posterior to anterior

The mobilisation subsequently stimulated receptors of the skin, respective joints and muscles that attached in this spinal region (Hegedus et al. 2011).

3.7.2 Control Group

The control group received a control procedure. The patient was placed in the prone-lying position for three minutes and remained motionless. This control was chosen because it replicates the procedure of the spinal mobilisation – the patient gets into the position for treatment and stays in this position for the duration of the treatment. This allows for the effects of time and repeated testing to be accounted for (Pithon 2013).

3.8 Study Procedure

Once the participant responded to an advert or approached the researcher from word of mouth, the researcher verbally explained the research and the participant had to answer pre-screening questions to ensure that they were an eligible participant for the study. After the patient had met all the inclusion criteria of the pre-screening consultation, they were given an appointment at the CDC of DUT for a consultation with the researcher. Prior to commencement of respective procedures, the patient was required to read and sign the letter of information and consent (Appendix G). In addition to this, a verbal explanation of the research was provided whereby a verbal consent was necessitated from the participant. At this point, the participant also had an opportunity to ask any questions concerning the research which was answered by the researcher. The patient was then screened by the researcher to confirm that they meet the inclusion criteria. A case history (Appendix H), physical examination
(Appendix I) and thoracic spine regional examination (Appendix J) was performed. The participant was allowed to take part in the study if there were joint fixations present, but they could not be symptomatic to pain in the thoracic region of the spine and no positive orthopaedic tests could be present (Kim et al. 2019).

Participants who did not meet the inclusion criteria of the study were excluded and provided with an option to make an appointment for chiropractic treatment (at their own expense) separate to the study or referred to an appropriate medical discipline. Once all physical findings were recorded, the participant was allocated to a group using the randomisation table (Esterhuizen, 2021). Following this, the skin area where the surface electrodes were to be placed were sterilised with alcohol and hairy skin was shaved with a disposable razor (Crisswell 2010). The electrodes were then placed on the trapezius muscles, based on the positioning mentioned above.

The researcher then demonstrated the MVIC of each muscle (as explained below) after which the patient was instructed to perform these contractions in order to ensure that the participant was performing them correctly as contractions had to be as identical as possible with each sEMG reading (Halaki and Ginn 2012). After perfecting the contraction movement, the patient was left to rest in the prone-lying position for two minutes before the experimental study continued.

Halaki and Ginn (2012) state that there is not enough confirmation as to which maximum voluntary isometric contractions are most effective. Therefore, the movements were chosen are based on what is most practical and what isolated each muscle the most.

Smith et al. (2004) proposed that the following isometric contractions are most effective for obtaining accurate results of the middle trapezius muscles activity: the patient was instructed to lie prone with their arm abducted by 90 degrees and externally rotated by 90 degrees (i.e. the thumb is facing upwards). This was done
while the patients arms were resisting against a strap that was attached to the bed in order to achieve a maximum contraction.

Arlotta, LoVasco and McLean (2010) established that the best way to measure the lower trapezius muscles activity was by having the patient in the prone-lying position with their arms resting on their sides (thumbs facing the ground). From this position the patient then had to lift their chest (approximately 10cm off the bed) and pronate their hands (so their thumbs are now facing the ceiling). It was important to emphasise to the patient that they had to retract the scapulae as if their finger-tips were trying to reach their feet and to maintain this position for the entire period.

Each muscle contraction was performed for 10 seconds (as a pre-test), three times with a 30 second rest period between each repetition to avoid muscle fatigue. These three sEMG readings were then recorded in order to calculate a mean value – this ensured greater accuracy and accounted for errors that may have taken place that the researcher was unaware of (Chowdhury et al. 2013). Following this, the patient had to rest for 3 minutes and then the intervention or control was performed on the individual. Following this, each muscle contraction was required to be performed again as a post-test (immediately after the intervention) by repeating the same procedure during the pre-test. The three post-test EMG readings were also recorded in order to calculate a mean value.

The electrodes were only removed once all readings had been recorded after which the patient’s skin was cleaned with alcohol again. Crisswell (2010) emphasises that the electrodes need to stay on the patient’s skin the whole time during the experimental procedure in order to avoid any minimal changes in electrode placement which may alter the results. The patient then had few minutes to rest, if required, and were thanked before they left.
3.9 Data Reduction and Analysis

At each consultation, the sEMG readings for muscle activity were measured during three sets of MVCs that occurred prior to the intervention and after the intervention. This raw data was processed using RMS that converted the data into a waveform making it suitable for analysis. The peak value from each set of the three contractions was recorded. The mean muscle activity was also recorded from each contraction; this value was later divided by three in order to calculate the mean muscle activity for each set of MVC’s.

According to Halaki and Ginn (2012), the data had to be normalised. Normalisation requires a conversion of the processed data into a scale relative to a known and repeatable value. This allowed the researcher to be able to compare the EMG activity between different individuals – irrespective of age and gender. For this study, the normalised values were displayed as a percentage of the peak muscle activity value MVC.

\[(\text{pre-mean MA ÷ pre-peak amplitude}) \times 100 = \%\]

\[(\text{post-mean MA ÷ pre-peak amplitude}) \times 100 = \%\]

\[(10\text{-minute post-mean MA ÷ pre-peak amplitude}) \times 100 = \%\]

Within-group comparison of the effect over time were done using paired t-tests while the within and between group comparisons were done using repeated measures ANOVA testing. A significant time by group interaction effect indicated a significant treatment effect. Profile plots were generated to graphically display the direction and trend of the intervention. A probability value (\(p\)) below 0.05 level was considered to be statistically significant, and IBM SPSS version 26 software was used to evaluate the data (Esterhuizen, 2021). This study procedure required a once-off consult with the participants, therefore, no dropouts were accounted for as there weren’t any dropouts.
3.10 Ethical Considerations

3.10.1 Autonomy
This component was maintained by warranting that the participants had to sign the letter of information and informed consent which was available in English and isiZulu (Appendix G) and were required to provide a verbal informed consent. No coercion was used to get participants to partake in the study. Participant confidentiality was ensured by assigning each participant with a specific code as opposed to using their actual names in the study. Due to a control procedure being present in Group B, all participants were informed of the possibility that they could potentially be a part of the control group. However, the inclusion criteria stated that the patients had to be asymptomatic implying that, in essence, they were withheld from any treatment.

3.10.2 Justice
The pillar of justice was obeyed because participant recruitment did not take race, religion, gender or nationality into consideration. More so, although selected participants were asymptomatic, should any illness or pathology have presented during the consultation, the participant was excluded from the study and referred to the appropriate medical practitioner. The participant was also offered to make an appointment to return to the CDC for chiropractic treatment – if required. The participant had the opportunity to withdraw from the study at any point without any detrimental consequences to them.

Thoracic spine mobilisation and maximum voluntary contraction of the trapezius muscles are not associated with any unfavourable consequences. The use of sEMG is not associated with any risks. The entire experiment was supervised by the researcher as well as the clinician on duty supervising the researcher at all times, this ensured that optimal patient care remained the main priority.

3.10.3 Non-maleficence and Beneficence
The patient’s health and safety were protected as the interventions and measurement tools are safe and registered with their appropriate disciplines. More so, the study was conducted in the CDC under supervision and indemnity cover of the clinic director, the
clinic and the university. Patient confidentiality was maintained by keeping the patient’s clinical file, containing signed informed consent letters under a code – hence preventing the participant’s details from appearing in any research publications. However, their patient files were stored in the CDC should they ever have returned for additional treatment. Their data from the research will be shredded after five years. Electronic data was password-secured on a USB and kept at the CDC. After five years this data will be deleted as well.

3.11 Conclusion
This chapter has illustrated the research protocol that was used in the study. This also included how the data was obtained and analysed in order to comprehend the findings of the study which will appear in the following chapter.
CHAPTER 4: RESULTS

4.1 Introduction

This chapter depicts the results of the study in the form of graphs and tables. Data that was obtained from participants was analysed as described in Chapter Three. The analysis included:

- Demographic data analysis comprising of age and gender which was analysed using independent samples T-tests and Chi Square Tests.
- Background variables comprising of weight, height and skinfold thickness – these were analysed with samples T-tests.
- Within-group compared pre and post intervention using paired T-Tests.
- Within and between-group comparisons were done using repeated measures ANOVA testing – a significant time by group interaction effect indicated a significant treatment effect.
- Profile plots were generated to graphically display the direction and trend of the intervention.

This research study was based on the following hypotheses:

- Null Hypothesis: There will be no difference in muscle activity between the intervention and control groups in terms of the surface electromyographic readings pre and post intervention.
- Alternate Hypothesis: There will be a statistically significant difference between the intervention and control groups in terms of the surface electromyographic readings pre and post intervention.

