The effect of lumbar spine manipulation on the muscle activity of the quadriceps femoris and hamstring muscle groups

By

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I, Jenna-Leigh Parkes, 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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DEDICATION

To my parents, Alan and Jenny. Thank you for all of your love, guidance and, support. Your sacrifices, although some unacknowledged, never go unnoticed. Thank you for providing me with a life filled with incredible opportunities, this journey would never have been possible without you. It’s with an endless amount of love and deep gratitude that I dedicate this to you.

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ABSTRACT

Background: The clinical use of spinal manipulative therapy is becoming increasingly evident in the treatment of musculoskeletal conditions. However, the exact neurophysiological mechanisms behind spinal manipulative therapy and its effects on muscle activity require further investigation. Fixated joints within the spine have been shown to cause changes in muscle activity in both segmentally related muscles and extremity muscles. When present in symptomatic or asymptomatic individuals, fixations in the spine may not only lead to local neurophysiological changes but may affect global neurophysiology. Chronic lower back pain has been associated with the presence of arthrogenic muscle inhibition in lower limb musculature which prevents an individual from fully activating the affected muscle. Although this form of inhibition is predominately present in symptomatic individuals, there is evidence to suggest that asymptomatic individuals undergo a similar neuromuscular change. However, further research is needed to determine if the extent of the effects of arthrogenic muscle inhibition differs between symptomatic and asymptomatic subjects.

Aim: This study aims to determine and compare the effects of a lumbar spine manipulation on the muscle activity in the quadriceps femoris and hamstring muscle groups in asymptomatic and symptomatic participants.

Method: This study was a randomised controlled trial which utilised a pre- and post-experimental design. A total of 48 participants between the ages of 18-45 years of both genders and all races were recruited. The sample population included a symptomatic and asymptomatic clinical group. Each clinical group had a treatment and control group. Once placed into their respective groups, individuals were randomly allocated to the intervention or control group. Muscle activity readings of the quadriceps femoris and hamstring muscles were obtained during sets of maximum voluntary contractions that occurred at the pre-intervention/control, post-intervention/control and post 10-minute intervention/control. IBM SPSS version 26 was used to analyse the data. Repeated measures ANOVA tests were used to compare each outcome between pre- and immediate post-treatment between the intervention and control groups for immediate effects, and pre- and 10-minute post-treatment between the intervention

and control groups for short-term effects. This was initially done within the asymptomatic and symptomatic participants separately.

Results: There were no statistically significant differences between the age and gender of the symptomatic and asymptomatic groups. With regards to muscle activity, no significant changes in the quadriceps femoris or hamstring muscle groups were identified in the asymptomatic group. Significant changes were detected in the right rectus femoris of the quadriceps muscle demonstrating evidence of a treatment effect (p=0.047). However, this change did not persist in the 10-minute readings. Although not statistically significant, there was clear evidence of a clinical trend that presented in the quadriceps femoris muscle (rectus femoris and vastus medialis) of the symptomatic group, as the overall muscle activity of the participants who received the intervention increased bilaterally. There were no statistically significant differences found when comparing the data between the symptomatic and asymptomatic clinical groups.

Conclusion: The results showed that lumbar spine manipulation did not produce significant differences in the immediate post-readings and the 10-minute readings in the quadriceps femoris and hamstring muscles of the symptomatic and asymptomatic groups. When comparing the clinical groups, there was no significant difference between the asymptomatic and symptomatic groups in terms of the pre-intervention readings of muscle activity and the post-intervention measures immediately and at the 10-minute interval following lumbar spine manipulation.

Key Indexing Terms: *Spinal manipulative therapy, muscle activity, arthrogenic muscle inhibition, quadriceps femoris, hamstrings*

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ABBREVIATIONS

Ach	:	Acetylcholine
AchE	:	Acetylcholinesterase
AP	:	Action potential
cm	:	centimetres
CN	:	Cranial nerves
CNS	:	Central nervous system
DJD	:	Degenerative joint disorder
DUT	:	Durban University of Technology
EMG	:	Electromyography
GTO	:	golgi tendon organ
Hz	:	Hertz
IREC	:	Institutional Research Ethics Committee
IVD	:	Intervertebral disc
IVF	:	Intervertebral foramen
LHAM	:	Left Hamstring
LRF	:	Left Rectus Femoris
L/S	:	Lumbar spine
LSM	:	Lumbar spine manipulation
LVM	:	Left Vastus Medialis
MVC	:	maximum voluntary contraction
MVIC	:	maximum voluntary isometric contraction
n	:	sample number
p	:	probability
PNS	:	peripheral nervous system
RCT	:	Randomised clinical trial

RHAM: Right Hamstring

RMS : Root mean square

ROM : Range of motion

RRF : Right Rectus Femoris

RVM : Right Vastus Medialis

SC : Spinal cord

sEMG : Surface electromyography

SM : Spinal manipulation

SMT : Spinal manipulative therapy

SP : Spinous process

TVP : Transverse process

VB : Vertebral body

VC : Vertebral column

% : percentage

DEFINITIONS

Afferent nerve:

A sensory nerve conveying impulses from the peripheral nervous system (PNS) to the central nervous system (CNS) (Tortora and Derrickson 2017).

Asymptomatic:

Without symptoms (Venes 2017).

Efferent neuron:

A nerve that conveys impulses from the brain or CNS towards the cranial and peripheral nerves (Tortora and Derrickson 2017).

Facilitation:

Hyperactivity or reinforcement of a reflex (Venes 2017).

Inhibition:

The weakening or depression of reflex activity (Venes 2017).

Joint Fixation/Joint Dysfunction:

Spinal segment where there is an alteration in the biomechanical relationship of two or more articulating joint surfaces that may occur in one or more directions (Bergman and Peterson 2011).

Motion palpation:

Procedure for the assessment and diagnosis of static and active range of motion of joints in the spine and extremities (Bergman and Peterson 2011).

Nociceptive:

Stimulus-response process involving the stimulation of pain sensitive nociceptors and the transmission of impulses along the peripheral nerves to the CNS (Venes 2017).

Pathological:

The physical, functional, biochemical and immunological changes caused by illness or disease (Venes 2017).

CHAPTER ONE: INTRODUCTION

This chapter serves to provides a brief overview of the study which will include the aims and objectives followed by the study rationale.

1.1 Introduction

A joint fixation within the vertebral column can be defined as a spinal segment where there is an alteration in the biomechanical relationship of two or more articulating joint surfaces (Haldeman 2004; Bergman and Peterson 2011). These joint fixations have various mechanisms of development or origin, and often present as a restriction in movement which can be identified during specific motion palpation of the spine. Therefore, when a spinal segment becomes hypomobile, due to biomechanical overload or other mechanical stresses, that particular spinal segment can be termed as fixated or dysfunctional (Owens Jr 2002; Henderson 2012).

Fixated or dysfunctional spinal segments may present with or without pain and have been shown to have several neurophysiological effects which may cause changes in the musculature around the spine (DeVocht, Pickar and Wilder 2005). These changes may also occur in muscles that are not anatomically connected to the spine but rather share a neurological link to the affected spinal levels (Dunning and Rushton 2009). One of the consequences of joint injury or fixation is the process known as arthrogenic muscle inhibition (AMI). AMI is a persistent reflex that occurs in response to injury or dysfunction of a joint and presents as a neural inhibition of muscles surrounding the injured joint leading to the inability to recruit or activate all motor units of a functional muscle group. This reflex is often experienced by symptomatic patients (i.e. low back pain) (Suter *et al.* 2000; Hammill, Beazell and Hart 2008).

Patients who suffer from low back pain often show evidence of AMI which may include changes in muscle activity and force output, as well as altered motor recruitment patterns (Bruno and Bagust 2007; Rice *et al.* 2014). Research has found that injury to the spinal column may cause the surrounding/local musculature as well as distal muscles, that are innervated by those particular levels, to undergo AMI (Fryer, Morris and Gibbons 2004; Hart *et al.* 2006). This is supported by Suter and Lindsay (2001), Verbunt *et al.* (2005) and Hart, Weltman and Ingersoll (2010) as they found that

participants with chronic lower back pain (CLBP) demonstrated a significant association to quadriceps inhibition when they were unable to fully activate the knee extensor. It was evident that the quadriceps muscle of the CLBP subjects had undergone AMI similar to the musculature surrounding the spine (erector spinae).

Spinal manipulative therapy (SMT) is used as a primary tool of treatment by chiropractors and other manual therapy practitioners for several neuromuscular conditions such as neck pain, headaches, and low back pain (Bronfort *et al.* 2012; Chaibi *et al.* 2017). SMT is applied to hypomobile joints, which are found during an assessment of joint motion and congruency, and is used to restore normal arthrokinematics of that particular joint. SMT allows for the restoration of normal joint mobility at the fixation and causes a stimulation of mechanoreceptors that are found within tissues in and around the spinal segment, which has been shown to cause changes within the spinal cord at that particular spinal segment restoring normal neurophysiological function (DeVocht, Pickar and Wilder 2005; Pickar and Bolton 2012). It has been theorised that SMT not only affects joint mobility but may also cause stimulation or modulation of the neuromuscular reflexes that will help normalise muscle activity (Pickar 2002). The stimulation or modulation of these reflexes through SMT has not only been shown to cause changes in the activity of the muscles around the affected spinal segment but can cause changes in the activity of extremity muscles that have a neurological connection to the dysfunctional or fixated spinal segments (Suter and McMorland 2002; Dunning and Rushton 2009).

Research has shown that SMT can elicit an effect on both asymptomatic and symptomatic individuals. Grindstaff *et al.* (2009) showed significant changes in quadriceps muscle activation and force output following a lumbopelvic joint manipulation in asymptomatic individuals (n=42). Niazi *et al.* (2015) found that SMT applied to dysfunctional segments found in the spine and sacroiliac joints in a subclinical pain population resulted in an increase in muscle activity and strength in the soleus muscle. Studies that suggest that spinal manipulation can affect muscle activity are further supported by Suter and McMorland (2002) where a cervical spine manipulation was shown to decrease elbow flexor inhibition in chronic neck pain patients (n=16); and Dunning and Rushton (2009) where results revealed that a single cervical spine manipulation caused a significant increase in the elbow flexors resting

activity in asymptomatic patients (n=54) bilaterally. Several previous studies have only examined the immediate effects of SMT on distal muscles (Suter and McMorland 2002; Dunning and Rushton 2009).

It has become evident that SMT can cause changes in the activity in muscles of the spine and extremities, but the actual mechanism of these changes is still not fully understood. This study aims to assess and compare the effects of spinal manipulative therapy on neuroanatomically connected segments of the lumbar spine to the quadriceps femoris and hamstring muscle groups in asymptomatic and symptomatic participants. The study will be used to investigate the immediate and short-term duration effects of lumbar spine manipulative therapy, and to determine whether the pain has a significant effect on these measurements.

1.2 Study Aims and Objectives

1.2.1 Aim

The aim of this study is to determine and compare the effects of a lumbar spine manipulation on the muscle activity in the quadriceps femoris and hamstring muscle groups in asymptomatic and symptomatic participants.

1.2.2 Objectives

Objective One

To determine the immediate and short-term effect of lumbar spinal manipulative therapy on the muscle activity on the quadriceps femoris and hamstring muscle groups in asymptomatic participants.

Objective Two

To determine the immediate and short-term effect of the lumbar spinal manipulative therapy on the muscle activity on the quadriceps femoris and hamstring muscle groups in symptomatic participants.

Objective Three

To compare and correlate the data between the asymptomatic and symptomatic groups.

1.3 Hypotheses

1.3.1 Null Hypotheses

1.3.1.1 Null Hypothesis One

There will be no significant difference between the pre-intervention readings of muscle activity and the post-intervention measures immediately and at the 10-minute interval following lumbar spine manipulation in asymptomatic participants ($p>0.05$).

1.3.1.2 Null Hypothesis Two

There will be no significant difference between the pre-intervention readings of muscle activity and the post-intervention measures immediately and at the 10-minute interval following lumbar spine manipulation in symptomatic participants ($p>0.05$).

1.3.1.3 Null Hypothesis Three

There will be no significant difference between the asymptomatic and symptomatic groups in terms of the pre-intervention readings of muscle activity and the post-intervention measures immediately and at the 10-minute interval following lumbar spine manipulation ($p>0.05$).

1.3.2 Alternative Hypotheses

1.3.2.1 Alternative Hypothesis One

There will be a significant difference between the pre-intervention readings of muscle activity and the post-intervention measures immediately and at the 10-minute interval following lumbar spine manipulation in asymptomatic participants ($p<0.05$).

1.3.2.2 Alternative Hypothesis Two

There will be a significant difference between the pre-intervention readings of muscle activity and the post-intervention measurements immediately and at the 10-minute interval following lumbar spine manipulation in symptomatic participants ($p<0.05$).

1.3.2.3 Alternative Hypothesis Three

There will be a significant difference between the asymptomatic and symptomatic groups in terms of the pre-intervention readings of muscle activity and the post-intervention measures immediately and at the 10-minute interval following lumbar spine manipulation ($p<0.05$).

1.4 Study Rationale

Spinal manipulative therapy is a form of manual intervention that is utilised to restore pain-free movement within fixated joints that are identified in the spine and the extremities of the body (Maigne and Vautravers 2003; Bergman and Peterson 2011). Fixated joints within the spine have been shown to cause changes in the activity in both segmentally-related muscles (Harvey and Descarreaux 2013) and extremity muscles (Christiansen *et al.* 2018), which reveals that fixations in the spine may not only lead to local neurophysiological changes but may also have an effect on global neurophysiology (Haavik and Murphy 2012). When assessing the effects of SMT on muscle activity, studies have indicated that SMT can normalise muscle activity and modulate cortical excitability (Herzog, Scheele and Conway 1999; DeVocht, Pickar and Wilder 2005; Niazi *et al.* 2015). However, there is a gap in the literature that defines the exact global neurophysiological mechanisms of SMT on the extremities.

Low back pain is considered to be one of the most common causes of work-related disabilities around the world (Balagué *et al.* 2012). There is an association between spinal pain and arthrogenic muscle inhibition in muscles of the spine (Lalanne, Lafond and Descarreaux 2009) and extremities (Niazi *et al.* 2015). Subjects that have undergone AMI often demonstrate the inability to fully activate affected musculature during voluntary contractions (Suter *et al.* 2000; Hillermann *et al.* 2006). Although AMI is a process that is normally identified in symptomatic subjects, several studies suggest that asymptomatic subjects that present with joint fixations undergo similar neurophysiological changes (Dunning and Rushton 2009; Grindstaff *et al.* 2009; Fryer and Pearce 2012). However, further research is needed to determine if the extent of the effects of AMI differs between symptomatic and asymptomatic subjects.