4.2 Consort Flow Diagram

The flow diagram below denotes the distribution of the participants throughout the research. After appropriately establishing that participants were asymptomatic, 24 participants were randomly allocated to the Intervention Group and 24 participants
were randomly allocated to the Control Group. Group allocation was done with the use of a randomisation table.

![Figure 4.1 Consort flow diagram](image)

**4.3 Demographic Characteristics and Background Variables of Participants**

**4.3.1 Age**

The sample size consisted of 48 participants who were between the ages of 18 to 45 years of age. All participants met the inclusion criteria and were randomly allocated into either the intervention or control Groups. Table 4.1 below presents the mean age of the sample within the two groups.

**Table 4.1 Mean age per group**

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intervention</td>
<td>24</td>
<td>24</td>
<td>3</td>
<td>0.492</td>
</tr>
<tr>
<td>Control</td>
<td>24</td>
<td>25</td>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>
In the intervention group the average age was 24 years compared to the control group which had an average age of 25 years. However, there was no statistical difference in age between the intervention and control groups ($p = 0.492$). This indicated that the two groups were randomised completely.

### 4.3.2 Gender Distribution

This study included both females and males. Table 4.2 below presents the gender distribution across the experimental groups.

**Table 4.2 Gender distribution**

<table>
<thead>
<tr>
<th>Gender</th>
<th>Intervention</th>
<th>Control</th>
<th>Total</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Count</td>
<td>Column (%)</td>
<td>Count</td>
<td>Column (%)</td>
</tr>
<tr>
<td>Male</td>
<td>12</td>
<td>50.0%</td>
<td>13</td>
<td>54.2%</td>
</tr>
<tr>
<td>Female</td>
<td>12</td>
<td>50.0%</td>
<td>11</td>
<td>45.8%</td>
</tr>
</tbody>
</table>

Twenty-four participants were randomised equally into intervention and control groups. There was a similar proportion of each gender within the intervention and control groups.

### 4.3.3 Background Variables

Height, weight and skinfold thickness were recorded, this is summarised in Table 4.3 below.

**Table 4.3 Background variables**

<table>
<thead>
<tr>
<th></th>
<th>Intervention</th>
<th>Control</th>
<th>Total</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Intervention (n = 24)</td>
<td>Control (n = 24)</td>
<td>Total (n = 48)</td>
<td></td>
</tr>
<tr>
<td>Height</td>
<td>Mean</td>
<td>169</td>
<td>172</td>
<td>170</td>
</tr>
<tr>
<td></td>
<td>Standard Deviation</td>
<td>8</td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td>Weight</td>
<td>Mean</td>
<td>70.5</td>
<td>69.0</td>
<td>69.7</td>
</tr>
<tr>
<td></td>
<td>Standard Deviation</td>
<td>13.5</td>
<td>13.8</td>
<td>13.6</td>
</tr>
<tr>
<td>Skinfold</td>
<td>Mean</td>
<td>9.4</td>
<td>7.8</td>
<td>8.6</td>
</tr>
<tr>
<td></td>
<td>Standard Deviation</td>
<td>3.4</td>
<td>3.9</td>
<td>3.7</td>
</tr>
</tbody>
</table>
Table 4.3 demonstrates that most participants had a skinfold thickness of 8.6mm which met the exclusion criteria requirements. More so, there was a minimal difference in mean skinfold between intervention-and control groups – with the average skinfold being slightly higher in the intervention group, but it was not statistically significant ($p = 0.134$). The average height of the participants was 170cm – participants in the control group were slightly taller with an average height of 172cm, however, once again, the difference was not statistically significant ($p = 0.179$). The average weight of participants was 69.7kg and participants in the intervention group had a slightly greater mean weight of 70.5kg and this was also not statistically significant ($p = 0.700$).

Tables 4.1, 4.2 and 4.3 highlight that none of the demographic or background variables differed significantly between the two groups, hence, the randomisation process was completed adequately and results could be compared accurately.

### 4.4 Muscle Activity

The EMG baseline readings were captured using the mean muscle activity values as well as the peak readings for each muscle contraction. In addition to this, values were normalised, and peak amplitudes were also calculated in order to ensure that data was comparable, irrespective of age, gender, height, weight or skinfold. Table 4.4 summarises the EMG baseline readings for the left middle trapezius (LMT), right middle trapezius (RMT), left lower trapezius (LLT) and right lower trapezius (RLT), pre-test.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Reading</th>
<th>Statistical Measure</th>
<th>Intervention</th>
<th>Control</th>
<th>Total</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>LMT</td>
<td>Mean muscle activity</td>
<td>Mean</td>
<td>.36864</td>
<td>.42336</td>
<td>.39600</td>
<td>0.483</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Standard Deviation</td>
<td>.24689</td>
<td>.28801</td>
<td>.26681</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Peak muscle activity</td>
<td>Mean</td>
<td>1.052765</td>
<td>1.208399</td>
<td>1.130582</td>
<td>0.445</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Standard Deviation</td>
<td>.634458</td>
<td>.760370</td>
<td>.697210</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Peak amplitude (%)</td>
<td>Mean</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Standard Deviation</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Normalisation (%)</td>
<td>Mean</td>
<td>35.30744</td>
<td>35.29987</td>
<td>35.30366</td>
<td>0.997</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Standard Deviation</td>
<td>7.36585</td>
<td>5.32846</td>
<td>6.35963</td>
<td></td>
</tr>
</tbody>
</table>

Table 4.4 Baseline surface electromyography readings and data pre-test
<table>
<thead>
<tr>
<th></th>
<th>Mean muscle activity</th>
<th>Peak muscle activity</th>
<th>Peak amplitude (%)</th>
<th>Normalisation (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMT</td>
<td>Mean</td>
<td>Standard Deviation</td>
<td>Mean</td>
<td>Standard Deviation</td>
</tr>
<tr>
<td></td>
<td>.29709</td>
<td>.19524</td>
<td>.88109</td>
<td>.51719</td>
</tr>
<tr>
<td></td>
<td>.38745</td>
<td>.27726</td>
<td>1.10129</td>
<td>.69794</td>
</tr>
<tr>
<td></td>
<td>.34227</td>
<td>.24157</td>
<td>.99119</td>
<td>.61778</td>
</tr>
<tr>
<td></td>
<td>0.198</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>.19524</td>
<td>.27726</td>
<td>.24157</td>
</tr>
<tr>
<td></td>
<td></td>
<td>.51719</td>
<td>1.10129</td>
<td>.69794</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.198</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.221</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.19524</td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>0.27726</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.24157</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LLT</td>
<td>Mean</td>
<td>Standard Deviation</td>
<td>Mean</td>
<td>Standard Deviation</td>
</tr>
<tr>
<td></td>
<td>.10622</td>
<td>.06523</td>
<td>.45687</td>
<td>.24880</td>
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<tr>
<td></td>
<td>.13512</td>
<td>.11140</td>
<td>.61038</td>
<td>.50047</td>
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<tr>
<td></td>
<td>.12067</td>
<td>.09148</td>
<td>.53362</td>
<td>.39860</td>
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<td>0.278</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>.06523</td>
<td></td>
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</tr>
<tr>
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<td></td>
<td>.11140</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>.09148</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RLT</td>
<td>Mean</td>
<td>Standard Deviation</td>
<td>Mean</td>
<td>Standard Deviation</td>
</tr>
<tr>
<td></td>
<td>.09266</td>
<td>.06147</td>
<td>.35608</td>
<td>.24835</td>
</tr>
<tr>
<td></td>
<td>.11388</td>
<td>.09415</td>
<td>.47637</td>
<td>.41053</td>
</tr>
<tr>
<td></td>
<td>.10327</td>
<td>.07939</td>
<td>.41622</td>
<td>.34111</td>
</tr>
<tr>
<td></td>
<td>0.360</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>.06147</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>.09415</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>.07939</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>0.226</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.923</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

When considering the baseline readings in Table 4.4, only the RMT pre-normalisation value differed significantly between the groups ($p = 0.036$) with the higher values being found in the control group.

### 4.4.1 Objective One

To determine the immediate effects of thoracic spine grade III mobilisation on the muscle activity of the middle and lower trapezius muscles.

#### 4.4.1.1 The Immediate Effects of Grade III Spinal Mobilisation on Muscle Activity

Table 4.5 summarises the effects of thoracic grade III mobilisation on the immediate post readings in the intervention group.
<table>
<thead>
<tr>
<th>Pair</th>
<th>Muscle</th>
<th>Reading</th>
<th>Mean</th>
<th>N</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>LMT</td>
<td>Average (pre-test)</td>
<td>0.3686379</td>
<td>24</td>
<td>0.24689248</td>
<td>0.05039672</td>
<td>0.020</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Average (post-test)</td>
<td>0.3248963</td>
<td>24</td>
<td>0.23812387</td>
<td>0.04860683</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>RMT</td>
<td>Average (pre-test)</td>
<td>0.2970925</td>
<td>24</td>
<td>0.19523609</td>
<td>0.03985240</td>
<td>0.655</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Average (post-test)</td>
<td>0.2889004</td>
<td>24</td>
<td>0.19669569</td>
<td>0.04015034</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>LLT</td>
<td>Average (pre-test)</td>
<td>0.1062204</td>
<td>24</td>
<td>0.06523134</td>
<td>0.01331529</td>
<td>0.230</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Average (post-test)</td>
<td>0.1287954</td>
<td>24</td>
<td>0.09717441</td>
<td>0.01983564</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>RLT</td>
<td>Average (pre-test)</td>
<td>0.0926583</td>
<td>24</td>
<td>0.06147159</td>
<td>0.01254784</td>
<td>0.582</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Average (post-test)</td>
<td>0.0971663</td>
<td>24</td>
<td>0.06313872</td>
<td>0.01288814</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>LMT</td>
<td>Peak (pre-test)</td>
<td>1.05276492</td>
<td>24</td>
<td>0.634458063</td>
<td>0.129508210</td>
<td>0.928</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peak (post-test)</td>
<td>1.0465467</td>
<td>24</td>
<td>0.80003658</td>
<td>0.16330678</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>RMT</td>
<td>Peak (pre-test)</td>
<td>0.8810946</td>
<td>24</td>
<td>0.51718995</td>
<td>0.10557096</td>
<td>0.709</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peak (post-test)</td>
<td>0.8642317</td>
<td>24</td>
<td>0.46503556</td>
<td>0.09492499</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>LLT</td>
<td>Peak (pre-test)</td>
<td>0.4568704</td>
<td>24</td>
<td>0.24880226</td>
<td>0.05078655</td>
<td>0.189</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peak (post-test)</td>
<td>0.5443337</td>
<td>24</td>
<td>0.27397656</td>
<td>0.05592523</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>RLT</td>
<td>Peak (pre-test)</td>
<td>0.3560754</td>
<td>24</td>
<td>0.24835362</td>
<td>0.05069497</td>
<td>0.299</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peak (post-test)</td>
<td>0.3929467</td>
<td>24</td>
<td>0.25897628</td>
<td>0.05286331</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>LMT</td>
<td>Peak Amplitude (pre-test)</td>
<td>100.00</td>
<td>24</td>
<td>.000</td>
<td>.000</td>
<td>0.861</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peak Amplitude (post-test)</td>
<td>101.0676992</td>
<td>24</td>
<td>29.47357865</td>
<td>6.01626905</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>RMT</td>
<td>Peak Amplitude (pre-test)</td>
<td>100.00</td>
<td>24</td>
<td>.000</td>
<td>.000</td>
<td>0.699</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peak Amplitude (post-test)</td>
<td>102.1723979</td>
<td>24</td>
<td>27.20414011</td>
<td>5.55302185</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>LLT</td>
<td>Peak Amplitude (pre-test)</td>
<td>100.00</td>
<td>24</td>
<td>.000</td>
<td>.000</td>
<td>0.024</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peak Amplitude (post-test)</td>
<td>155.1348050</td>
<td>24</td>
<td>111.44032582</td>
<td>22.74766125</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>RLT</td>
<td>Peak Amplitude (pre-test)</td>
<td>100.00</td>
<td>24</td>
<td>.000</td>
<td>.000</td>
<td>0.179</td>
</tr>
</tbody>
</table>

Table 4.5 Paired sample statistics for immediate effect in intervention group
Based on Table 4.5 above, it is noted that there was a significant decrease in average mean muscle activity in the LMT over time ($p = 0.020$). Although a significant decrease was noted, it was not statistically significant.

4.4.2 Objective Two

To determine the immediate effects of a control on the muscle activity of the middle and lower trapezius muscles.

4.4.2.1 Immediate Effects of a Control on Muscle Activity

Table 4.6 below summarises the immediate effects of a control on muscle activity. The control group did not receive any form of intervention or placebo – they lay prone for 3 minutes.

Table 4.6 Paired sample statistics for immediate effect in control group

<table>
<thead>
<tr>
<th>Pair</th>
<th>Muscle</th>
<th>Reading</th>
<th>Mean</th>
<th>N</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>LMT</td>
<td>Average (pre-test)</td>
<td>.4233629</td>
<td>24</td>
<td>.28801398</td>
<td>.05879061</td>
<td>0.142</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Average (post-test)</td>
<td>.4028179</td>
<td>24</td>
<td>.27072023</td>
<td>.05526054</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Average (pre-test)</td>
<td>Average (post-test)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>-------------------</td>
<td>-------------------</td>
<td>---</td>
<td>---</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>RMT</td>
<td>.3874471</td>
<td>.3551817</td>
<td>24</td>
<td>.27726097</td>
<td>.25732424</td>
<td>.05659566</td>
</tr>
<tr>
<td>3</td>
<td>LLT</td>
<td>.1351246</td>
<td>.1461692</td>
<td>24</td>
<td>.11140370</td>
<td>.11174887</td>
<td>.02274019</td>
</tr>
<tr>
<td>4</td>
<td>RLT</td>
<td>.1138846</td>
<td>.1347592</td>
<td>24</td>
<td>.09415199</td>
<td>.11009704</td>
<td>.01921870</td>
</tr>
<tr>
<td>5</td>
<td>LMT</td>
<td>1.20839875</td>
<td>1.1197367</td>
<td>24</td>
<td>.760369986</td>
<td>.6813535</td>
<td>.155209873</td>
</tr>
<tr>
<td>6</td>
<td>RMT</td>
<td>1.1012921</td>
<td>1.9763546</td>
<td>24</td>
<td>.69794025</td>
<td>.61081586</td>
<td>.14246646</td>
</tr>
<tr>
<td>7</td>
<td>LLT</td>
<td>.6103750</td>
<td>.5875442</td>
<td>24</td>
<td>.50047466</td>
<td>.44831779</td>
<td>.10215896</td>
</tr>
<tr>
<td>8</td>
<td>RLT</td>
<td>.4763733</td>
<td>.4328054</td>
<td>24</td>
<td>.41053023</td>
<td>.32325900</td>
<td>.08379913</td>
</tr>
<tr>
<td>9</td>
<td>LMT</td>
<td>100.00</td>
<td>94.9551930</td>
<td>23</td>
<td>.000</td>
<td>19.25183614</td>
<td>4.01428533</td>
</tr>
<tr>
<td>10</td>
<td>RMT</td>
<td>100.00</td>
<td>93.6458908</td>
<td>24</td>
<td>.000</td>
<td>29.56558001</td>
<td>6.03504875</td>
</tr>
<tr>
<td>11</td>
<td>LLT</td>
<td>114.1946813</td>
<td>108.0604613</td>
<td>24</td>
<td>66.62028907</td>
<td>59.77480305</td>
<td>12.20148058</td>
</tr>
<tr>
<td>12</td>
<td>RLT</td>
<td>35.2998725</td>
<td>36.0681100</td>
<td>24</td>
<td>5.32846445</td>
<td>6.22626053</td>
<td>1.08766825</td>
</tr>
<tr>
<td>13</td>
<td>LMT</td>
<td>37.3448350</td>
<td>35.7282979</td>
<td>24</td>
<td>7.55563224</td>
<td>6.12439637</td>
<td>1.54228697</td>
</tr>
</tbody>
</table>
### 4.4.3 Objective Three

To compare and correlate the electromyographic data between the intervention and control groups.

#### 4.4.3.1 Mean Muscle Activity

Repeated ANOVA tests were utilised to compare the pre and post outcomes in both intervention and control groups. The effects of spinal mobilisation on the relevant muscles; LMT, RMT, LLT, and RLT in each experimental group have been described in relation to the main objectives of this study. Table 4.7 below summarises the effects of thoracic spine grade III mobilisation on the immediate post readings in the intervention group.

**Table 4.7 ANOVA for immediate effect on mean muscle activity by time and treatment group**

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Statistical Test</th>
<th>Effect</th>
<th>Value</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>LMT</td>
<td>Wilks’ lambda</td>
<td>Time</td>
<td>0.155</td>
<td>0.006</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time*group</td>
<td>0.023</td>
<td>0.299</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Group</td>
<td>0.788</td>
<td>0.379</td>
</tr>
<tr>
<td>RMT</td>
<td>Wilks’ lambda</td>
<td>Time</td>
<td>0.941</td>
<td>0.097</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time*group</td>
<td>0.978</td>
<td>0.319</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Group</td>
<td>1.382</td>
<td>0.246</td>
</tr>
<tr>
<td>LLT</td>
<td>Wilks’ lambda</td>
<td>Time</td>
<td>0.951</td>
<td>0.131</td>
</tr>
</tbody>
</table>

On the analysis of Table 4.6, the mean average muscle activity of RMT did show evidence of a borderline decrease ($p = 0.050$). Peak muscle activity of the LMT showed a significant decrease over time ($p = 0.043$).
Table 4.7 shows that there was no evidence of a significant treatment effect of the intervention for the LMT (\(p = 0.299\)), RMT (\(p = 0.319\)), LLT (\(p = 0.600\)), and RLT (\(p = 0.215\)). For the greater part of the muscles, there was also an insignificant time effect and group effect – indicating that the changes were insignificant overall and the group differences (between intervention and control) were also not statistically significant. This is further highlighted in Figure 4.2 – the profile plot depicted is parallel and the rate of change was a slight decrease in both experimental groups over time.