Spinal manipulative therapy has been shown to be an effective form of conservative treatment that has many clinical benefits such as decreasing pain and tenderness and improving range of motion (Liebenson 2007). The clinical benefits and uses for SMT have become increasingly apparent, but the understanding of the mechanism of these effects still remains unclear (Henderson 2012; Currie *et al.* 2016). Theories suggest that the mechanical stimulus applied during SMT may not only cause biomechanical changes but may result in an initiation of neurophysiological mechanisms that are responsible for these effects (Pickar 2002; Pickar and Bolton 2012). SMT may exert its physiological influence by the combination of the biomechanical and neurophysiological mechanisms. Despite the growing body of evidence supporting these theories, further research is required to obtain a clearer understanding of these mechanisms.

This study, therefore, aims to investigate the effects of lumbar SMT on the lower limb musculature, specifically the quadriceps femoris and hamstring muscle groups, in symptomatic and asymptomatic subjects. Studying the effects of SMT in both symptomatic and asymptomatic individuals will allow for further insight and comparison of the neurophysiological consequences that pain has on different structures of the body (Grindstaff *et al.* 2009; Currie *et al.* 2016). Testing the subjects under the same conditions and procedures will provide more extensive information on the extent of the effects of SMT to determine whether the pain is the discriminating factor of the results shown between the symptomatic and asymptomatic groups. This study aims at contributing to the body of evidence assessing the effects of SMT on joint dysfunction and AMI in lower limb musculature in asymptomatic and symptomatic participants.

1.5 Flow of Dissertation

The subsequent chapters will be structured as follows:

Chapter Two, the literature review, will provide an overview of the anatomy of the lumbar spine and relevant muscles, followed by an analysis of the current and past literature on spinal manipulative therapy.

Chapter Three details the methodology of the study which help achieve the aims and objectives. All study designs, measurements, methods and techniques are explained.

Chapter Four will provide the results of the study.

Chapter Five provides a discussion of the results in terms of the current literature.

Chapter Six will provide a conclusion along with any limitations related to the study and recommendations stemming from the investigation.

CHAPTER TWO: LITERATURE REVIEW

2.1 Introduction

This chapter will provide the reader with the anatomy of the lumbar spine and relevant muscles, overview of skeletal muscle and nervous system. This will be followed by a review of the literature related to SMT and the effects on muscle activity.

Spinal manipulative therapy is a common form of treatment that is widely used by several complementary health practitioners (Bronfort *et al.* 2012; Chaibi *et al.* 2017). SMT is described as a high-velocity, low-amplitude (HVLA) thrust that can be applied to the vertebral column to restore joint motion (Pickar 2002; Pickar and Bolton 2012). Although the clinical benefits of spinal manipulation have started to become apparent, the neurophysiological mechanisms underlying the SMT remain unclear in both symptomatic and asymptomatic individuals (Dunning and Rushton 2009; Chaibi *et al.* 2015).

2.2 Overview of the Lumbar Spine

The lumbar spine, which is located between the thoracic vertebrae and the sacrum, is made up of five articulating vertebrae (L1-L5) and is mainly responsible for providing mobility and aiding in stability by bearing an enormous amount of force that is produced during activity from the spine and upper extremities (Moore, Dalley and Agur 2013). The lumbar lordosis increases the amount of weight that the lumbar spine can withstand during activities such as walking, running, or jumping (Cramer and Darby 2017). There are several ligaments in the lumbar spine that serve to help accommodate movement and energy absorption. The majority of these ligaments that are present in the lumbar spine are often thicker and stronger than any other region of the spine due to the need for protection from excessive movement and stress (Cramer and Darby 2017).

The intervertebral foramen (IVF) which is formed by the intervertebral discs, the adjacent pedicles, laminae, and articular processes, allows for an opening to be formed for the passage of structures (i.e. spinal nerve roots) between the spinal canal and the periphery (Waxenbaum and Futterman 2019). In the lumbar region, the spinal cord

typically terminates at the level of L1-L2 which allows for a unique variation compared to the rest of the spinal column where the nerve root travels inferiorly and laterally to exit the desired IVF. For example, the L3 nerve root will exit through the IVF from the level of L3-L4. The lumbar spine is a region of high clinical significance as it has been shown that the size of IVFs decreases as the spinal cord descends whereas the size of the spinal nerve root increases. This may lead to an increased susceptibility of nerve root compression (Cramer and Darby 2017; Waxenbaum and Futterman 2019).

2.2.1 Lumbar Vertebrae

The lumbar vertebrae can be categorised in two different groups known as typical (L1-L4) and atypical (L5) vertebrae. The four typical lumbar vertebrae often share the following characteristics (Moore, Dalley and Agur 2013; Cramer and Darby 2017):

- The vertebral body normally has a large kidney-shaped structure that is wider laterally when compared to the anterior than to posterior surface. The vertebral bodies are convex from side to side anteriorly and concaved posteriorly which is said to aid in the weight-bearing function of the lumbar spine.

- The vertebral foramen is triangular in shape and is larger than the foramen in the thoracic region and smaller than in the cervical region.

- Transverse processes are long and thin with a posterolateral orientation. On the posterior surface of the transverse process is a small accessory process that functions as an attachment site for the intertransversarii muscles. This is a unique characteristic of the lumbar spine.

- The spinous processes are short, thick and sturdy, and are often hatchet shaped.

- The articular processes are thick and strong with the concave-shaped superior articular processes facing posteromedially and the convex-shaped inferior processes facing anterolaterally.

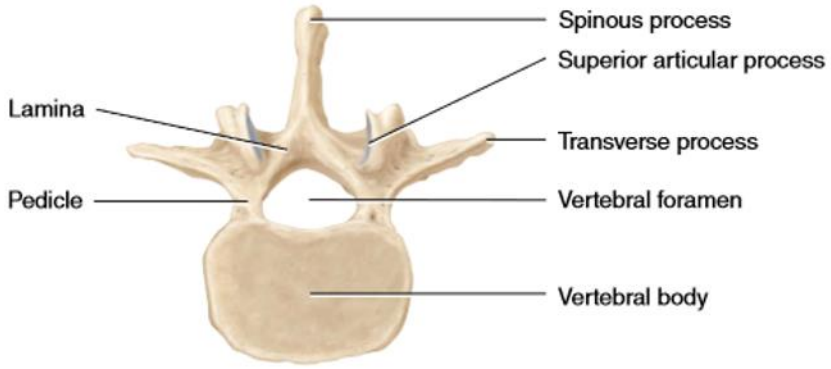

Figure 2.1: Superior view of typical lumbar vertebra (Tortora and Derrickson 2017)

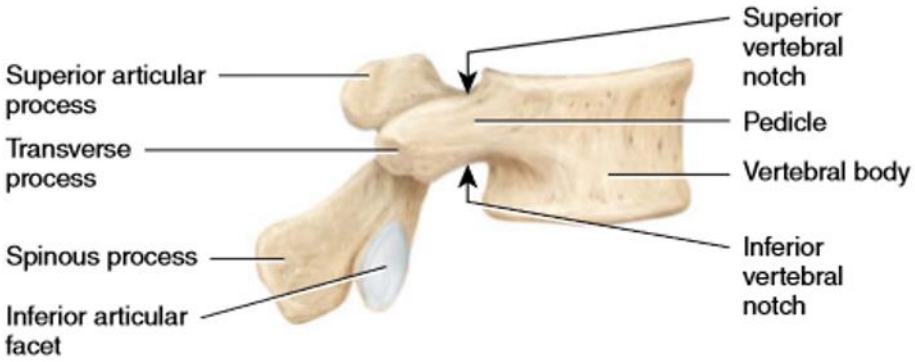

Figure 2.2: Right lateral view of typical lumbar vertebra (Tortora and Derrickson 2017)

The atypical lumbar vertebra (L5) possesses the following unique features (Bergman and Peterson 2011; Moore, Dalley and Agur 2013):

- The vertebral body has an elliptical shape rather than the kidney-shaped bodies of the above typical vertebrae. The anterior surface is markedly larger than the posterior surface which aids in the formation of the lumbar lordosis.
- The transverse processes are shorter and thicker.
- The spinous processes are shorter and more rounded.
- The articular processes differ from the above vertebrae as the superior processes of L5 are orientated more posteriorly than medially. The inferior articulating processes are situated further apart and are orientated in the coronal plane.

2.2.2 Articulations of the Lumbar Spine

2.2.2.1 Intervertebral Discs

The intervertebral discs (IVD) of the lumbar spine are thicker compared to any other region of the spine. As stated above, the lumbar spine is mainly responsible for absorbing force during axial loading which is aided by the increased thickness of the IVD in this region. The IVD is composed of a central nucleus pulposus, outer annulus fibrosis, and the cartilaginous vertebral endplates found on the superior and inferior vertebrae. The annulus fibrosis (AF) of the lumbar spine consists of fibrocartilaginous rings, or lamellae, that are closely organised collagen and elastic fibres (Tortora and Derrickson 2017; Ralston *et al.* 2018).

The most superficial lamellae of the AF attach directly to the adjacent vertebral body (above and below) to form an extremely strong anchor and has been shown to play a major role in both loadbearing and preventing extreme torsional stress. The outer third of the AF is the only area that receives sensory innervation. The nucleus pulposus is an avascular, gelatinous mass found in the centre of the IVD and receives its nourishment from the vessels found in the perimeter of the AF and the vertebral body. The IVD of the lumbar spine are often thicker anteriorly to help form and maintain the lumbar lordosis for optimal weight-bearing (Moore, Dalley and Agur 2013; Cramer and Darby 2017; Tortora and Derrickson 2017).

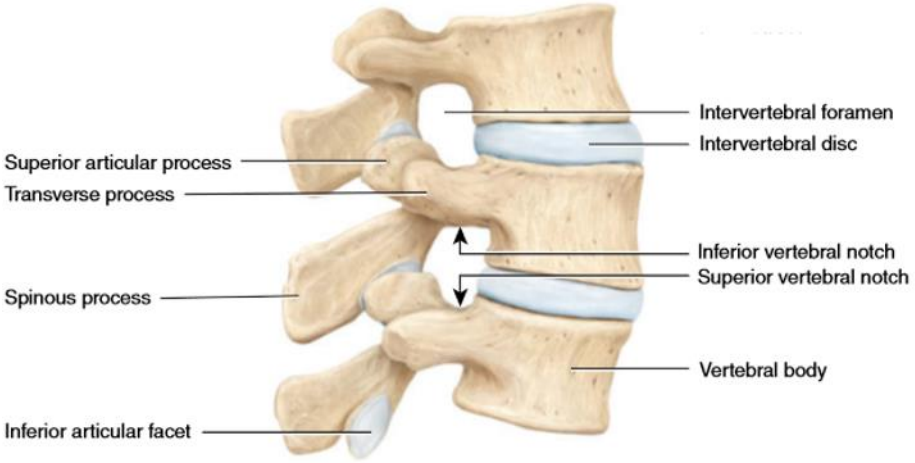

Figure 2.3 Right lateral view of articulating lumbar vertebrae (Tortora and Derrickson 2017)

2.2.2.2 Facet Joints

The facet joints, or alternatively known as the zygapophyseal joints, are formed by the articulation of the superior and inferior articular process of two adjacent vertebrae. These are complex synovial joints that are able to both encourage and limit movement dependant on the orientation of the joints. As the spine transitions from the thoracic to the lumbar region, the facet joints have a sagittal orientation but begin to become more coronally orientated as the spine descends to the lumbosacral junction. This facet joint arrangement allows for more flexion and extension movements to occur in the lumbar spine but adds a limitation to rotational flexibility. Each articular process is covered by a thin layer of hyaline cartilage with an articular capsule surrounding the posterolateral surface that is divided into an outer dense fibroelastic connective tissue, vascular central layer, and an inner synovial membrane layer. The articular capsule functions as a stabiliser to the zygapophyseal joints and provides torsional strength to the lumbar spine during movement (Cramer and Darby 2017; Saladin, Gan and Cushman 2018).

The facet joints have a rich sensory supply which originates from the medial branch of the posterior primary dorsal rami from the level of the joint as well as from the levels above and below. The medial branches that are responsible for the innervation of the zygapophyseal joint terminate as one of four types of sensory receptors known as free nerve endings, complex unencapsulated nerve endings, and capsulated nerve endings (Cramer and Darby 2017).

Wyke (1987) categorised the different types of sensory receptors according to their function:

- Type 1 – Extremely sensitive static and dynamic mechanoreceptors that are continuously stimulated even when the joint is not being moved.
- Type 2 – Less sensitive mechanoreceptors that are only activated during movement.
- Type 3 – Mechanoreceptors that are found in extremity joints. These are not present in the zygapophyseal joints.
- Type 4 – Slow conducting nociceptors.

2.2.3 Nerves relevant to the study

2.2.3.1 Femoral Nerve

The femoral nerve is made up of three converging anterior rami nerve roots from the lumbar spine (L2, L3, L4) and is considered to be the largest nerve within the lumbar plexus. Functions of the femoral nerve are of both motor and sensory innervation, the nerve supplies several muscles in the thigh for motor function and gives rise to branches that have cutaneous supply to the anteromedial thigh and medial surface of the leg (Bordoni and Varacallo 2018).

From the lumbar plexus, the femoral nerve passes inferiorly through the psoas major muscle of the posterior abdominal wall to enter the thigh. The femoral nerve then travels underneath the inguinal ligament where it enters the femoral triangle. Here the nerve will be positioned laterally to the femoral vessels (i.e. femoral vein, artery). Once passing beneath the inguinal ligament the nerve splits into two divisions known as the anterior and posterior division. The anterior gives rise to the anterior cutaneous branch and branches to the sartorius and pectineus muscles. The posterior division gives rise to saphenous nerve and, most importantly to this study, branches to the quadriceps femoris muscle group (Saladin, Gan and Cushman 2018).

The branch of the femoral nerve that is responsible for innervating the quadriceps femoris group is directed towards the muscle via its vasti muscles. The portion directed towards the vastus lateralis muscle will divide into two branches which further divide into another two branches travelling around the muscle from proximal anteriorly and distal posteriorly. The rectus femoris muscle receives innervation near its attachment by the anterior inferior iliac spine. The vastus medialis is innervated by the femoral nerve as it travels along its anteromedial border. The vastus intermedius receives innervation at the middle section of the muscle whereas the tensor portion is innervated proximally (Bordoni and Varacallo 2018; Saladin, Gan and Cushman 2018).

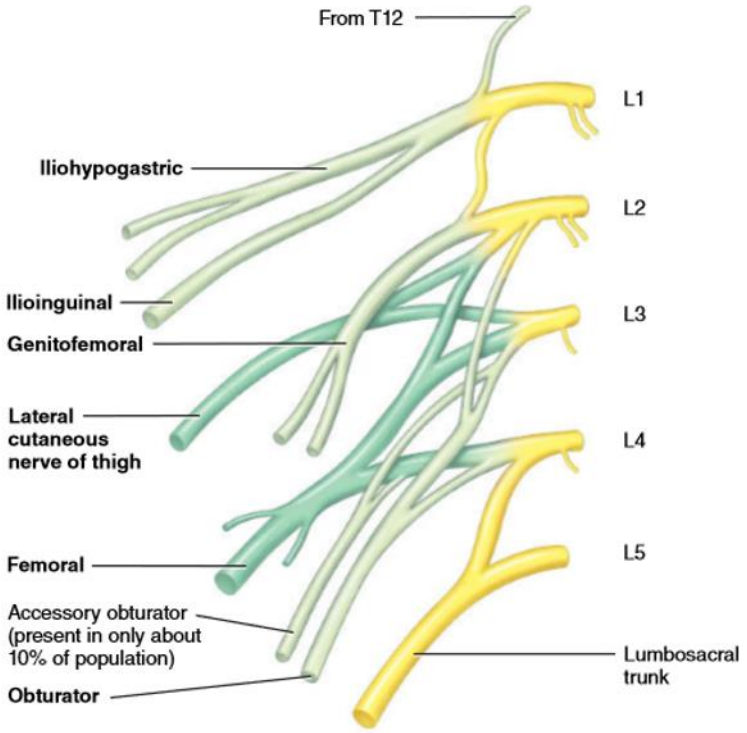

Figure 2.4: The lumbar plexus (Tortora and Derrickson 2017)

2.2.3.2 Sciatic Nerve

The sciatic nerve is considered to be the largest nerve within the human body and originates from the lumbosacral plexus and can be described as two separate nerve bundles, the tibial and common fibular nerves, which are bound together by a connective sheath. The sciatic nerve originates from both lumbar and sacral nerve roots (L4-S3) and has, similarly to the femoral nerve, both motor and sensory functions. Once formed, the sciatic nerve travels out of the pelvis to enter into the gluteal region via the greater sciatic foramen, where it emerges beneath the piriformis muscle. As the nerve passes through the gluteal region is passes by several muscles in order to enter the posterior thigh. As the nerve enters the posterior thigh, it gives rise to various branches that are responsible for innervating the adductor magnus and hamstring muscles. The long head of the biceps, semitendinosus and semimembranosus muscles are innervated by the tibial division of the sciatic nerve, whereas the short head of the biceps is innervated by the fibular component (Saladin, Gan and Cushman 2018; Stępień *et al.* 2019).

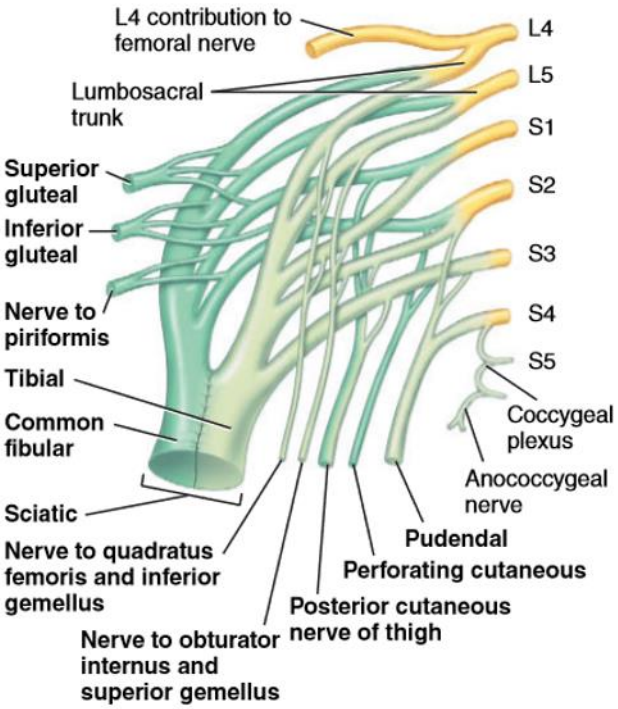

Figure 2.5: The lumbosacral plexus (Tortora and Derrickson 2017)

2.3 Overview of the Muscles

2.3.1 Quadriceps Femoris

The quadriceps femoris muscle is considered to be one of the largest and strongest muscles in the body and is located within the anterior compartment of the thigh. The quadriceps femoris is also known as a composite muscle as it consists of four separate muscles; the rectus femoris, vastus lateralis, vastus medialis, and vastus intermedius (Moore, Dalley and Agur 2013). These four muscles converge into a common tendon known as the quadriceps tendon that inserts into the patella and attaches inferiorly at the tibial tuberosity. The rectus femoris, which lies in between the vastus lateralis and vastus medialis, is the only muscle that crosses both the hip and knee joint as it attaches to the anterior inferior iliac spine of the pelvis and inserts into the patella tendon inferiorly. The three vasti muscles only cross the knee joint as they attached proximally to the femur shaft and join the rectus femoris with insertion into the patella tendon distally. Collectively these muscles allow for the controlled extension of the knee which is crucial in activities such as walking or running. Although knee extension

is the primary function of the quadriceps, it has been shown that it aids in hip flexion, specifically the rectus femoris muscle, and becomes a knee fixator by contracting eccentrically when performing knee-bent activities such as downhill walking or squats. The quadriceps femoris muscle is innervated by the femoral nerve (Tortora and Derrickson 2017).

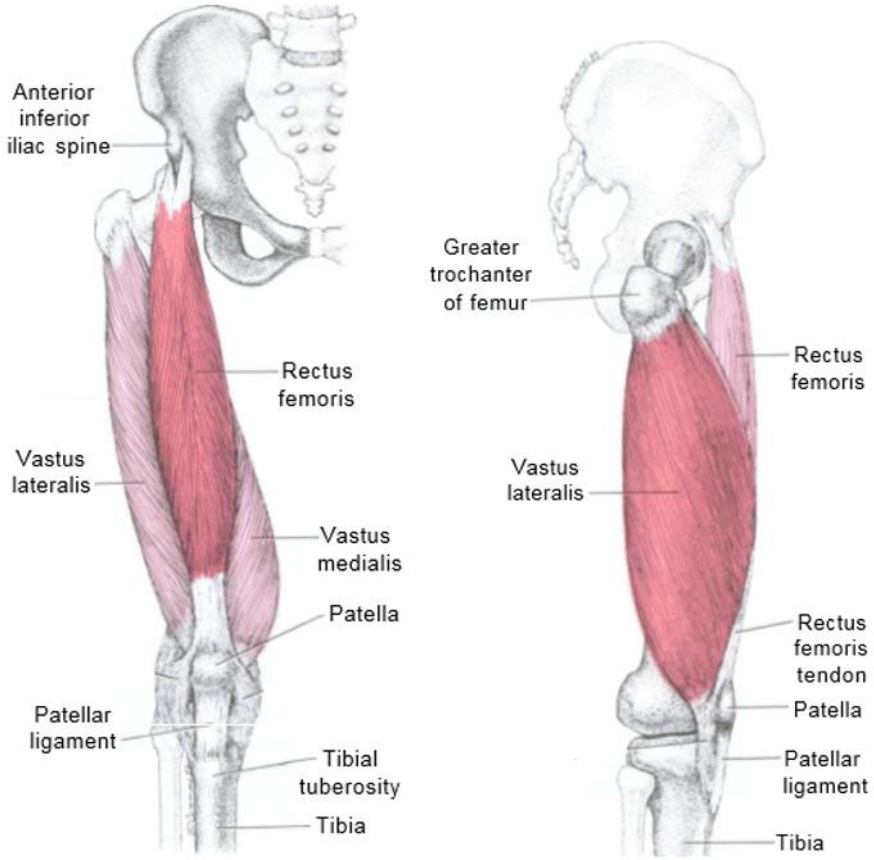

Figure 2.6: Anterior and lateral view of the quadriceps femoris (Donnelly *et al.* 2019)

2.3.2 Hamstring

The hamstring muscle, like the quadriceps, is made up of a collection of three separate muscles; the biceps femoris, the semitendinosus, and semimembranosus. These three muscles are known as two-joint muscles as they cross both the hip and knee joint with attachments proximally onto the ischial tuberosity of the pelvis and distally onto the lateral surfaces of the fibula and tibia. However, the biceps femoris, located on the

lateral side of the hamstring, is slightly unique in that it has both a long head and a short head. The short head component of the biceps femoris is the only part of the muscle that does not cross two joints as it attaches proximally to the posterior surface of the femur and crosses over the knee joint to attach to the lateral surface of the fibula distally. Although the hamstring muscle group is seen to be much weaker than the quadriceps, these muscles are responsible for flexion at the knee and aid in the extension of the thigh as their attachments span over both the knee and hip joint. The innervation of this muscle comes from the divisions of the sciatic nerve (Martini, Tallitsch and Nath 2017; Tortora and Derrickson 2017).

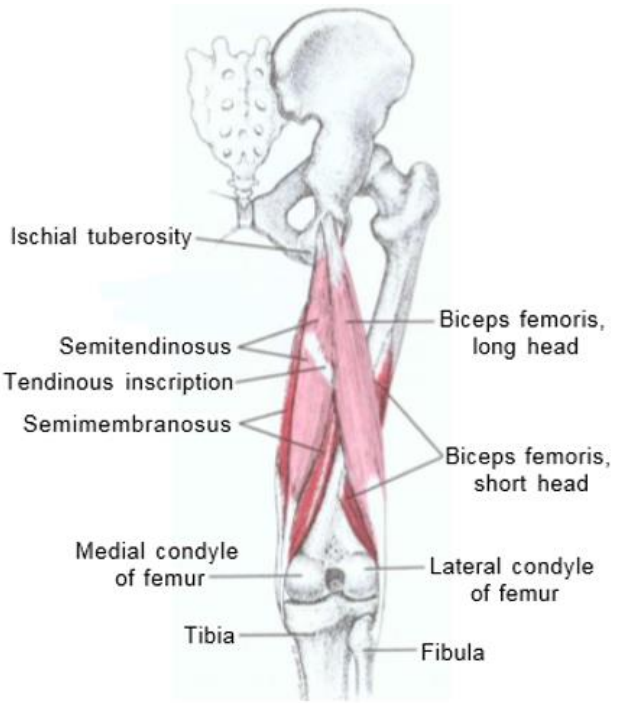

Figure 2.7: Posterior view of the hamstring (Donnelly *et al.* 2019)

2.4 Overview of the Nervous System

The nervous system, which is the smallest organ system within the human body, is a complex structure that comprises billions of interconnected neurons and neuroglia that helps the body adjust and react to internal and external or environmental changes. It allows for communication of signals between the brain and the rest of the body by

acting as the body's 'electrical system' by sending afferent information to the brain and efferent information to the desired location in the body. Structurally, the nervous system can be categorised into two groups; the central nervous system (CNS) and the peripheral nervous system (PNS) (Saladin, Gan and Cushman 2018).

Collectively, the nervous system has three main functions; sensory function to detect both internal and external stimuli, integrative function to analyse the acquired sensory information and make an appropriate response, and motor function by eliciting the response after the sensory information is integrated (Cramer and Darby 2017; Martini, Tallitsch and Nath 2017; Tortora and Derrickson 2017).

2.4.1 Central Nervous System

The central nervous system (CNS) consists of the brain, which sits within the skull, and the spinal cord which is located within the vertebral canal or foramen of the vertebral column. The brain is responsible for processing all the information that it receives from sensory receptors found around the human body. Once this information has been processed and the appropriate response is formed, the CNS will send the command signal down the spinal cord. The spinal cord acts as a pathway for command signals and allows for a connection to be formed between the CNS and PNS. The information that is exchanged between the brain and the body travels up and down the spinal cord in tracts or pathways (Mtui, Gruener and Dockery 2015; Splittgerber 2018).

Within the spinal cord, two areas can be easily defined known as the gray and white matter. White matter is divided by the gray matter and is organised into three areas known as the ventral, dorsal and lateral white columns. Spinal cord tracts, also known as fasciculi, are found in these columns and are bundled together by having a common origin or course and function in terms of relaying similar information (Siegel and Sapru 2015; Splittgerber 2018). Within these ventral, dorsal or lateral columns there may be ascending or descending tracts. Ascending tracts are mainly responsible for relaying sensory information from sensory nerve endings up to the brain. The descending tracts, which originate from higher centres in the brain, have a primary function of relaying forms of motor information from the higher centres to the spinal cord (Cramer and Darby 2017). The spinal cord tracts that are relevant to this study will be discussed below.