### Figure 4.2 Mean muscle activity

#### 4.4.3.2 Peak Muscle Activity

Table 4.8 ANOVA for immediate effect on peak muscle activity by time and treatment group

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Statistical Test</th>
<th>Effect</th>
<th>Value</th>
<th>(p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LMT</td>
<td>Wilks’ lambda</td>
<td>Time</td>
<td>0.970</td>
<td>0.241</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time*group</td>
<td>0.970</td>
<td>0.307</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>Group</td>
<td>0.316</td>
<td>0.577</td>
</tr>
<tr>
<td>RMT</td>
<td>Wilks’ lambda</td>
<td>Time</td>
<td>0.906</td>
<td>0.034</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time*group</td>
<td>0.943</td>
<td>0.103</td>
</tr>
</tbody>
</table>
Table 4.8 illustrates that there was no evidence of a significant treatment effect of the intervention for LMT ($p = 0.307$), LLT ($p = 0.224$), and RLT ($p = 0.209$). Furthermore, there was no evidence of a treatment effect in the RMT either ($p = 0.103$). There was a significant treatment effect seen in the RMT over time ($p = 0.034$), however, this appears to be largely driven by the decrease in the control group; this is further illustrated in Figure 4.2 below.

<table>
<thead>
<tr>
<th></th>
<th>Wilks' lambda</th>
<th>F</th>
<th>Group</th>
<th>Time</th>
<th>Time*group</th>
</tr>
</thead>
<tbody>
<tr>
<td>LLT</td>
<td></td>
<td>0.989</td>
<td>0.968</td>
<td>0.224</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.317</td>
<td>0.474</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RLT</td>
<td></td>
<td>0.943</td>
<td>0.966</td>
<td>0.209</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.337</td>
<td>0.916</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.870</td>
<td>0.224</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 4.3 Peak muscle activity for right middle trapezius
4.4.3.3 Peak Amplitude

Table 4.9 ANOVA for immediate effect on peak amplitude by time and treatment group

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Statistical Test</th>
<th>Effect</th>
<th>Value</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>LMT</td>
<td>Wilks’ lambda</td>
<td>Time</td>
<td>0.933</td>
<td>0.588</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time*group</td>
<td>0.985</td>
<td>0.407</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>Group</td>
<td>0.702</td>
<td>0.407</td>
</tr>
<tr>
<td>RMT</td>
<td>Wilks’ lambda</td>
<td>Time</td>
<td>0.994</td>
<td>0.613</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time*group</td>
<td>0.977</td>
<td>0.304</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>Group</td>
<td>1.081</td>
<td>0.304</td>
</tr>
<tr>
<td>LLT</td>
<td>Wilks’ lambda</td>
<td>Time</td>
<td>0.870</td>
<td>0.012</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time*group</td>
<td>0.951</td>
<td>0.129</td>
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<tr>
<td></td>
<td>F</td>
<td>Group</td>
<td>2.386</td>
<td>0.129</td>
</tr>
<tr>
<td>RLT</td>
<td>Wilks’ lambda</td>
<td>Time</td>
<td>0.955</td>
<td>0.149</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time*group</td>
<td>0.993</td>
<td>0.572</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>Group</td>
<td>0.325</td>
<td>0.572</td>
</tr>
</tbody>
</table>

Table 4.9 shows that there was no evidence of a significant treatment effect of the intervention for the LMT ($p = 0.407$), RMT ($p = 0.304$), LLT ($p = 0.129$), and RLT ($p = 0.572$). For the majority of the muscles, there was also an insignificant time effect and group effect – indicating that the changes were insignificant overall, and there was no statistical difference between the two treatment groups.

4.4.3.4 Normalisation

Table 4.10 ANOVA for immediate effect on normalisation values by time and treatment group

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Statistical Test</th>
<th>Effect</th>
<th>Value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LMT</td>
<td>Wilks’ lambda</td>
<td>Time</td>
<td>0.971</td>
<td>0.251</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time*group</td>
<td>0.927</td>
<td>0.063</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>Group</td>
<td>1.683</td>
<td>0.201</td>
</tr>
<tr>
<td>RMT</td>
<td>Wilks’ lambda</td>
<td>Time</td>
<td>0.989</td>
<td>0.484</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time*group</td>
<td>0.991</td>
<td>0.515</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>Group</td>
<td>5.780</td>
<td>0.019</td>
</tr>
<tr>
<td>LLT</td>
<td>Wilks’ lambda</td>
<td>Time</td>
<td>0.980</td>
<td>0.341</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time*group</td>
<td>0.986</td>
<td>0.425</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>Group</td>
<td>0.022</td>
<td>0.883</td>
</tr>
<tr>
<td>RLT</td>
<td>Wilks’ lambda</td>
<td>Time</td>
<td>0.957</td>
<td>0.156</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Time*group</td>
<td>0.955</td>
<td>0.149</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>Group</td>
<td>1.178</td>
<td>0.283</td>
</tr>
</tbody>
</table>
Based on Table 4.10 above, it is evident that the LMT displayed no treatment effect \( (p = 0.063) \) as further illustrated by Figure 4.3. More so, there was no significant treatment effect for RMT \( (p = 0.515) \), LLT \( (p = 0.425) \), and RLT \( (p = 0.149) \).

Figure 4.4 Normalisation of muscle activity in left middle trapezius

4.5 Conclusion

This chapter summarises that thoracic spine grade III did not produce a significant change in muscle activity, nor did the control group. More so, there were no statistically significant differences between the two groups for sEMG readings, therefore the null hypothesis is not rejected. The interpretation and meaning of these results are interpreted in the next chapter.
CHAPTER 5: DISCUSSION

5.1 Introduction

This chapter entails the discussion of the results presented in Chapter Four in context of current literature. The statistical and clinical significance of the sEMG data from the intra-group and inter-group analysis will be discussed regarding possible theories. References will be made to relevant sections in Chapter Four as well as the studies reviewed in Chapter Two.

5.2 Demographic Data and Background Variables

5.2.1 Age

Age was an inclusion criterion in this study and was limited to individuals between the ages of 18 – 45 years. This age range was chosen as parental or guardian consent is always provided for research for participants below the age of 18 years and to restrict individuals in their fifth decade of life and onwards who would have had an increased likelihood of presenting with pathologies such as degeneration; this may have altered the results of the study (Walker et al. 2014; Boccia et al. 2015). Although age does not appear to influence sEMG levels at rest, age-related pathologies, such as spinal degeneration, may result in aberrant movement patterns and muscle co-contraction which would have resulted in poor sEMG data during MVIC (Crisswell 2010). Therefore, by limiting age, the risk of obtaining altered sEMG readings was minimised.

Table 4.1 shows that there was a mean age of 24 years in the intervention group and 25 years in the control group. It is evident that these groups were comparable to one another due to their mean values being so similar ($p = 0.492$). This is comparable to studies conducted by Krekoukias, Petty and Cheek (2007) where the participants had a mean age of 26.8 years and Pecos-Martin et al. (2015), where the mean age was 24 years.
5.2.2 Gender

Participants of both genders were involved in this study as gender is not a compounding nor varying factor when assessing muscle activity. According to Crisswell (2010), there are no specific distinctions or differences between genders when evaluating sEMG readings. More so, a study by Meduri et al. (2016) further emphasises that there is no statistically significant difference in sEMG readings between genders.

There were no significant differences found in the distribution of the women and men between the intervention and control groups, making these groups comparable. In the entire sample population, there was a greater percentage of male participants (52.1%) compared to female participants (47.9%). This is similar to previous studies where both genders were included, with male patients compromising the greater percentage within the sample size (Lee and Lee 2017; Shih et al. 2018).