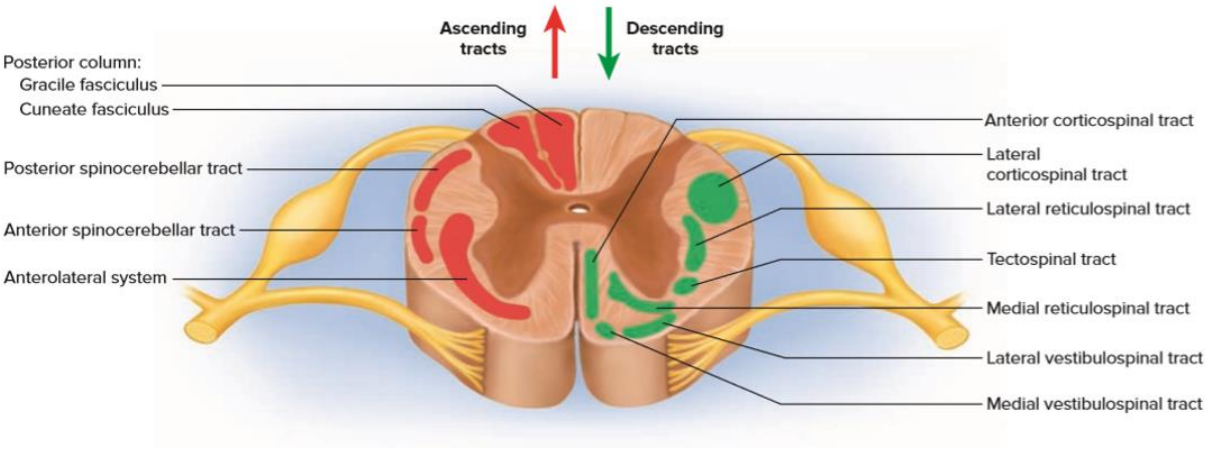

Figure 2.8: Tracts of the spinal cord (Saladin, Gan and Cushman 2018)

2.4.1.1 Corticospinal Tract

The corticospinal tract, which originates from the cortex or the brainstem, functions as a descending tract and is considered to be the largest voluntary motor pathway that controls movement in the trunk and extremity muscles. About 40% of the fibres in this tract originate from the primary motor cortex in the precentral gyrus, but other sources may include the lateral premotor cortex, the parietal lobe, somatic sensory cortex, and the cingulate gyrus. Fibres located on the medial side of the precentral gyrus have been shown to control over the foot and leg movements, whereas laterally located fibres control trunk, arms, hand, face, and tongue (Mtui, Gruener and Dockery 2015; Welniarz, Dusart and Roze 2017). From the cortex, the neurons converge and travel inferiorly through the internal capsule, the crus cerebri of the midbrain, the pons, and medulla. At the most inferior portion of the medulla, the tract divides into separate entities known as the anterior and lateral corticospinal tract. At the point of separation, the lateral tract decussates (crosses over to the other side of the spinal cord) and travels down the spinal cord terminating at the ventral horn of each individual segment. From the ventral horn, the lower motor neurons supply efferent impulses to the muscles of the body. Unlike the lateral corticospinal tract, the anterior tract does not decussate but rather remains ipsilateral. This tract then descends inferiorly down the spinal cord and decussates at the desired level and terminates in the ventral horn. The anterior corticospinal tract ends in the midthoracic level (Welniarz, Dusart and Roze 2017; Splittgerber 2018).

2.4.1.2 Reticulospinal Tract

The reticulospinal is considered to be an extrapyramidal tract as it originates from the brainstem and carries motor fibres into the spinal cord. Similarly, to the corticospinal tracts, the reticulospinal tract divides into two separate tracts, the lateral and medial reticulospinal tract. The lateral reticulospinal tract, which forms in the medulla, functions to inhibit voluntary movement and reduce muscle tone. In contrast, the medial reticulospinal tract, which forms in the pons, is responsible for facilitating voluntary movement and increases muscle tone (Crossman and Neary 2015; Splittgerber 2018).

2.4.2 Peripheral Nervous System

The peripheral nervous system (PNS) is made up of all the peripheral nerves and nerve tissue that is located beyond the CNS, this may consist of the motor and sensory nerves, peripheral nerve trunks, plexuses, and ganglia. All of these structures are responsible for linking the body with the CNS (Crossman and Neary 2015). The PNS consists of two forms of nerves; cranial nerves and spinal nerves. Each nerve has a specific path and function within the body. The PNS is divided into two systems; the somatic and autonomic systems.

There are 31 pairs of spinal nerves that create a path of communication between the spinal cord and specific areas and structures of the body such as the skin, muscles, joints, glands, and viscera. Each pair of the spinal nerves is given a name by the region and level of the vertebral column segment which they emerge. Initially, the spinal nerve roots emerge from the spinal cord and join to form the dorsal and ventral nerve roots. The dorsal root is responsible for conducting sensory information to the CNS from sensory receptors found in the skin, muscles and internal organs. Each dorsal nerve root only contains sensory axons and posterior swelling known as the dorsal root ganglion, which is responsible for holding the cell bodies of the sensory neurons. The ventral nerve root functions as a pathway for efferent impulses from the CNS to the muscles and glands. These roots only contain axons of motor neurons (Tortora and Derrickson 2017; Splittgerber 2018).

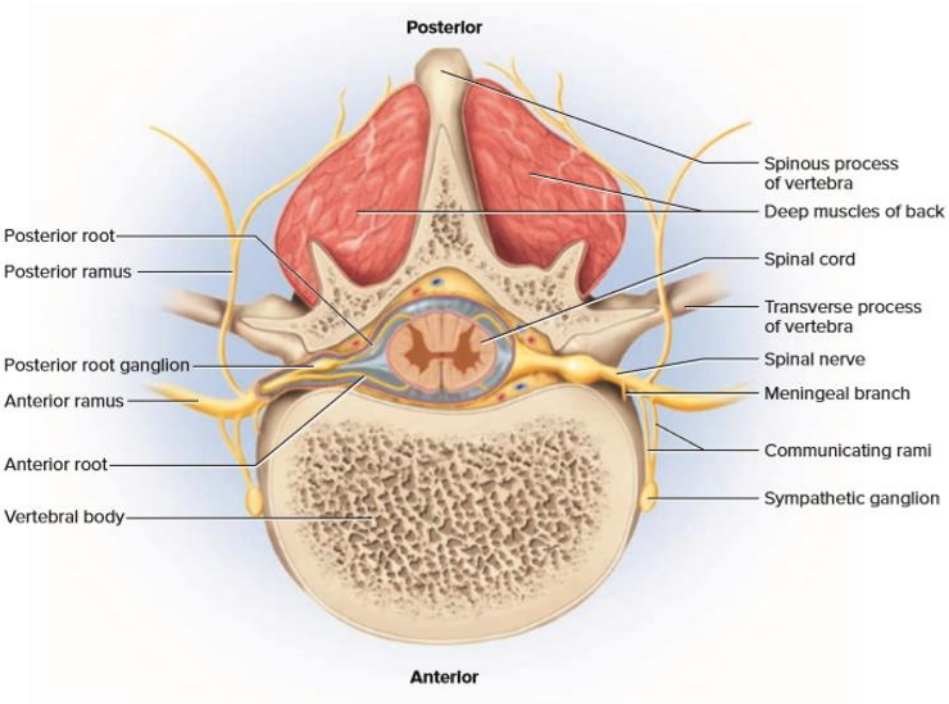

Figure 2.9: Details of the spinal nerve (Saladin, Gan and Cushman 2018)

The dorsal and ventral nerve roots merge, just proximal to the intervertebral foramen, to form the spinal nerve. Once the spinal nerve exits the intervertebral foramen (IVF) it divides into several branches known as the dorsal ramus, ventral ramus, and the meningeal branch. The dorsal ramus functions as innervation to the deep muscles and skin to the posterior surface of the trunk. The ventral ramus serves innervation to the muscles and structures of the upper and lower limbs as well as the skin over the lateral and anterior surfaces of the trunk. The meningeal branch, which differs slightly compared to the dorsal and ventral rami, re-enters into the vertebral cavity via the IVF to supply the vertebrae, vertebral ligaments, blood vessels supplying the spinal cord and the meninges (Siegel and Sapru 2015; Tortora and Derrickson 2017).

The PNS has nerve fibres that can be divided into sensory neurons, motor neurons, and interneurons. The interneurons function as an analyser of sensory input and a co-ordinator of motor output. The number of interneurons involved depends on how intricate and complex the response to stimulus needs be, the more complex the response, the increase in the number of interneurons used. These interneurons are classified as inhibitory or excitatory. Sensory neurons are responsible for delivering information about the external and internal environment to the CNS and are also known

as afferent neurons. These afferent fibres are located between the sensory receptor and the CNS (Siegel and Sapru 2015; Martini, Tallitsch and Nath 2017).

Table 2.1: Classification of sensory nerve fibres

Type	Size (μm)	Conduction Velocity (m/s)	Characteristics	Receptor
Ia	13 – 20	80 – 120	Responds and detects muscle length and any rate of change in length	Muscle spindle.
Ib	13 – 20	80 – 120	Responds to changes in muscle tension	Golgi tendon organ.
II	6 – 13	35 – 75	Stretch receptor, non-adapting	Cutaneous mechanoreceptors, secondary receptors to muscle spindles.
III	1 – 5	3 – 35	Responds to pain	Follicular endings (touch and pressure), nociceptor (fast pain) and thermal receptors.
IV	0.2 – 1.5	0.5 – 2	Responds to pain	Nociceptor (slow pain), itch, touch receptors (warmth).

(Adapted from Pickar (2002), Cramer and Darby (2017), and Ralston *et al.* (2018)).

The motor neurons, forming the efferent division of the PNS, are responsible for carrying information from the CNS to stimulate or activate peripheral tissue, organs and organ systems. The axons of motor neurons are myelinated and are large in diameter which ensures that action potentials are able to propagate at high velocities to reach their destination (i.e. skeletal muscle) with minimal delay. The cell body of the motor neuron is located inside the CNS, specifically in the medulla oblongata or the grey matter of the spinal cord, and has axons that extend to the effector organ. Once the axon reaches the effector organ it forms a neuromuscular junction (NMJ) with the muscle (Martini, Tallitsch and Nath 2017; Saladin, Gan and Cushman 2018).

Table 2.2: Classification of motor nerve fibres

Type	Size (µm)	Conduction (m/s)	Function
Alpha (Largest and fastest)	8 – 20	35 – 120	Innervates slow and fast twitch fibres of the extrafusal muscle fibres
Beta (Medium)	6 – 12	35 – 70	Innervation of skeletal muscle and muscle spindles
Gamma (Smallest and slowest)	2 – 8	10 – 50	Innervate intrafusal fibres of muscle spindles

(Adapted from Mtui, Gruener and Dockery (2015) and Cramer and Darby (2017)).

2.4.2.1 Sensory Receptors

Sensory receptors gather information about the body both internally and externally. This information is then delivered to the CNS where it is processed in order to execute the desired response. Sensory receptors are classified based on the stimulus that they respond to. These groups are known as mechanoreceptors, thermoreceptors, photoreceptors, chemoreceptors, and nociceptors (Tortora and Derrickson 2017).

Table 2.3: Summary of sensory receptors

Receptor Type		Location	Sensations	Adaptations
Mechanoreceptors	Merkel's disc	Epidermis	Touch, pressure and texture	Slow
	Hair follicle receptors	Hair follicles	Motion, direction	Rapid
	Type 1: Ruffini endings	Dermis of the skin, ligaments and tendons	Skin stretch, joint position and movement	Slow
	Type 2: Pacinian corpuscles	Deep within the dermis and ligaments & tendons, periosteum and superficial layer of the joint capsule. Stretching	Fast vibrations, deep pressure, joint position and movement	Rapid
	Meissner's corpuscles	Dermal papillae of hairless skin	Fine or discriminative touch, pressure and slow vibrations	Rapid
	Muscle spindles	Most striated skeletal muscle	Muscle length	Slow
	Type 3: Golgi tendon organs	Ligaments and tendons	Muscle tension, joint position and movement	Slow

(Adapted from Mtui, Gruener and Dockery (2015) and Cramer and Darby (2017)).

2.5 Physiology of Muscles

The human body contains three types of muscle tissue: skeletal muscle, cardiac muscle, and smooth muscle. Skeletal muscle is responsible for voluntarily moving bone, cardiac muscle forms the majority of the heart wall and adjacent vessels (i.e. aorta) and allows for an involuntary contraction and relaxation of the heart, and smooth muscle forms structures such as the blood vessels, airways and other internal organs (Tortora and Derrickson 2017).

Although these types of muscle tissues differ in their composition and location, according to Martini, Tallitsch and Nath (2017), they all share these four properties:

- Excitability: the ability of the muscle to respond to various stimuli. Skeletal muscle will respond to stimulation sent from the nervous system, whereas smooth muscle will respond to circulating hormones.

- Contractility: the ability of the muscle to contract in response to stimulation.

- Extensibility: the ability of the muscle to stretch without sustaining any damage. The connective tissue is responsible for keeping the muscle within its contractile limits.

- Elasticity: the ability of the muscle tissue to return to its original length once it has undergone contraction or extension.

Skeletal muscle is considered to be one of the largest organs that accounts for nearly 50% of the total body mass. The majority of the skeletal muscle attaches directly to bone by tendons, a tough connective tissue made up of many organised collagen fibres, but some muscles attach directly to skin (i.e. facial muscles) or to other muscles to carry out their function (Donnelly *et al.* 2019). According to Martini, Tallitsch and Nath (2017), skeletal muscles provide the following functions:

- Produce movement: this is the primary function of skeletal muscle. Once the contraction takes place it will exert a pull on bone to achieve gross movement such as walking or running or specific movement when grasping an object.

- Maintain posture: certain muscles are constantly being activated subconsciously to allow and maintain an upright posture against gravity.

- Protection: in the abdominal region of the body the strong abdominal and pelvic floor muscles provide protection to internal organs.

- Vascular pump: contractions of skeletal muscle can promote movement of fluid in the lymph and venous systems.

- Maintain body temperature (thermogenesis): during muscle contraction body heat is produced, this helps with maintaining normal body temperature.

2.5.1 Morphology

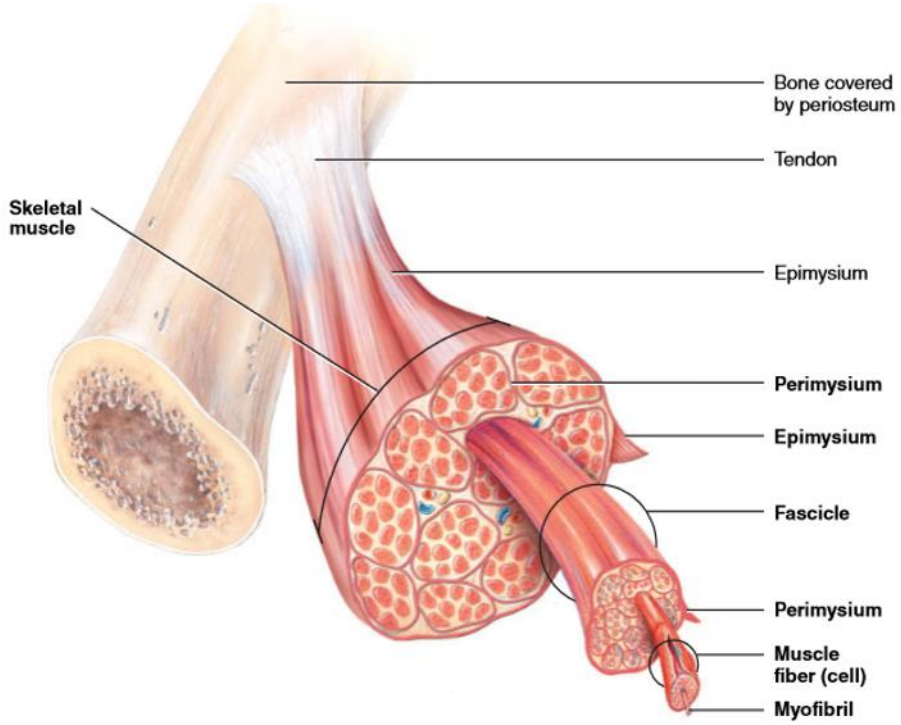

Figure 2.10: Components of skeletal muscle (Tortora and Derrickson 2017)

As depicted above in figure 2.10, at gross appearance the skeletal muscle is composed of many organised, elongated muscle fibres that are grouped together to form fascicles. These muscle fibres are individually wrapped in a connective tissue known as the endomysium, which not only helps bind neighbouring fibers to each other but also supports the capillaries that are distributed within the fascicle in order to allow for sufficient blood supply to each individual fibre. Similarly, to the muscle fibres, the fascicles bind together to form the bulk of the muscle, with each of these fascicles being surrounded by a connective tissue made up of collagen and elastic fibers known as perimysium (Cael 2010; Martini, Tallitsch and Nath 2017).

There is a high distribution of the blood vessels and nerves within the perimysium to supply each fascicle. The epimysium is the outermost layer of each muscle that is composed of dense irregular connective tissue. The epimysium is not only responsible

for encasing the muscle but provides separation from other organs. The collagen fibres from the endomysium, perimysium, and epimysium extend past the muscle and converge to form a tendon. As the tendon attaches to the bone it forms an extremely strong union with the periosteum and matrix of that particular bone, so when the muscle actively contracts it is able to pull on the bone and produce movement (Cael 2010; Martini, Tallitsch and Nath 2017; Tortora and Derrickson 2017).

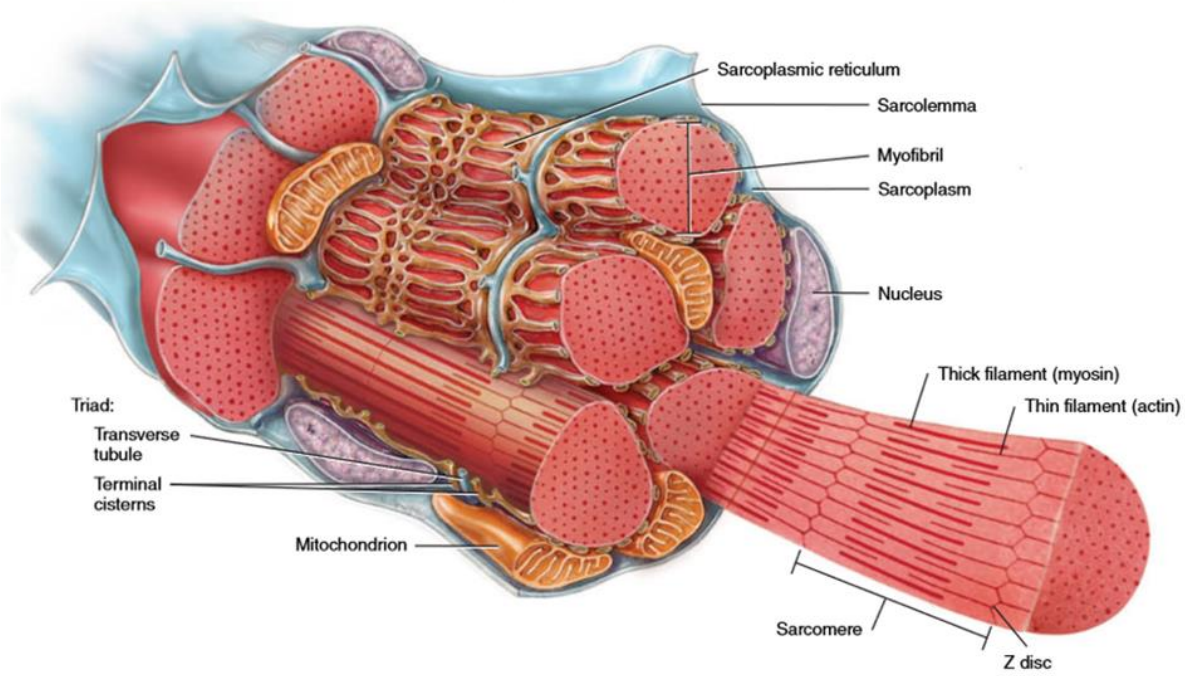

Figure 2.11: Details of a skeletal muscle fibre (Tortora and Derrickson 2017)

At a microscopic level, the most important component of the skeletal muscle is the muscle fibre. Muscle fibres differ in comparison to other cells of the body as they are much larger, a single muscle fibre's length can be equivalent to the length of the whole muscle (30-40 cm). These fibres appear as multinucleated as they develop from the fusion of groups of embryonic cells known as myoblasts. Some myoblasts do not fuse but remain within the muscle tissue to form myosatellite cells which are responsible for regenerating and repairing muscle tissue after injury (Tortora and Derrickson 2017). The sarcolemma is a transparent sheath that envelopes each individual muscle fibre and forms the plasma membrane which acts as the conduction site for action

potentials. Within the sarcolemma is the sarcoplasm, this is normally known as the cytoplasm in other cells, which is filled with significant amounts of glycogen, myoglobin, and mitochondria. Along the surface of the sarcolemma are indentations that form a deep network known as transverse (T) tubules. These T tubules form a network from the outer surface to the centre of each fibre which allows muscle action potentials to not only excite or stimulate the surface but rather ensures that the entire muscle fibre is stimulated (Cohen and Hull 2016).

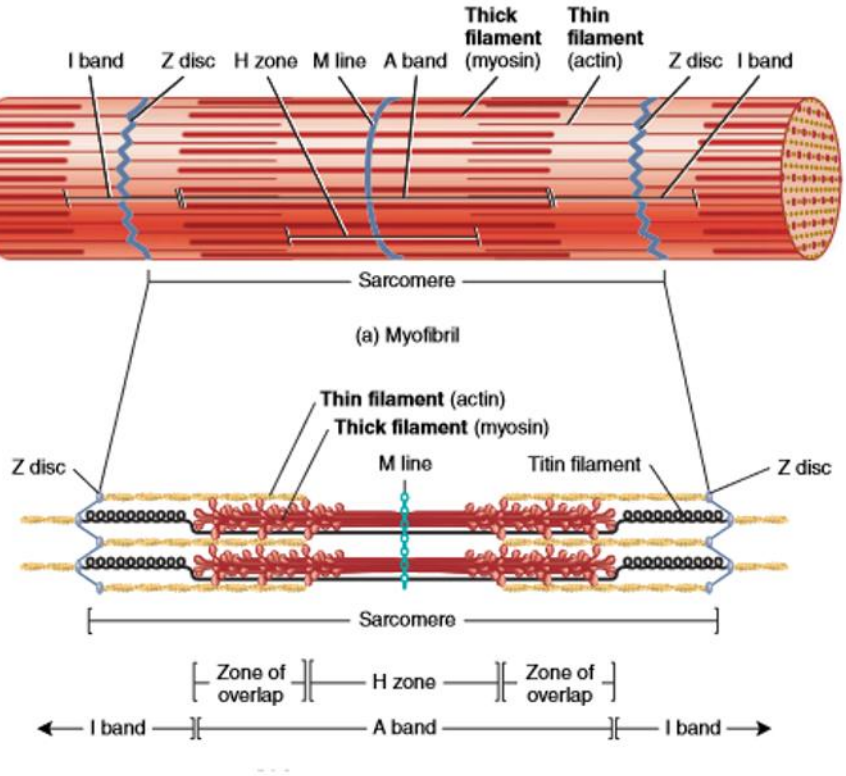

Figure 2.12: Details of a sarcomere (Tortora and Derrickson 2017)

The sarcoplasm is filled with small cylindrical structures known as myofibrils. Myofibrils are the contractile organelles of skeletal muscle and are composed of protein filaments, or rather myofilaments, that can be described as thick or thin. Thick filaments are composed of myosin whereas thin filaments are from a protein known as actin and are arranged in such a manner that there are two thin filaments for every one thick filament (Cohen and Hull 2016). These filaments do extend throughout the entire

muscle fibre but are rather organised into compartments known as sarcomeres. Each sarcomere is responsible for attributing to the unique striated appearance of skeletal muscle due to alternating light (I) bands and dark (A) bands.

The dark A bands are situated in the middle of the sarcomere and extend along the entire length of the thick filament. At the end of A band, there is an area known as the zone of overlap where the A bands overlap with the light I bands and the thin filaments are found in between the thick filaments. Passing through the centre of the I bands is a dense protein known as a Z line, these separate one sarcomere to the next, therefore each sarcomere extends from Z line to Z line. Surrounding each individual myofibril is a membrane complex known as the sarcoplasmic reticulum. The sarcoplasmic reticulum (SR) is responsible for storing and releasing calcium from its sac-like membrane, therefore it plays a huge role in determining whether a myofibril contraction will take place. On either side of a T tubule, the SR undergoes an expansion and fuses to form chambers known as terminal cisternae. A pair of terminal cisternae combined with a T tubule is known as a triad (Cael 2010; Tortora and Derrickson 2017).

Myofibrils are composed of the following three types of proteins (Tortora and Derrickson 2017):

- Contractile proteins, which aid in generating a force during contractions.
- Regulatory proteins, which aid in activating or deactivating the contraction process.
- Structural proteins, which enable the myofibril to have sufficient elasticity and extensibility, hold the thick and thin filaments in proper alignment and create a connection of the myofibrils to the sarcolemma and extracellular matrix.

The two contractile proteins are actin or myosin or also known as the thin and thick filaments respectively. Myosin is the main component of the thick filament and considered to be a motor protein. Motor proteins are responsible for converting chemical energy into mechanical energy of motion to produce force in order to achieve movement. Each myosin molecule twists together with the tails pointing towards the M line in the centre of the sarcomere. All of the myosin tails lie parallel to each other to form the shaft of the thick filament. At the opposite end of the molecule are two globular

heads which act as a binding site, one for actin and one for adenosine triphosphate (ATP). The binding site specifically for the ATP also acts as an enzyme that splits the ATP for energy during muscle contraction. The main component of the thin filament is actin (Tortora and Derrickson 2017; Saladin, Gan and Cushman 2018).

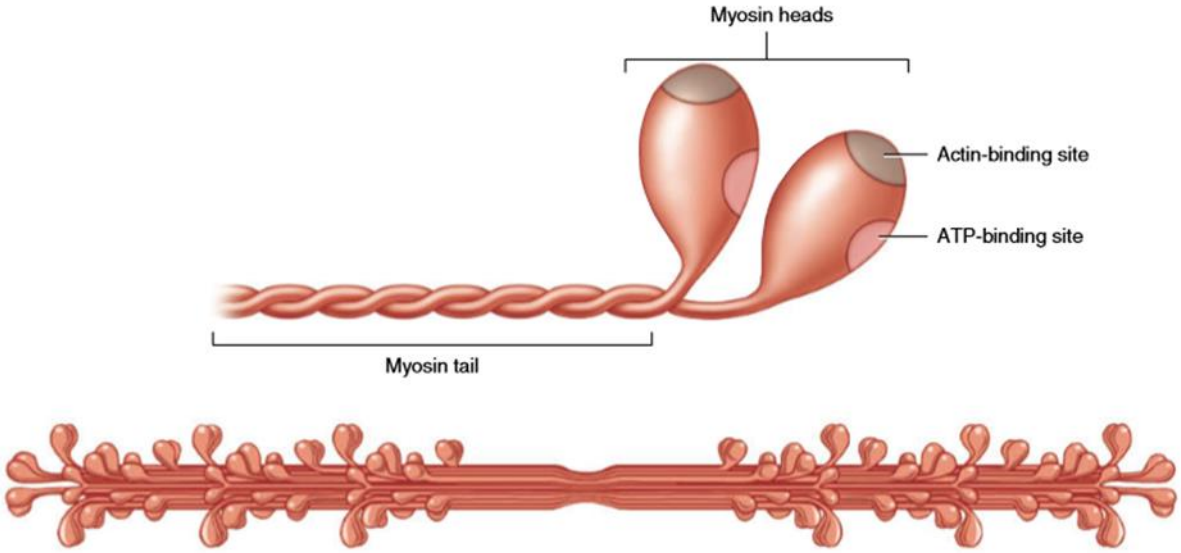

Figure 2.13: Details of a myosin molecule and a thick filament (Tortora and Derrickson 2017)

Actin molecules bind together and twist into a helix-shaped structure. On each actin molecule, there is myosin-binding site for the attachment of the myosin head. On the surface of the actin molecule is the presence of two regulatory proteins known as tropomyosin, which prevents the binding of the myosin to the actin when the muscle is in a relaxed state because the tropomyosin covers the myosin binding site, and troponin a protein that is responsible for holding the tropomyosin in place unless calcium binds to the troponin molecule which causes the molecule to change shape and pull tropomyosin away from the myosin-binding site. Subsequently, this will lead to the binding of the myosin onto the actin allowing for a contraction to take place (Cohen and Hull 2016; Saladin, Gan and Cushman 2018).

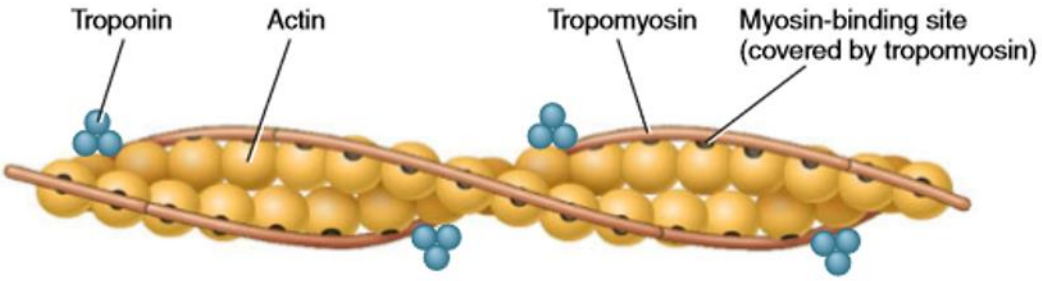

Figure 2.14: Details of a thin filament (Tortora and Derrickson 2017)

2.5.2 Motor Unit

A motor unit comprises a motor neuron and the group of muscle fibres that it innervates. Each muscle may have a number of motor units where the muscle fibres belonging to each motor unit may be dispersed and overlap with other muscle fibres from other units, rather than being grouped together. Depending on the size of the muscle, fibres from a motor unit may spread to only a part or through to the entire muscle (Cramer and Darby 2017).