5.2.3 Background Variables

The mean height of participants was 170cm and the mean weight was 69.7kg. The mean skinfold thickness of the total sample size revealed to be 8.6mm. Skinfold thickness of 19mm and above was an exclusion criteria because the presence of excess subcutaneous adipose tissue would have altered the accuracy of the sEMG readings (Baniqued et al. 2016). The measurement of skinfold was preferred instead of using body mass index (BMI); although body mass index provides insight to the overall health of the patient, it is not an accurate predictor of the amount of adipose tissue present directly over the trapezius muscles. Many patients have a BMI that indicates obesity because muscle weighs more than fat and their BMI readings are high, despite a low skinfold thickness. Therefore, skinfold thickness is a more accurate way to measure the amount of adipose tissue over the trapezius muscles (Etchison et al. 2011).
5.3 Muscle Activity

There was a statistically significant difference in the baseline sEMG normalisation values for the right middle trapezius between the intervention and control groups pre-test ($p = 0.036$) with the higher values being in the control group. However, this difference does not add value to the study, nor does it alter the results negatively, as this difference is noted in pre-test readings implying that no intervention or control had been implemented yet. The values for the remaining muscle groups showed no statistical significance.

5.3.1 Intervention Group

Neurophysiological changes occur when spinal mobilisation is applied to joints and this, in turn, alters muscle activity within the relevant muscles (Hegedus et al. 2011; Pecos-Martín et al. 2015). In previous studies, it had been observed that spinal manipulation therapy of fixated spinal segments in the spine had the ability to significantly alter muscle activity levels, by decreasing the muscle activity, of the muscles that attach to the respective areas of the spine (Haavik and Murphy 2012; Hengeveld, Banks and Maitland 2014; Masaracchio et al. 2019). Similarly, studies have demonstrated that spinal mobilisation therapy has the potential to achieve similar results (Pecos-Martín et al. 2015; Kamel, Raoof and Tantawy 2016; Patterson, Dickerson and Ribeiro 2020). In earlier literature, Krekoukias, Petty and Cheek (2007) reported that lumbar mobilisation caused a decrease in erector spinae muscle activity compared to the control and placebo groups. Similarly, Kamel, Raoof and Tantawy (2016) studied the effects of lumbar mobilisation in combination with ultrasound and infrared therapy in post-partum females with lower back pain and also found that lumbar mobilisation resulted in a significant decrease in erector spinae muscle activity compared to the placebo and control groups.

The result of this study is similar to the results of Krekoukias, Petty and Cheek (2007) and Kamel, Raoof and Tantawy (2016), but majority of the results were not significant meaning there was no overall change. The intervention group that received spinal
mobilisation displayed a decrease in muscle activity in the LMT ($p = 0.020$), the results of the remaining muscle groups were not significant. Although there was no overall statistical significance between the intervention and control groups, the findings in the left middle trapezius muscle of the intervention group are the nature of the trend that was, ultimately, expected for the entire study. There was a significant increase in the LLT peak amplitude ($p = 0.024$) over time, but due to it being the peak amplitude reading it is attributed to factors such as muscle fatigue and right-sided dominance.

5.3.2 Control Group

In the control group, there was a significant decrease in the peak muscle activity of the left middle trapezius ($p = 0.043$) and a borderline significant decrease in the mean muscle activity of the right middle trapezius over time ($p = 0.050$) there was no significant change in the remaining muscle groups. Due to these patients being asymptomatic, a decrease in muscle activity does not necessarily imply a clinical effect and could be attributed to muscle fatigue. Abboud, Nougarou and Descarreaux (2016) explains that joint dysfunction, whether there is pain or not, alters the normal biomechanics within the spine and, consequently, increases the fatigability of the muscles that are anatomically related to the dysfunctional joints. Although asymptomatic, the participants all presented with joint dysfunction within the thoracic spine and participants in the control group were, in essence, more prone to muscle fatigue than that of the intervention group due to the lack of spinal mobilisation therapy to the dysfunctional vertebral segments.

5.3.3 Comparison Between Intervention and Control Groups

Only asymptomatic participants were selected to be a part of the study in order to assess the effects of spinal mobilisation therapy without the added psychological and physiological consequences of pain. Pecos-Martín et al. (2015) suggested that future studies on spinal mobilisation should focus on asymptomatic participants because the response of symptomatic participants to spinal mobilisation would differ due to the neurophysiological consequences of pain. The use of an asymptomatic sample size was intended to further research and contribute to the body of knowledge by assessing
the effects of spinal mobilisation on dysfunctional joints without neurophysiological changes associated with the presence of pain.

In this study, thoracic grade III mobilisation did not result in any significant changes in the immediate readings of muscle activity of the middle and lower trapezius muscles which favours the null hypothesis. It was anticipated that due to the principle of the application of the mobilisation technique described by Bergmann and Peterson (2011) and the close anatomical relationship between the thoracic spine and the trapezius muscle, that applying spinal mobilisation therapy to the dysfunctional segments in the relevant levels would lead to changes in the activation of the middle and lower trapezius muscles within the intervention group. However, the findings of this differ from the findings of previous studies and are rather in accordance with Mehyar et al. (2017) who found that grade IV lumbar joint mobilisation did not alter the muscle activity of the erector spinae muscle and Shih et al. (2018) who observed that joint mobilisation improved perceived joint instability, mobility, and balance performance, but the effects of muscle activity remained uncertain.

Although there was no statistically significant effect and outcome of the thoracic spine grade III mobilisation in the middle and lower trapezius muscles, there was an evident trend between the participants who received the intervention compared to the control group. The participants that received the intervention inclined towards a decrease in muscle activity immediately after the spinal mobilisation when compared to the pre-intervention muscle activity values as seen by the marginal treatment effect apparent in the left middle trapezius muscle normalisation values ($p = 0.063$). This illustrates that spinal mobilisation did, in fact, elicit a beneficial effect on the muscle activity of the trapezius muscle and agrees with the findings from studies by Krekoukias, Petty and Cheek (2007), Pecos-Martín et al. (2015) and Kamel, Raoof and Tantawy (2016). However, the control group also elicited a decrease in muscle activity over time, but the clinical trend in this group was less distinct when compared to the intervention group. For the greater part of the study, there were no significant differences between the intervention and control groups in terms of establishing a treatment effect.
5.4 Conclusion

This study was not able to demonstrate that thoracic spine grade III mobilisation therapy was able to affect the muscle activity within the middle and lower trapezius muscles bilaterally. Although some indication of change in muscle activity was present, the data was not strong enough for there to be a statistically significant effect of the intervention on muscle activity when compared to the control group. However, if muscle activity readings were taken minutes after the immediate reading, these trends may have been of more statistical value. Therefore, further investigation is warranted.
CHAPTER 6: CONCLUSION, LIMITATIONS AND RECOMMENDATIONS

6.1 Conclusion

The results of this study show that, overall, there was no statistical difference between the two groups based on the sEMG measures, which suggests that grade III thoracic spine mobilisation did not result in a change in sEMG measures of the middle and lower trapezius muscle. There was a slight trend noted in the middle trapezius muscles when comparing the post-intervention data to the control group. However, these changes observed were not statistically significant. As a result, the null hypothesis was not rejected.

6.2 Limitations

The following limitations were identified throughout the study:

1. Although the power of the study was 81%, which is acceptable in the research context, the sample size (n=48) was too small, since slight trends were identified. The study could potentially have shown a positive treatment effect if the sample size was larger.
2. The placement of the sEMG electrodes. Although much effort was taken to warrant reproducibility, the exact sEMG electrode placement between participants could not be standardised or verified. It was challenging to isolate the lower trapezius muscle specifically and this should be considered in future sEMG studies when determining electrode placement and respective muscle contractions. In addition, the researcher was not blinded to treatment interventions and measurement readings leading to a potential bias. This was done for the sake of convenience only.
3. Effects on muscle activity over time and muscle fatigue was not accounted for. Muscle activity readings were recorded immediately after the intervention in order to establish the immediate effects of spinal mobilisation. If a second reading was
taken a few minutes after the immediate readings, clinical trends may have been more significant and muscle fatigue may not have been a factor.

### 6.3 Recommendations

In future research, the following recommendations should be considered:

- A larger sample size should be considered in future studies in order to further assess the positive trends present in this study.
- A research assistant should be considered in future studies to assist in sEMG electrode placement and study with the aim to standardise and verify electrode placement.
- Future studies should consider an additional sEMG reading, possibly ten minutes, after the immediate reading following the intervention to assess the effects of spinal mobilisation therapy on muscle activity over time.
- Additional measurement tools should be considered in further studies in order to add depth to the data obtained – for example thoracic ROM.
- Symptomatic participants should also be included into future studies to compare the effect of spinal mobilisation therapy on muscles that are already hypertonic to the point where they are causing pain.
REFERENCES


Crisswell, E. 2010. Cram's introduction to surface electromyography, 2d ed. 34 (Generic)


Krekoukias, G., Petty, N. J. and Cheek, L. 2007. Comparison of surface electromyographic activity of erector spinae before and after the application of central...