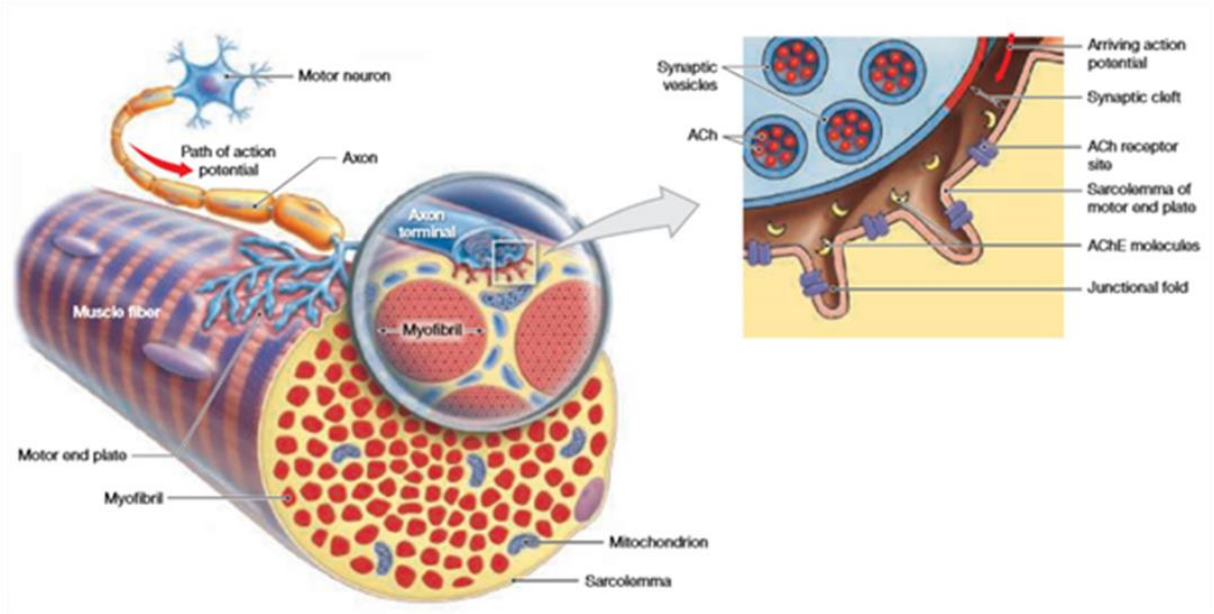

Figure 2.15: Details of a neuromuscular junction (Martini, Tallitsch and Nath 2017)

2.5.3 Skeletal Muscle Contraction

Once movement is required, the CNS sends impulses to the specific motor neuron needed to initiate the contraction process in the desired muscle. These impulses often result in some form of contraction or movement and are therefore known as a motor impulse and the nerves that carry these impulses - motor neurons. The neuromuscular junction (NMJ) is the area at which the nerve attaches to the muscle fibre (Cramer and Darby 2017).

As depicted in figure 2.15, within the cytoplasm of axon terminal there are synaptic vesicles (mitochondria and small secretory vesicles) that contain a substance, or rather a neurotransmitter called acetylcholine (ACh). A space known as the synaptic cleft separates the axon terminal from the motor endplate of the desired skeletal muscle fibre, therefore a neurotransmitter, in this case, ACh is required to allow the motor neuron to communicate with surrounding cells. When a motor impulse arrives at the axon terminal, ACh is released into the synaptic cleft where it interacts and attaches to the receptors of the sarcolemma on the motor endplate or it interacts with an enzyme known as acetylcholinesterase (AChE) which is responsible for splitting the ACh molecule and preventing the neurotransmitter from stimulating the endplate. However, once the ACh binds to the receptors, it causes a change in the membrane potential causing an action potential to stimulate the sarcolemma and deep network of the T tubules (Martini, Tallitsch and Nath 2017).

This stimulation causes the lateral chambers of the SR to release stored calcium ions into the sarcoplasm and around the sarcomeres. The calcium ions bind to troponin which produces a change in the troponin-tropomyosin complex causing the troponin to remove the tropomyosin and uncover the myosin-binding sites on the thin filament. Myosin cross-bridges form as the globular heads of the myosin migrates from the sides of the thick filament to bind to the active sites of the thin filament. With the use of stored energy, the myosin pulls the actin filaments together within the sarcomeres which results in shortening of the cell. Regenerated ATP is used to detach the myosin and allow for them to return to their original position, this is known as the ‘power stroke’. Muscle contraction will continue until this cycle has been broken by either ACh diffusing out of the synapse or the action of the AChE breaking down the ACh and preventing any further development of action potentials. As the cycle is halted, the SR reabsorbs

the calcium ions which depletes the calcium concentration within the sarcoplasm allowing the troponin-tropomyosin complex to return to its normal position, thus preventing myosin from binding onto the actin filament and stopping further contractions (Martini, Tallitsch and Nath 2017; Tortora and Derrickson 2017).

2.5.4 Motor Unit Recruitment

In movements where fine control is required (i.e. intrinsic muscles of the hand) small motor units may only innervate a few muscle fibres. However, when fine control is unnecessary during movements, such as knee flexion, a large motor unit may innervate a large number of muscle fibres. When a motor neuron is activated it will cause an activation of the muscle fibres that it innervates causing a small contraction of that muscle. For there to be a stronger contraction within the desired muscle more motor units need to be activated. Motor unit recruitment is the measure of how many motor neurons and muscle fibres are being activated in a specific muscle. Therefore, the higher the motor unit recruitment the larger the contraction of that muscle (Splittgerber 2018).

In the ventral horn of the spinal cord, there are three different forms of motor neurons. Motor neurons that are specific to skeletal muscle are known as alpha motor neurons and can be separated into fast, slow-twitch and intermediate fibers. Large fibres can cause rapid contraction and relaxation, they are responsible for producing large forces but fatigue after a short period of time. Slow-twitch fibres contract slowly and precisely to produce small contractile forces and are highly resistant to fatigue. Intermediate fibres share properties from both the fast and slow fibres, these fibres can produce a moderate contraction force while being moderately fatigue resistant. Each skeletal muscle has all three types of fibres, but the proportion of the motor units for each muscle may differ due to the function of that specific muscle. The other two motor neurons are known as the beta motor neuron, which innervates skeletal muscle as well as muscle spindles, and the gamma motor neuron which can be further segregated into static and dynamic types that are responsible for the innervation of the contractile portion of neuromuscular spindles. These motor neurons are mixed to form motor neuron pools, with a motor neuron pool innervating one particular muscle (Mtui, Gruener and Dockery 2015; Cramer and Darby 2017).

Stimulating entire motor neuron pools compared to each motor neuron allows for the central nervous system (CNS) to easily control and regulate movements. Firstly, the motor units are stimulated in a specific order from the weakest to the strongest. A weak input would initially stimulate the small, slow fibres which would generate a small constant contraction within the muscle. As the input increases the larger fibres will be stimulated leading to an increase in the force of contraction. Secondly, the CNS alters the frequency of the input to help modulate the muscle force. As the CNS increases firing to the motor neuron pool, consecutive twitches can summate effectively causing contraction within that muscle. The motor neuron pools receive both inhibitory and excitatory input from adjacent interneurons (Cramer and Darby 2017; Splittgerber 2018).

2.5.5 The role of Golgi Tendon Organs and Muscle Spindles

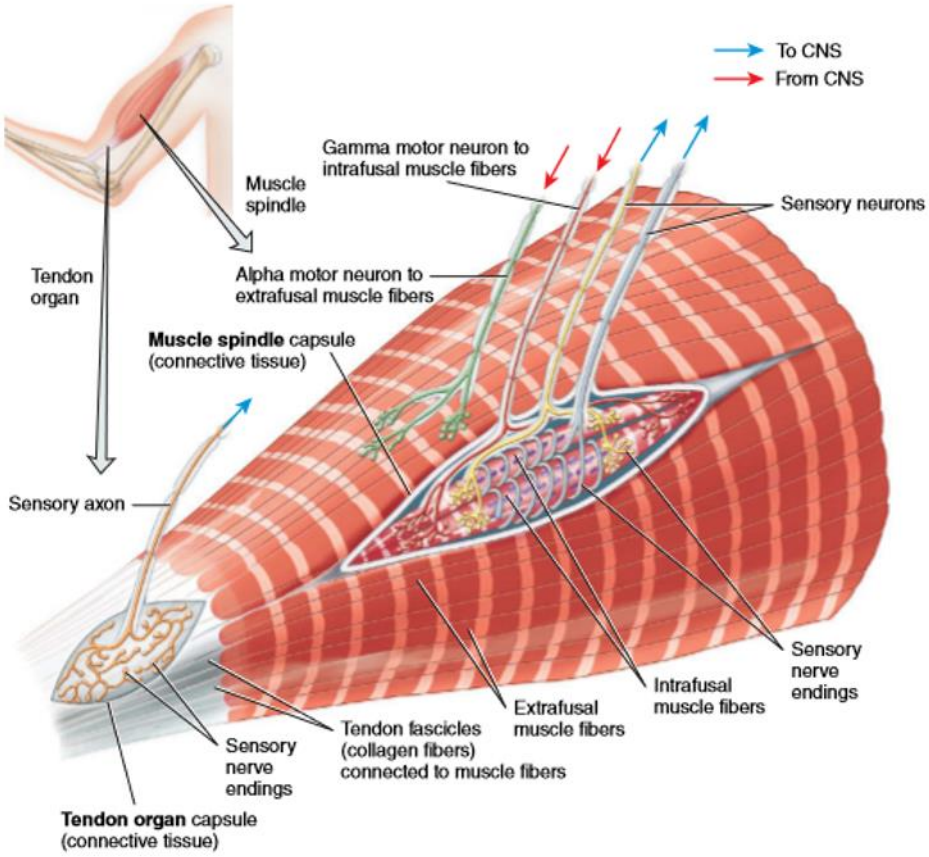

Figure 2.16: Details of golgi tendon organ and muscle spindle (Tortora and Derrickson 2017)

2.5.5.1 Golgi Tendon Organs

Golgi tendon organs (GTO), also known as neurotendinous spindles, are a type of encapsulated mechanoreceptor that is located within the musculotendinous junction of a muscle. The GTO plays an important role in providing the CNS with sensory information on the state of the muscle at that particular moment in time which allows for increased protection to the tendon and surrounding muscles. During muscle contraction, the tendon of that muscle is stretched causing an increase in tension, which will result in a stretching and stimulation of the GTO (Tortora and Derrickson 2017; Netter 2018).

The GTO will generate nerve impulses that travel along the Ib sensory fibres to the spinal cord where they will form an excitatory synapse with inhibitory neurons. Excitation of these neurons will result in an inhibition in the alpha motor neurons, which are responsible for innervating the muscle, causing a decrease in muscle contraction. The GTO forms a feedback mechanism with the CNS that will not only protect the muscle-tendon complex from sustaining damage during excessive muscle contraction or passive stretching, but also provides information that will aid and influence voluntary muscle contraction (Martini, Tallitsch and Nath 2017; Tortora and Derrickson 2017; Netter 2018).

2.5.5.2 Muscle Spindle

Muscle spindles, also known as neuromuscular spindles, are a type of sensory receptor that protects the muscle from undergoing extreme stretching which may result in fibre damage. Muscle spindles are able to perform this function by constantly relaying information to the CNS regarding the muscle length and rate of change in the muscle length. Muscle spindles are slow-adapting sensory nerve endings that are encapsulated by a thin connective tissue which may contain two to ten intrafusal muscle fibres. These sensory receptors are located within the muscle belly and lie parallel to the extrafusal fibres with attachments to the endomysium and perimysium of that muscle. The central part of the muscle spindle has no presence of actin and myosin due to it only being used primarily for sensory perception. In muscles that are required to perform fine motor skills, these spindles are dispersed throughout the

length of the muscle. When a muscle is required for gross movement there are fewer muscle spindles present (Cael 2010; Netter 2018).

When a muscle is stretched, it causes a stimulation or activation of the intrafusal fibres which results in nerve impulses being relayed via the Ia sensory neurons to the spinal cord and CNS. Within the spinal cord, these impulses create an excitatory synapse with the alpha motor neuron which causes a contraction response in the muscle by stimulating the extrafusal fibres. This is known as the muscle stretch reflex is responsible for preventing any overstretching or tearing of the muscle (Martini, Tallitsch and Nath 2017).

As this process occurs, gamma motor neurons activate and cause the endings of the spindles to contract. This results in an increase in the sensitivity of the spindle due to it being taut within the contracting muscles. Not only will this allow the stretch reflex to be activated more quickly, but it will also help the muscle spindles detect and provide more accurate information to the CNS. For the stretch reflex to be effective, axon collaterals from the MS neurons produce excitation of the inhibitory interneurons, which have an inhibitory effect on the alpha motor neurons to the opposing muscle group, resulting in relaxation of the opposing muscle group, known as reciprocal inhibition (Tortora and Derrickson 2017; Netter 2018).

2.6 Surface Electromyography and Muscle Activity

The use of surface electromyography (sEMG) has been dated as far back as the 1600s and is become an increasingly important investigative tool to quantify muscle's electrical activity. This is known as muscle activity. Surface electromyography allows for a safe, non-invasive method of obtaining vital information about the human muscular system which can include the timing and intensity of the activation of superficial muscles, which can occur at rest or during contraction of that specific muscle. The sEMG has undoubtedly become an important tool in research investigations, but over time the use for the sEMG has become more of a clinical application when used to diagnose or monitor conditions such as muscular dystrophy or other neuromuscular diseases (Criswell 2011; Moore, Dalley and Agur 2013).

The sEMG allows the action potentials given off by motor units to be amplified and interpreted (in millivolts) by the system to form a wave-like measurement on a display screen. As the muscle moves through the different phases of contraction the waveform can be visible as peaks and troughs to allow for muscle activity analysis. The sEMG signal can be assessed by different means and methods. An integrated sEMG signal was used for this study which is defined as the area under the curve of the rectified sEMG signal, measured in millivolts. Root Mean Square (RMS) analysis was used as it more easily establishes the maximum value of the sEMG signal (Disselhorst-Klug, Schmitz-Rode and Rau 2009; Criswell 2011; Tortora and Derrickson 2017).