Appendix A: Permission to conduct research at DUT

14th May 2020

Ms Shilaya Elizabeth Smit
Department of Chiropractic and Osteopathy
Faculty of Health Sciences
Durban University of Technology

Dear Ms Smit,

PERMISSION TO CONDUCT RESEARCH AT THE DUT

Your email correspondence in respect of the above refers. I am pleased to inform you that the Institutional Research and Innovation Committee (IRIC) has granted Full Permission for you to conduct your research “The immediate effects of thoracic spine grade III mobilisation on muscle activity of the middle and lower trapezius muscles” at the Durban University of Technology.

The DUT may impose any other condition it deems appropriate in the circumstances having regard to nature and extent of access to and use of information requested.

We would be grateful if a summary of your key research findings can be submitted to the IRIC on completion of your studies.

Kindest regards,
Yours sincerely

DR LINDA ZIKHONI LINGANISO
DIRECTOR: RESEARCH AND POSTGRADUATE SUPPORT DIRECTORATE
MEMORANDUM

To: Prof Adam
   Chair: IREC

From: Dr Laura O’Connor
       Head of Department: Chiropractic

       Dr Desiree Varatharajullu
       Clinic Director: Chiropractic Day Clinic: Chiropractic

Date: 12.05.2020

Re: Request for permission to use the Chiropractic Day Clinic for research purposes

Permission is hereby granted to:

Ms Shinay Smit (Student Number: 21609341)

Research title: “The immediate effects of thoracic spine grade III mobilisation on the muscle activity of the middle and lower trapezius muscles”.

Ms Smit, is requested to submit a copy of her FRC/IREC approved proposal along with proof of her M.Tech: Chiropractic registration to the Clinic Administrator/s before she starts with her research in order that any special procedures with regards to her research can be implemented prior to the commencement of her seeing patients.

Thank you for your time.

Kind regards


Dr L O’Connor
Head of Department: Chiropractic

Dr D Varatharajullu
Clinic Director: Chiropractic Day Clinic: Chiropractic

Cc: Mrs Linda Twiggs: Chiropractic Day Clinic

Dr A. Abdul-Rasheed: Supervisor
Appendix C: IREC Approval

21 May 2020
Miss S E Smit
8 Broadview
11 Wallace Road
Durban
4001

Dear Miss Smit,

The immediate effects of the thoracic spine grade III mobilisation on muscle activity of the middle and lower trapezius muscle.

Ethical Clearance number IREC 016/20

The Institutional Research Ethics Committee acknowledges receipt of your gatekeeper permission letters.

Please note that FULL APPROVAL is granted to your research proposal. You may proceed with data collection.

Any adverse events [serious or minor] which occur in connection with this study and/or which may alter its ethical consideration must be reported to the IREC according to the IREC Standard Operating Procedures (SOP’s).

Please note that any deviations from the approved proposal require the approval of the IREC as outlined in the IREC SOP’s.

Yours Sincerely

Professor J K Adam
Chairperson: IREC
18 March 2020

To Whom It May Concern:

RE: The immediate effects of thoracic spine grade III mobilisation on the muscle activity of the middle and lower trapezius muscles.

As project manager for the Pan African Clinical Trial Registry (www.pactr.org) database, it is my pleasure to inform you that your application to our registry has been accepted. Your unique identification number for the registry is PACTR202003504433002.

Please be advised that you are responsible for updating your trial, or for informing us of changes to your trial.

Please note that it is now a WHO requirement to include, at a minimum, summary results or a link to summary results within the trial registration record. This should be done within 12 months of the study completion date.

Additionally, please provide us with copies of your ethical clearance letters as we must have these on file (via email or post or by uploading online) at your earliest convenience if you have not already done so.

Please do not hesitate to contact us at +27 21 938 0835 or email epienar@mrc.ac.za should you have any questions.

Yours faithfully,

Elizabeth D Piernaar
www.pactr.org Project Manager
+27 021 938 0835
Appendix E: Advertisement

Are you between the ages of 18 – 45?

Are you free of upper back pain?

Do you want to be involved in an interesting research study?

Contact:

Shinay Smit  -  083 637 1182
OR
DUT Chiropractic Clinic  –  031 373 2511

For more information
Ingabe Uneminyaka eyi-18 kuya kuma-45?

INGABE UKHULULEKILE, AWUNABO
UBUHLUNGU BOMGO GODLA NGENHLA?

Ungathanda ukubamba iqhaza kucwankingo oluthakazelisayo nolunetshisekelo?

Xhumana:
NoShinay Smit – 083 637 1182

NOMA

Umtholampilo weKhayirophrakthikhi wase Nyuvesi yaseThekwini – 031 373 2511

Ukuze uthole yonke imininingwane mayelana nalolu cwankingo
27 July 2019

Request for Permission to Conduct Research

Dear Sir/Madam

My name is Shinay Elizabeth Smit, a BTech Chiropractic student at the Durban University of Technology. The research I wish to conduct for my Masters dissertation involves the immediate effects of thoracic spine grade III mobilization on the muscle activity of selected muscles in the upper thoracic region.

I am hereby seeking your consent to place an advertisement, which is attached, on your premises to recruit participants for my research.

I have provided you with a copy of my proposal which includes copies of the data collection tools and consent and/or assent forms to be used in the research process, as well as a copy of the approval letter which I received from the Institutional Research Ethics Committee (IREC).

If you require any further information, please do not hesitate to contact me (083 637 1182; shinay123@gmail.com). Thank you for your time and consideration in this matter.

Yours sincerely,

Shinay Elizabeth Smit
Durban University of Technology
Appendix G: Letter of Information and Informed Consent (in English and isiZulu)

LETTER OF INFORMATION

Title of the Research Study: The immediate effects of thoracic spine grade III mobilisation on the muscle activity of the trapezius muscle.

Principal Investigator/researcher: Shiny Elizabeth Smit [Btech: Chiropractic]

Co-Investigator/supervisor(s): Dr. Ashura Abdul-Rasheed [MTech: Chiropractic]

Brief Introduction and Purpose of the Study: This research study aims to determine the immediate effects of thoracic spine grade III mobilisation on the muscle activity of the trapezius muscles in asymptomatic patients. 48 individuals will be required to complete the study.

Outline of the Procedures: If you agree to take part in this research, you will need to sign a consent form. Following this, the researcher will take a case history and perform a general physical examination and combined spinal regional examination. This is done to ensure that you meet the inclusion criteria of the study. If you do, you will then be allocated to one of the two groups in the research – the one group will be the intervention group and the other group will be a control group to compare the intervention to. You will then be asked to remove any clothing covering the upper back area and appropriate clothing (clinic gown for females) will be provided. You may be required to remove any hair in the upper back region for placement of the electrodes in this area. Three baseline sEMG readings will be obtained while you are asked to perform specific muscle contractions for 10 seconds each time. Treatment will then be administered according to group allocation. If you are in the intervention group, you will receive a grade III spinal mobilisation at fixedated areas and if you are in the control group, you will receive an alternative intervention. sEMG readings will then be taken straight after the intervention where you will perform the same muscle contractions again.

Risks or Discomforts to the Participant: The treatment intervention or control given to you will be under the supervision of a qualified chiropractor at all times. Spinal mobilisation is a non-invasive form of manual therapy and is very safe.

Benefits: You will benefit by receiving insight into what chiropractors do and I, the researcher, will benefit by completing my dissertation and publishing it.

Reasons why the Participant May Be Withdrawn from the Study: If you experience any adverse effects and wish to withdraw from the study – you are allowed to do so. If you are not compliant with what is expected of you during the study, you will be withdrawn. You are free to withdraw from the study at any time and withdrawal will not prevent you from receiving future treatment at the Chiropractic Day Clinic at normal clinic rates.

Remuneration: There will be no form of remuneration offered to you for taking part in this study.

Costs of the Study: You will not have to contribute to any costs of the study.
Confidentiality: All your medical information will be kept confidential and will be stored at the Chiropractic Day Clinic for 5 years – after which it will be shredded. All electronic data will be password-secured and stored in the Chiropractic Day Clinic for 5 years – after which it will be deleted as well. Your name and personal details will not appear on any of the data sheets nor will it appear in the dissertation.

Research-related Injury: No injuries are anticipated during this study. There will be no compensation in the event of an injury.

Persons to Contact in the Event of Any Problems or Queries:

Please contact the researcher, Shinay Smit on 083 637 1182, my supervisor, Dr Ashura Abdul-Rasheed on 060 506 2394 or the Institutional Research Ethics Administrator on 031 373 2375. Complaints can be reported to the Director: Research and Postgraduate support, Prof. Kevin Duffy on 031 373 2377 or kevind@duc.ac.za.