With the sEMG becoming a popular tool in investigative studies, it is important to ensure that the highest quality signal is obtained. When acquiring an EMG signal, it is important to take into consideration that various factors that may alter the interpreted waveform. Some of these factors may be influenced or affected by the investigator. This may include but is not limited to electrode placement, skin preparation, and electrode inter-distance. Intrinsic factors that can alter the EMG signals may be dependent on the structure and composition of the tested subject. This may include skin temperature, skin formation, subcutaneous composition and internal blood flow. Other factors that may alter EMG readings may be cross-talk, which occurs when readings from another muscle group move into the field of the muscle being tested due to a close proximity and movement artefact, which can be created by the movement of the different layers of the skin or by the movement of the cables attached to the electrodes (Disselhorst-Klug, Schmitz-Rode and Rau 2009; Criswell 2011; Chowdhury *et al.* 2013).

The normalisation of EMG signals is important when comparing the EMG activity between different muscles as well when comparing the EMG activity between different individuals. In order to normalise an EMG signal, the signal needs to be divided during a task by a reference EMG value obtained by the same muscle. This allows for a relative measure of activation compared to a reference value. A reference value should be of high repeatability especially in the same test subject in the same session of testing. There are different methods of obtaining a reference value, but for this study, the EMG signals will be normalised by assessing the maximum (peak) activation levels during maximal voluntary isometric contractions (MVIC). It was recommended that at

least three repetitions of the MVIC are performed in order to ensure a higher chance of repeatability (Criswell 2011; Halaki and Ginn 2012).

2.7 Spinal Manipulative Therapy

The use of spinal manipulative therapy (SMT) as a form of treatment has been traced back to around 2700 BC and has now become the primary tool of treatment for osteopaths and chiropractors with increasing popularity in other health care professions. There are various definitions of SMT as some health care professionals consider the term to encompass all forms of manual treatment. However, for the purpose of the study, the definition of a high-velocity, low amplitude (HVLA) thrust will be used to describe SMT (Maigne and Vautravers 2003; Haldeman 2004).

SMT is applied to joints that present or are deemed as fixated. To determine whether there is a joint fixation, an individual will need to undergo a motion palpation screening examination, this allows the practitioner to identify the area of restriction as well as decide if SMT is an appropriate form of treatment. Manual motion palpation is a primary evaluative tool that is used by a physical practitioner to identify areas of hypomobility, hypermobility, pain, tenderness, areas of abnormal ROM and temperature changes (Bergman and Peterson 2011). A dysfunctional segment or joint fixation within the vertebral column can be defined as a restricted joint found during specific palpation of intersegmental range of motion that can be accompanied by palpable tenderness (Owens Jr 2002).

There are several theories have been developed in order to explain the effects of spinal manipulation, but within all these theories there are similar findings that explain that changes in the normal anatomical, biomechanical and physiological relationships of adjacent vertebrae can negatively affect the normal functioning of the nervous system. It has been proposed that joint fixations increase the amount of mechanical strain felt by a vertebral segment which can have a significant impact on the tissues in and around that particular segment. SMT applied to these dysfunctional segments may provide enough energy to restore normal biomechanical movement and reduce the amount of stress and strain present as it has been hypothesised that SMT has the ability to restore zygapophyseal joint mobility (Pickar and Bolton 2012). Other theories suggest that the mechanical input delivered during SMT may also cause a

physiological response due to an influx of sensory information sent to the central nervous system. This may lead to changes such as stimulating or inhibiting mechanoreceptors found in skin, muscles, joints, and tendons, and reduce nociceptive input. It is believed that in dysfunctional segments the mechanical overload may lead to an alteration in the signalling properties of mechanical and chemical sensitive neurons. Abnormal changes of the sensory input may cause altered efferent somatomotor or visceromotor activity leading to symptoms such as discomfort, pain and altered muscle or visceromotor function (Pickar 2002; Herzog 2010; Henderson 2012).

When there is a joint fixation within a vertebral segment, Korr (1975) hypothesised that the hypertonicity of the segmental muscles occurs due to a sudden shortening of the vertebral attachments, causing a slackening or significant shortening of the muscle spindle and thereby silencing the contracting ability of the annulospiral endings. Without any information being sent to the CNS via the annulospiral endings, the CNS registers that the length of the muscle spindle needs to be adjusted in order for it to provide accurate information on the state of the muscle. The CNS is able to adjust the muscle spindle by increasing the gamma motor neuron activity; however, this will simultaneously increase the alpha motor neuron activity causing further contraction of the already contracted segmental muscles. The vertebral segment is unable to return to its normal position causing an increase in mechanical strain. Korr (1975) proposed that SMT had the ability to silence or dampen the increased gamma motor neuron activity by causing a rapid stretch of the extrafusal and intrafusal muscle fibres affecting the muscle spindle producing a cannonade of sensory impulses from the group Ia and II afferents.

Spinal manipulation is the action of applying a controlled low-amplitude high-velocity thrust of varying direction, dependant on the fixation present, in order to move a joint past its physiological range of motion in order to influence or restore the joint's biomechanics and neurophysiological functioning. SMT is often accompanied by an audible 'pop' or cavitation which occurs when there is a physical gapping of the joint causing the change in inter-articular pressure (Haldeman 2004; Bergman and Peterson 2011). When the SMT procedure occurs, it causes various changes to the structures around the fixated spinal segment. Biomechanically, the HVLA thrust

causes a physical separation of the facet joints, a decrease in intervertebral disc pressure, normalisation of activity of the paraspinal muscles and an increase in vertebral segment motion (Maigne and Vautravers 2003).

It has been proposed that a hypomobile spinal segment alters the normal biomechanical movement of the zygapophyseal joints of a vertebral segment which may lead to a disruption of the normal neurophysiological functioning of that particular spinal level. Altered afferent input due to a fixation may cause an over sensitisation of spinal neuron pools allowing for the development of pathological somatovisceral and somatosomatic reflexes. This may lead to signs and symptoms of somatic or visceral dysfunction which includes, but is not limited to, hypertonicity, asymmetry, referred pain and hyperesthesia (Owens Jr 2002; Maigne and Vautravers 2003).

2.8 Physiological effects of pain in relation to Muscle Activity

The physiological effects of pain play an important role in this study. Millan *et al.* (2012) suggested that spinal pain (i.e. low back pain) may have consequences at both local and regional areas that are related to the segmental innervation. The perception of pain may differ between individuals as it is seen to be a non-linear construct that is affected by many factors. However, in the literature several processes have been identified which may explain the adaptations seen in an symptomatic population that may not be present in a similar manner in the asymptomatic population (Knutson and Owens Jr 2003; Millan *et al.* 2012; Harvey and Descarreaux 2013).

2.8.1 Arthrogenic Muscle Inhibition

Arthrogenic muscle inhibition (AMI) occurs after there is a form of injury to a joint, it is defined as an ongoing reflex reaction of the musculature surrounding a joint which can present as inhibition or facilitation (Haavik-Taylor and Murphy 2007). When inhibition occurs, the ability to voluntarily activate the muscles around the joint will be decreased, whereas with facilitation there will be an increase in the potential for muscle activation of the surrounding muscles (McVey *et al.* 2005).

Arthrogenic muscle inhibition is a natural response to excessive stress or injury to a joint, it puts the joint into a protective state to prevent further damage. AMI commonly occurs in the quadriceps femoris muscle and is often associated with pain, disuse,

swelling, and structural damage, but has been suggested to be caused by abnormal information relayed by the disrupted joint receptors, also known as mechanoreceptors (Hart *et al.* 2006; Rice *et al.* 2014). This information acts on inhibitory interneurons that synapse onto the motor neuron pool of the joint musculature, causing a decrease in the ability to recruit and activate muscle fibers which may lead to a decrease in the force of the contraction of the affected muscle (Hopkins and Christopher 2000).

After an injury occurs, an individual may experience a decrease in range of motion (ROM) and marked muscle weakness. The decreased ROM may be due to swelling, pain or the inability to contract the involved muscles maximally (muscle inhibition). If the injured joint is not treated there will be an ongoing cycle of AMI which can lead to further injury or a possible re-injury (Hopkins and Christopher 2000; Rice *et al.* 2014). Arthrogenic muscle inhibition has been shown to occur in several areas of the human body and can have an effect on musculature that is surrounding an injured joint (McVey *et al.* 2005; Rice *et al.* 2014) or extremity musculature that is distant from the spine but is neurologically innervated by the injured spinal segments (Suter and Lindsay 2001; Suter and McMorland 2002)

2.9 The effect of Spinal Manipulative Therapy on Muscle Activity

The evidence to support the neurophysiological mechanisms behind SMT is still controversial, but studies have shown that there is clinical evidence that SMT has the ability to cause an effect on several components of the human body (i.e. muscles and joints). Effects of SMT on local muscles are well documented, but the effects on distal muscles are still limited (Pickar and Bolton 2012). These effects have been shown to be either inhibitory (DeVocht, Pickar and Wilder 2005; Lalanne, Lafond and Descarreaux 2009) or excitatory (Keller and Colloca 2000; Harvey and Descarreaux 2013). Table 2.4, below, describes studies that assess the effect of SMT on muscle activity of muscles related to the spine.

Table 2.4: The Effect of Spinal Manipulative Therapy on Muscle Activity

Author	Sample Size	Study Design	Intervention	Outcome Measures	Results
Currie *et al.* (2016)	n=40, low back pain and asymptomatic	Pre-test, post-test experimental design	1. L/S and SIJ SMT	Muscle activity and onset delays	Increase muscle activity onset delays in the symptomatic group.
Harvey and Descarreaux (2013)	n=60, low back pain	RCT	1. L/S SMT 2. Control	Muscle activity, pain intensity, and lumbo-pelvic kinematics	Increase in paraspinal activity in control during task when compared to SMT group.
Lalanne, Lafond and Descarreaux (2009)	n=27, chronic low back pain	RCT	1. L/S SMT 2. Control	Trunk and pelvic angles, and paraspinal activity during flexion-extension task	Significant decrease in paraspinal activity during full flexion following SMT.
DeVocht, Pickar and Wilder (2005)	n=16, non-specific low back pain	RCT	1. SMT 2. Activator SMT	Resting muscle activity of paraspinal muscles	Significant decrease in muscle activity. Initial increase in activity followed by decrease compared to pre-intervention levels.
Lehman and McGill (2001)	n=14, non-specific low back pain	Analytical cohort	1. L/S SMT	Paraspinal and abdominal muscle activity, and lumbo-pelvic kinematics	No significant changes following SMT.
Keller and Colloca (2000)	n=40, low back pain	RCT	1. Activator SMT (20) 2. Control (10) 3. Sham (10)	Muscle activity of paraspinals during trunk extension (maximum voluntary contraction)	Significant increase in paraspinal activity following manually assisted SMT.
Herzog, Scheele and Conway (1999)	n=10, asymptomatic	Pre-test, post-test experimental design	1. SMT (11 clinically relevant treatments)	SEMG of 16 back and upper limb muscles	Reflex responses in target areas post SMT.

Herzog, Scheele and Conway (1999) were one the first researchers to assess the effects of SMT on neuromuscular reflexes and muscle activity by showing that SMT was able to elicit a repeatable and measurable electromyographic response. It was one of the first studies to show the results of reflex activation of muscles following the application of SMT to the spine and sacroiliac joints in asymptomatic participants (n=10). It has been hypothesised that the clinical benefits of the recorded electromyographic responses may cause a decrease in muscle hypertonicity, reducing pain and improving functional ability.

In a low back pain population (n=40), Keller and Colloca (2000) found that 19 out of 20 participants had a significant increase in erector spinae muscle activity during a voluntary contraction once manually assisted SMT was applied. However these findings were later contradicted by DeVocht, Pickar and Wilder (2005) in a participant population of non-specific low back pain (n=16) who researched and assessed the effect of manual and activator (mechanically assisted) SMT which showed that majority of the cases had a significant reduction in paraspinal muscle activity with only two cases showing an initial increase in activity, but then shortly decreasing to an activity measurement lower than pre-intervention levels. Lalanne, Lafond and Descarreaux (2009) in a placebo-controlled trial found that chronic low back pain participants that received the lumbar SMT had a significant decrease in paraspinal muscle activity during full flexion. These authors suggested that by stimulating the somatosensory system during SMT, there was not only an improvement in pain, but SMT also has the potential to improve trunk functional ability and increase the spinal range of motion.

Similar studies were done by Lehman and McGill (2001) and Harvey and Descarreaux (2013). Both reportedly assessed the effects of SMT on trunk muscles during tasks. Harvey and Descarreaux (2013) found during their randomised controlled trial that participants with low back pain (n=60) that fell into the control group, had an increase in both pain and paraspinal activity during the last 30 minutes of performing the trunk flexion/extension tasks in comparison to the intervention group. This suggests that SMT can reduce sensitisation and muscle fatigue.

Lehman and McGill (2001), during an analytical cohort study (n=14), found that there were no consistent or significant kinematic or electromyographical changes across the population post-intervention, but noted that there were individual changes with the

largest changes occurring in the participants that experienced the greatest amount of pain. Currie *et al.* (2016) observed that the muscle activity in both the asymptomatic and symptomatic population group responded to the application of SMT. Although there was no significant difference between the two groups, the symptomatic group presented with less changes in muscle activity compared to participants with no pain. The authors suggested that an individual may respond differently to the manipulation and that pain could be a factor in the amount of change that occurs.

2.10 The effect of Spinal Manipulation on Muscle Activity of Muscles in the extremities

The studies above have discussed the effects of SMT on the activity within local muscles (i.e. muscles that have direct attachments to the spine), but there is evidence to suggest that these effects may not be limited to those muscles. Studies that support this theory can be seen in table 2.5.

Table 2.5: The Effect of Spinal Manipulation on Muscle Activity of Muscles in The Extremities

Author	Sample Size	Study Design	Intervention	Outcome Measures	Results
Christiansen *et al.* (2018)	n=11, subclinical spinal pain	RCT crossover design	1. SMT 2. Passive Movement	MVC of plantar flexors, Soleus H-reflex and V-wave	SMT increased strength and corticospinal excitability.
Niazi *et al.* (2015)	n=10, subclinical low back pain	RCT	1. L/S SMT 2. Control	Surface EMG V-wave, H-reflex, M-wave, and MVC of ankle plantar flexors	Significant increase in motor neuron pool excitability, cortical drive and preventing fatigue.
Cardinale *et al.* (2015)	n=27, asymptomatic	RCT crossover design	1. L/S SMT 2. L/S Stretching 3. Sham	Force fluctuation task, modified Sorenson's test, and sit and reach. Surface EMG of PS and gastrocnemius muscles.	No improvement superior to other modalities for force output and sEMG parameters post L/S SMT.