Yours Sincerely,

Shinay Elizabeth Smit
(Researcher)
CONSENT

Statement of Agreement to Participate in the Research Study:

- I hereby confirm that I have been informed by the researcher, Shinay Elizabeth Smit, about the nature, conduct, benefits and risks of this study - Research Ethics Clearance Number: 016/20.
- I have also received, read and understood the above written information (Participant Letter of Information) regarding the study.
- I am aware that the results of the study, including personal details regarding my sex, age, date of birth, initials and diagnosis will be anonymously processed into a study report.
- In view of the requirements of research, I agree that the data collected during this study can be processed in a computerised system by the researcher.
- I may, at any stage, without prejudice, withdraw my consent and participation in the study.
- I have had sufficient opportunity to ask questions and (of my own free will) declare myself prepared to participate in the study.
- I understand that significant new findings developed during the course of this research which may relate to my participation will be made available to me.

_________________________  __________________  _____________
Full Name of Participant    Date        Time         Signature / Right Thumbprint

I, Shinay Elizabeth Smit, herewith confirm that the above participant has been fully informed about the nature, conduct and risks of the above study.

_________________________
Shinay Elizabeth Smit       Date          Signature

_________________________
Full Name of Witness (If applicable)  Date          Signature

_________________________
Full Name of Legal Guardian (If applicable)  Date          Signature

100
INCWADI YOLWAZI

Isihloko socwania go sophenyw: Imiphumela epheshayo yesiwayo se-III yokugqugquzuza ngokwelulwa komgqogqo maphakathi emsebenzini weziciwe zomzipa wemase la ithraperheziyasi.

Umphenyi/ umcwanyi omkhulu: UShinayi Elizabeth Smit [i-BTech: yeKhayirophraktikhi]

Isingeniso kufushane kanye nenhliso yocwania go yophenyw: Lolu cwaningolo luhoze ukuthola imiphumela epheshayo yokugqugquzela ngokwelulwa komgqogqo maphakathi emsebenzini weziciwe zomzipa wemase la ithraperheziyasi kwizigulul ozingakhombisi izimpele. Kuzodingekela abantu abangana - 48 ukuse kuhlungulwwe lolu cwaningolo.


Ubugozi noma Ukuphazamiseka kukambambiqhaza: Ukweselwa okanye ukualulwa ozonwaka kona njalo kuzobe kuqashwe iKhayirophraktikha esigqoqile. Ukweselwa umgqogqo kuyendlela engiakhlaseli, eyenzwa ngezindla futhi iphephile kakhulu.

inzuko: Uzozusa ngokwanda kabanezi lokho okwenziwa anaKhayirophraktikha bese kusho mina, mwcwania, ngizozusa ngokuthi ngiphuthule incwandy esicwcleniye ngicwcleniye bese njisyanicilela ukuthi ibonwe yizwe lonke.

Umkomelo/ umholo: Angeke kube khona mhlo okanye mali ozoyinkeswa ngokumbamba iqhaza kuolu cuwinge.

Inani elikhokhwa umbambiqhaza wocwaninggo/ wopheny: Ayikho imali ozoyikhokha ngokuqhanganyela okanye ngokumbamba kwakho iqhaza kuolu cuwinge.

Ubumfihiho: Wonke amarekhodi aphathelene nokwelaswa kwakho zozocinwa eyinfithlo lutsho evutuwe emtholampilo weKhayirophrakthikhisi iminyaka ey-i, bese kuthi emusa kwakholo azobu eseyakhaphazwa/ erazi ezimthi. Wonke zama-elektronikhi khophi nzwedla zozocinwa evukela ngephasiwedi emtholampilo weKhayirophrakthikhisi iminyaka ey-i bese emusa kwakholo ziyaciswa. Igama Isiko angeke isichicilele kwizaba shithi okanye kwincwadi yami yezocwaninggo.


Abantu ongaxhuma nabo uma unezinkinga okanye unemibuzo:

Uyaqela ukuqulwa ukhuma nomcwangeni, uShinay Smit ku 083 637 1182, umphathi wani omkhulu, uDkt. Ashura Abd-Rashed ku 060 500 2394, nomu Umwiluli wewiniso zokuhle kwacwaninggo weSidshingku ku 031 373 2375. Izinkonondo okanye isikhulazfo zingxelulisele kuMphathshi/Mpondisi: Wasesekelo saZocwaninggo, uSolwazi Kevin Duffy ku 031 373 2577 nomu ku kevird@dur.ac.za.

Yini ozithobyo,

UShinay Elizabeth Smit
(Umcwaningi/ umphenyi)
IMYUME

Isitamendwe semvumelwano yokubamba iqhaza kucwango/ kuphensyo:

- Mina ngiyaphelele ukuthi umcowaningi: uShiray Elizabeth Smit ungazisile rgenkambo, uhlobo, inzuze kanye noburongoza balolu cwango- hombolo yezimiso zokuhle yokwaningi: 016/20
- Ulwazi kanye nencelelo emvelana nalo lu cwozingo ebhalwe lapha ngenhla (Incwadi Yolwazi laMbambiqhaza) nayo ngiyitholile, ngayifunda futhi ngayiqondisa.
- Ngisqonda ukuthi inphumela yokwaningi, okubala iminingiwe yami yobuhlili, iminyaka, usuku lokuzala, ama-nishi�i ni nokuthi ngaphethwe yini kuzosethenshawu ngokungaziwa/ ngobumfihlo ukuse kwenzwiwe umbiko wokwaningi.
- Ngokubona izinga zokwaningi, mina ngiyazume ukuthi iminingiwe yami etholakale kulolu cwozingo isethenshawu ngu mcowangi ohleleni lesekelo ephumuleni.
- Ngingakwazi, kunoma yisiphi isigaba, ukuthi ngaphamisa imyume nokubamba kwami iqhaza kulolu cwozingo ngalo kwengicindezi.
- Ngibe nethu Salvation Lokuzela imibuzo futhi (ngokuzikhethele/ ngokuzithandela kwami) ngiyakhe ukuthi ngikulungele ukubamba iqhaza kulolu cwozingo.
- Ngisqonda ukuthi lolo lwazi olusha, olubalulekile futhi olushintana nokubamba kwami iqhaza rgenkathi kwenzwiwe lolu cwozingo agizovumela ukulwazi.

Igama eliphelele loMbambiqhaza Usuku Isikathi Isiginisha /Isithupha
sakwisasisa sokukuda

Mina, Shiray Elizabeth Smit: ngiyaphelele ukuthi lo mambiqhaza orgeniwa uchestlewe kabanzi ngohlobo, inlambelo kanye noburongoza balolu cwozingo olungeniwa.

Shiray Elizabeth Smit
Igama eliphelele loMcowaningi Usuku Isiginisha

Igama eliphelele likaFakazi (Uma kufaneleleki) Usuku Isiginisha

Igama eliphelele loMnakeleli Osemthethweni (Uma kufaneleleki) Usuku Isiginisha
1. **Source of History:**

2. **Chief Complaint:** (patient's own words):

3. **Present Illness:**

<table>
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<tr>
<th>Complaint 1 (principal complaint)</th>
<th>Complaint 2 (additional or secondary complaint)</th>
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<td>Previous Occurrences</td>
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<td>Past Treatment</td>
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<tr>
<td>Outcome</td>
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4. **Other Complaints:**

5. **Past Medical History:**
   - General Health Status
   - Childhood Illnesses
   - Adult Illnesses
   - Psychiatric Illnesses
   - Accidents/Injuries
   - Surgery
   - Hospitalizations
6. **Current health status and lifestyle:**

- Allergies
- Immunizations
- Screening Tests incl. x-rays
- Environmental Hazards (Home, School, Work)
- Exercise and Leisure
- Sleep Patterns
- Diet

**Current Medication**
- Analgesics/week:
- Other (please list):

**Tobacco**
- Alcohol
- Social Drugs

7. **Immediate Family Medical History:**

- Age of all family members
- Health of all family members
- Cause of Death of any family members

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<td>TB</td>
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<td></td>
<td>Other (list)</td>
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</tbody>
</table>

8. **Psychosocial history:**

- Home Situation and daily life
- Important experiences
- Religious Beliefs
9. **Review of Systems** (please highlight with an asterisk those areas that are a problem for the patient and require further investigation)

- General
- Skin
- Head
- Eyes
- Ears
- Nose/Sinuses
- Mouth/Throat
- Neck
- Breasts
- Respiratory
- Cardiac
- Gastro-intestinal
- Urinary
- Genital
- Vascular
- Musculoskeletal
- Neurologic
- Haematological
- Endocrine
- Psychiatric
Appendix I: Physical Examination

CHIROPRACTIC DAY CLINIC
PHYSICAL EXAMINATION

Patient: ___________________________ File#: ________ Date: ________

Clinician: __________________________ Signature: __________________________

Student: __________________________ Signature: __________________________

1. **Vitals**

   Pulse rate: __________________________
   Respiratory rate: __________________________
   Blood pressure: R L __________________________ Medication if hypertensive: __________________________
   Temperature: __________________________
   Height: __________________________
   Weight: __________________________ Any change Y/N If Yes: how much gain/loss __________________________
   Over what period: __________________________

2. **General Examination**

   General Impression: __________________________
   Skin: __________________________
   Jaundice: __________________________
   Pallor: __________________________
   Clubbing: __________________________
   Cyanosis (Central/Peripheral): __________________________
   Oedema: __________________________
   Lymph nodes: - Head and neck: __________________________
   - Axillary: __________________________
   - Epitrochlear: __________________________
   - Inguinal: __________________________
   Urinalysis: __________________________

3. **Cardiovascular Examination**

   1) Is this patient in **Cardiac Failure**?
   2) Does this patient have signs of **Infective Endocarditic**?
   3) Does this patient have **Rheumatic Heart Disease**?

   **Inspection**: - Scars - Chest deformity: - Precordial bulge: - Neck -JVP:

   **Palpation**: - Apex Beat (character + location): - Right or left ventricular heave: - Epigastric Pulsations:
   - Palpable P2: Palpable A2:

Page 1 of 5
Pulses:  - General Impression:  - Dorsalis pedis:
   - Femoral:
   - Posterior tibial:
   - Popliteal:
   - Carotid:
   - Radial:

Percussion:  - borders of heart

Auscultation:  - heart valves (mitral, aortic, tricuspid, pulmonary)
   - Murmurs (timing, systolic/diastolic, site, radiation, grade).

4. **RESPIRATORY EXAMINATION**

1) Is this patient in Respiratory Distress?

**Inspection**
   - Barrel chest:
   - Pector carinatum/cavintum:
   - Left precordial bulge:
   - Symmetry of movement:
   - Scars:

**Palpation**
   - Tracheal symmetry:
   - Tracheal tug:
   - Thyroid Gland:
   - Symmetry of movement (ant + post)
   - Tactile fremitus:

**Percussion**
   - Percussion note:
   - Cardiac dullness:
   - Liver dullness:

**Auscultation**
   - Normal breath sounds bilateral:
   - Adventitious sounds (crackles, wheezes, crepitations)
   - Pleural frictional rub:
   - Vocal resonance
   - Whispering pectoriloquy:
   - Bronchophony:
   - Egophony:

5. **ABDOMINAL EXAMINATION**

1) Is this patient in Liver Failure?

**Inspection**
   - Shape:
   - Scars:
   - Hernias:

**Palpation**
   - Superficial:
   - Deep = Organomegally:
   - Masses (intra- or extramural)
   - Aorta:

**Percussion**
   - Rebound tenderness:
   - Ascites:
   - Masses:

**Auscultation**
   - Bowel sounds:
   - Arteries (aortic, renal, iliac, femoral, hepatic)

**Rectal Examination**
   - Perianal skin:
   - Sphincter tone & S4 Dermatome:
   - Obvious masses:
   - Prostate:
   - Appendix:
6. **G.I.T. EXAMINATION**

External genitilia:
Hernias:
Masses:
Discharges:

7. **NEUROLOGICAL EXAMINATION**

**Gait and Posture:**
- Abnormalities in gait:
  - Walking on heels (L4-L5):
  - Walking on toes (S1-S2):
  - Romberg’s test (Pronator Drift):

**Higher Mental Function:**
- Information and Vocabulary:
- Calculating ability:
- Abstract Thinking:

**G.C.S.:**
- Eyes:
- Motor:
- Verbal:

**EVIDENCE OF HEAD TRAUMA:**

**Evidence of Meningism:**
- Neck mobility and Brudzinski’s sign:
- Kernig’s sign:

**Cranial Nerves:**

I Any loss of smell/taste:
Nose examination:

II External examination of eye:
- Visual Acuity:
  - Visual fields by confrontation:
    - Pupillary light reflexes
      = Direct:
      = Consensual:
    - Fundoscopy findings:

III Ocular Muscles:
Eye opening strength:

IV Inferior and Medial movement of eye

V a. Sensory
  - Ophthalmic:
    - Maxillary:
    - Mandibular:
  b. Motor
    - Masseter:
    - Jaw lateral movement:
  c. Reflexes
    - Corneal reflex
    - Jaw jerk

VI Lateral movement of eyes

VII a. Motor - Raise eyebrows:
  - Frown:
    - Close eyes against resistance:
    - Show teeth:
    - Blow out cheeks:
  b. Taste - Anterior two-thirds of tongue:
VIII General Hearing:
  Rinn's = L:
  R:
Weber's lateralisation:
Vestibular function
  - Nystagmus:
  - Romberg's:
  - Wallenberg's:

Otoscope examination:

IX & Gag reflex:

X uvula deviation:
Speech quality:

XI Shoulder lift:
S.C.M. strength:

XII Inspection of tongue (deviation):

Motor System:

a. Power
  - Shoulder = Abduction & Adduction:
  = Flexion & Extension:
  - Elbow = Flexion & Extension:
  - Wrist = Flexion & Extension:
  - Forearm = Supination & Pronation:
  - Fingers = Extension (Interphalangeals & M.C.P's):
  - Thumb = Opposition:
  - Hip = Flexion & Extension:
  = Adduction & Abduction:
  - Knee = Flexion & Extension:
  - Foot = Dorsiflexion & Plantar flexion:
  = Inversion & Eversion:
  = Toe (Plantarflexion & Dorsiflexion):

b. Tone
  - Shoulder:
    - Elbow:
    - Wrist:
      - Lower limb - Int. & Ext. rotation:
  - Knee clonus:
  - ankle clonus:

c. Reflexes
  - Biceps:
  - Triceps:
  - Supinator:
  - Knee:
  - Ankle:
  - Abdominal:
  - Plantar:
Sensory System:
a. Dermatomes
   - Light touch:
   - Crude touch:
   - Pain:
   - Temperature:
   - Two point discrimination:
b. Joint position sense
   - Finger:
   - Toe:
c. Vibration:
   - Big toe:
   - Tibial tuberosity:
   - ASIS:
   - Interphalangeal Joint:
   - Sternum:

Cerebellar function:
Obvious signs of cerebellar dysfunction:
   = Intention Tremor:
   = Nystagmus:
   = Truncal Ataxia:
Finger-nose test (Dysmetria):
Rapid alternating movements (Dysdiadochokinesia):
Heel-shin test:
Heel-toe gait:
Reflexes:
Signs of Parkinsons:

8. SPINAL EXAMINATION: (SEE REGIONAL EXAMINATION)
Obvious Abnormalities:
Spinal Percussion:
R.O.M:
Other:

9. BREAST EXAMINATION:
Summon female chaperon.

Inspection
   - Hands rested in lap:
   - Hands pressed on hips:
   - Arms above head:
   - Leaning forward:

Palpation
   - masses:
   - tenderness:
   - axillary tail:
   - nipple:
   - regional lymph nodes:
Appendix J: Thoracic Spine Regional Examination

**THORACIC SPINE REGIONAL EXAMINATION**

Patient: __________________________ File: __________ Date: __________

Student: __________________________ Signature: __________________

Clinician: ________________________ Signature: __________________

**STANDING:**
- Posture (incl L/S & C/S)
- Muscle tone
- Skyline view – Scoliosis
- Spinal Percussion
- Breathing (quality, rate, rhythm, effort)
- Deep Inspiration

**RANGE OF MOTION:**
- Forward Flexion 20 – 45 degrees (15cm from floor)
- Extension 25 – 45 degrees
- L/R Rotation 35 – 50 degrees
- L/R Lat Flex 20 – 40 degrees

**RESISTED ISOMETRIC MOVEMENTS:** (in neutral)
- Forward Flexion
- Extension
- L/R Rotation
- L/R Lat Flexion

**SEATED:**
- Palpate Auxillary Lymph Nodes
- Palpate Ant/Post Chest Wall
- Costo vertebral Expansion (1 – 7cm diff. at 4th intercostal space)
- Slump Test (Dural Stretch Test): LOCAL PAIN (T/S) DISTAL PAIN (L/S) DISTAL PAIN (LEG)

**SUPINE:**
- Rib Motion (Costo Chondral joints)
- Soto Hall Test (#, Sprains)
- SLR
- Palpate abdomen

**PRONE:**

---

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### Passive Scapular Approximation
### Facet Joint Challenge
### Vertebral Pressure (P-A central unilateral, transverse)
### Active myofascial trigger points:

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<th>Active</th>
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<td>Lower Trapezius</td>
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<td>Serratus Posterior</td>
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<td>Pectoralis Major</td>
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<td>Quadratus Lumborum</td>
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### COMMENTS:

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### Basic LOWER LIMB (neuro):

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### MOTION PALPATION:

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