Fryer and Pearce (2012)	n=14, asymptomatic	RCT crossover design	1. L/S SMT 2. Control	Surface EMG of right gastrocnemius muscle, M-wave and H-reflex, and MEP amplitudes	Significant decrease in corticospinal and spinal reflex excitability post L/S SMT.
Dunning and Rushton (2009)	n=54, asymptomatic	RCT	1. C/S SMT 2. Sham 3. Control	Resting EMG of biceps brachii muscle bilaterally	Immediate increase in EMG activity following C/S SMT.
Suter and McMorland (2002)	n=16, chronic neck pain	RCT	C/S SMT	Active CROM, elbow flexor force, surface EMG of elbow flexor and pressure pain threshold	After C/S SMT was applied there was significant decrease in muscle inhibition and increase in force output, CROM and pain threshold.

Suter and McMorland (2002) found that subjects who suffered from chronic neck pain (n=16) had shown a significant amount of muscle inhibition within the biceps muscle. The authors theorised that this inhibition had occurred because the affected areas of the cervical spine (C5, C6, C7) were the same levels that gave neurological supply to the bicep muscles. Once SMT was applied to the levels of C5/C6 and C6/C7 they found that there was a significant reduction in muscle inhibition and an increase in biceps force output. This suggested that the neurophysiological effects of SMT can occur in muscles that are distant from the spine by altering the sensory neurological input which leads to an altered efferent pathway. However, the reliability of the findings was lowered due to the small sample size and lack of a control group.

Dunning and Rushton (2009) had similar findings in their placebo-controlled, single-blinded study where they found that SMT applied to the cervical spine at the level of C5/C6 in asymptomatic subjects (n=54) resulted in an immediate increase in the electromyographical activity of the biceps bilaterally when compared to the control group. This change occurred regardless of whether cavitation occurred or not. Although Dunning and Rushton (2009) had a larger sample size and a control group

in comparison to Suter and McMorland (2002), neither study determined how long the effect persisted once undergoing SMT suggesting that further examination is required.

Christiansen *et al.* (2018) found that a single session of SMT resulted in an increase in strength and cortical excitability of the ankle plantar flexor muscle in subclinical pain Taekwondo athletes (n=11) in comparison to the control. Subjects in the treatment group had an increase in their maximum voluntary contractions (MVC) over time which directly contrasted the findings in the control group, these subjects' MVC had decreased over the same period of time suggesting that the control group had undergone a form of muscle fatigue. This may support the findings of the study performed by Harvey and Descarreaux (2013). Similarly, Niazi *et al.* (2015) found in participants with subclinical low back pain (n=10) there was a significant improvement in the MVC following SMT when compared to the control. The authors suggested that the improvement in the MVC was most likely attributed to the increase in cortical excitability and modulation of the sensory afferents.

Supporting the findings of Lalanne, Lafond and Descarreaux (2009), Fryer and Pearce (2012) performed a study that showed that lumbar SMT resulted in a significant decrease in the spinal reflex and cortical excitability in asymptomatic subjects (n=14). This indicates the SMT is able to cause an immediate decrease in the motor neuron excitability. In a randomised controlled, cross over trial Cardinale *et al.* (2015) examined the effects of lumbar spine SMT in comparison to lumbar spine stretching and sham intervention on the gastrocnemius and erector spinae muscles in asymptomatic subjects (n=27). After each treatment subjects were required to perform three different tasks: force fluctuation task, modified Sorensen's test, and sit and reach test. The results showed that the lumbar SMT showed no improvements that were superior to any of the other two treatments to which the subjects were exposed.

The current literature has varying results when examining the exact effects of SMT. Increased muscle activity is most likely attributed to an increase in corticospinal excitability, improved motor recruitment and altered sensory afferents (Suter and McMorland 2002; Dunning and Rushton 2009; Harvey and Descarreaux 2013). However, in some studies, the results contrasted these findings suggesting that response to SMT may differ between individuals (Lehman and McGill 2001; Fryer and

Pearce 2012). The conflicting results of the effects of SMT on muscle activity warrant further investigation.

2.11 The effects of Spinal Manipulation on Knee Extensor Muscle Activity

It has been proposed that areas of joint fixation may demonstrate a state of altered sensory input (Haavik-Taylor and Murphy 2007). The consequence of developing an abnormal sensory input is altered efferent output to both local and distant muscles and joints which has been shown to result in altered motor recruitment patterns, abnormal muscle activity and decreased force output predisposing an individual to injury (Suter and McMorland 2002; Niazi *et al.* 2015; Christiansen *et al.* 2018). The following studies, found in table 2.6, assess the effects of SMT applied to joint fixations in the spine and the effects it has on the muscles relevant to this study.

Table 2.6: The effects of spinal manipulative therapy of knee extensor muscle activity

Author	Sample Size	Study Design	Intervention	Outcome Measures	Results
Grindstaff *et al.* (2014)	n=75, history of knee joint injury	RCT	1. L/P SMT 2. Sham 3. Grade IV Patellar mobs 4. Grade I Patellar mobs 5. Control	Surface EMG H-reflex of quadriceps over time (pre, post, 30, 60, 90 min)	No significant differences in H-reflex between the groups
Grindstaff *et al.* (2009)	n=42, asymptomatic	RCT	1. L/P SMT 2. Lumbar passive ROM 3. Prone extension on elbows for 3 min	Quadriceps force output and activation over time (pre, post, 20, 40, 60 min)	Immediate significant change in quadriceps activation and force output following L/P SMT
Hillermann *et al.* (2006)	n=20, patellofemoral pain syndrome	RCT	1. Tibiofemoral Joint Man 2. SIJ Man	Quadriceps force output	Significant change after L/P SMT from pre to post.
Suter *et al.* (2000)	n=28, anterior knee pain	RCT	1. SIJ Man 2. Control	Quadriceps strength, muscle inhibition and activity.	Significant decrease in muscle inhibition following SIJ man.

Pollard and Ward (1996)	n=30, asymptomatic	RCT	1. L/S SMT (L3/L4) 2. Sham	Quadriceps force output	Significant short-term increase in quadriceps strength

Grindstaff *et al.* (2009) conducted a study using asymptomatic participants (n=42) to determine whether a lumbopelvic manipulation, lumbar mobilisation or a prone extension position would alter the muscle activation and force output capability of the quadriceps muscle. Following the lumbopelvic manipulation, subjects had a significant immediate increase in both activation and force output of the quadriceps muscle. However, these changes were no longer present when the subjects were retested at the 20-minute interval. The authors theorised that the use of asymptomatic patients may explain why the changes remained for only a short period of time suggesting that participants with a history of knee pathology may allow for a larger capacity of change in quadriceps activation. In more recent years, this theory was contradicted when Grindstaff *et al.* (2014) performed a randomised controlled trial using subjects with a history of knee pain (n=75) to determine if a lumbopelvic manipulation or various patella mobilisation techniques could alter the quadriceps' neuromuscular excitability. The results suggested that the lumbopelvic manipulation, patella mobilisation, and lumbopelvic positioning produced no significant immediate changes to the quadriceps excitability.

Suter *et al.* (2000) examined the effects of sacroiliac SMT on knee extensor inhibition in participants with anterior knee pain. The results showed a significant decrease in the muscle inhibition of the affected leg following sacroiliac joint (SIJ) manipulation suggesting that SMT may be an effective treatment of muscle inhibition in the lower limb musculature. Furthermore, Hillermann *et al.* (2006) investigated the effects of an SIJ manipulation compared to tibiofemoral manipulation on quadriceps' strength in participants with patellofemoral pain syndrome (n=20). There was a significant change in the quadriceps in the treatment group that received the SIJ manipulation. Pollard and Ward (1996) found that during a randomised controlled trial using lumbar SMT applied specifically to the level of L3/L4 resulted in an immediate significant increase in quadriceps' strength in an asymptomatic population (n=30).

These studies suggest that by applying manipulation to the spine, it resulted in a change of the sensory input that may have altered efferent pathways at the segmental

level which had an effect at body parts distal to the spine. Research has shown that spinal manipulation is able to cause an effect on muscle activity in both asymptomatic and symptomatic participants. However, several previous studies have only examined the immediate effects of SMT on distant muscles (Suter and McMorland 2002; Dunning and Rushton 2009; Cardinale *et al.* 2015). There is a gap in the literature regarding the duration of the effects of the SMT when comparing asymptomatic to symptomatic individuals due to the fact that studies often compared the immediate effects on asymptomatic and symptomatic groups to control subjects where they had not undergone any intervention.

2.12 Summary of the literature

Several studies have demonstrated that lumbar and sacroiliac joint SMT can have an effect on quadriceps' activation in individuals that suffer with a form of knee pain, due to AMI occurring on the muscles surrounding the affected joints, as well as in asymptomatic individuals (Hillermann *et al.* 2006; Grindstaff *et al.* 2009). However, no studies have examined the effects of SMT on quadriceps muscle activity in individuals who suffer from low back pain despite that is has been clearly observed that they exhibit similar changes in the quadriceps muscle even though there is no direct attachment (Verbunt *et al.* 2005; Hart, Weltman and Ingersoll 2010). Spinal manipulative therapy is used as a treatment for many musculoskeletal conditions, but the actual neurophysiological mechanism behind SMT still requires further investigation. Thus, this study aims to determine the effect of SMT on the quadriceps femoris and hamstring muscle activation in symptomatic (low back pain) and asymptomatic individuals, and ultimately observing the duration and magnitude of this effect.

CHAPTER THREE: METHODOLOGY

3.1 Introduction

This chapter discusses the design of the study, the sample population and recruitment process, the criteria for inclusion and exclusion as well as the group allocation and randomisation. It also outlines the research procedure and protocol followed and the measurement tools utilised in the study. The intervening treatments, the process of statistical data analysis and ethical considerations are discussed.

3.2 Study Design

This study's research design provided the opportunity for both asymptomatic and symptomatic participants to be randomly placed into either the control or intervention group. All groups were tested prior to the administration of the intervention or control procedures, then following the intervention/control post-test measurements were obtained in order to determine the effectiveness of the intervention (Kumar 2014).

This study was performed using a quantitative approach and was a randomised controlled trial which utilised a pre-and-post experimental design that included a symptomatic and asymptomatic group. Each clinical group had a treatment and control group (Dunning and Rushton 2009; Grindstaff *et al.* 2009; Cardinale *et al.* 2015).

3.3 Study Location

The study was conducted at the Chiropractic Day Clinic (CDC) at the Ritson campus of Durban University of Technology (DUT), a facility that provides chiropractic treatment to the public by students who are completing their Master's Degree in Technology of Chiropractic. Students are overseen by qualified chiropractic clinicians. Gatekeeper's permission was obtained from the Clinic Director for the study to take place on the premises (Appendix A)

3.4 Ethical Considerations

This study was approved by the Institutional Research Ethics Committee (IREC) (Ethical Clearance number IREC 113/19) (Appendix B) and was registered with the

Pan African Clinical Trial Registry (PACTR202001586284982) (Appendix C). Permission had been obtained from the Research director (Appendix D) to have access to the university staff and students.

- Participation in this study was completely voluntary. The participants were allowed to leave the study at any time if they felt that they did not want to participate in the study going further.

- The participants were required to complete a letter of informed consent (Appendix L). Information about the study, as well as any questions/concerns that the participant or potential participant had been addressed.

- Participant's information was strictly confidential by using codes rather than their name. All patient information was stored at the DUT CDC. Participant information was only accessible by the researcher, supervisor, and statistician.

- Justice was accounted for as there was no discrimination when the participants were placed into intervention groups. Patient participation was not based on gender, race, nationality, or religion.

- All participants that were in the symptomatic control group received a treatment voucher to attend the DUT CDC. The study was not used to assess any improvement in pain, but rather to assess how pain affected the human body. If these participants wished to seek treatment following the study, they were able to do so with the treatment voucher (Appendix E).

- Patients were informed that there was a chance that they would be allocated to any of the groups (treatment or control).

- Non-maleficence and benefit would be enforced by ensuring no harm was done to the participants as the interventions and measurement tools are safe and registered.

3.5 Sampling

3.5.1 Study Population

The study population consisted of 48 participants who reside in the eThekwini municipality area. The population had 24 symptomatic participants, who presented with non-specific low back pain, and 24 asymptomatic participants, who were experiencing no low back or lower extremity pain. All participants met the inclusion criteria. This sample size was determined by power analysis calculations (Appendix F) and reflects the minimum sample required for effects to be noticeable (Esterhuizen 2019).

3.5.2 Recruitment

Participants were recruited from the eThekwini municipal area by means of advertising. Advertisements (Appendix G) were placed throughout the DUT campuses and around other areas in Durban such as local universities, gyms and health clubs. Some participants were recruited via word of mouth. Potential participants contacted the researcher (using the information supplied by the advertisements or by word of mouth). Those who wished to participate were asked the following qualifying questions found in table 3.1 below.

Table 3.1: Telephonic screen

Question	Yes	No	Comments
Would you mind answering a few questions?		X	
How old are you?			Between the ages of 18-45.
Have you been experiencing any low back pain?	X	X	
How long have you experienced the low back pain for?			At least three months duration.
Have you had any injuries or surgery to your low back?		X	
Have you recently had any injuries or surgery to either of your hips, knees or ankles?		X	

Are you on any pain medication? If so, what medication?	X	X	If yes, name of medication is required.
Have you had any low back chiropractic treatment in the last four weeks?		X	
Do you suffer from a chronic illness?		X	

Those who were eligible and still agreed to participate were scheduled for an appointment at the DUT CDC. At the appointment, a full case history (Appendix H), physical examination (Appendix I) and lumbar spine regional examination (Appendix J) were conducted to determine if the participant met the study inclusion and exclusion criteria.

3.5.3 Inclusion Criteria

- Individuals were required to have completed the letter of informed consent (Appendix L) and sign the letter of information (Appendix M) to participate in this study.

- Participants between the ages of 18-45 years were required for this study. Subjects older than 45 years would not be able to participate in the study as (Ding *et al.* 2005) found evidence that knee cartilage defects were far more common in subjects over 45 years of age than those who were younger.

- Participants had to be older than 18 years old to avoid having to obtain parental permission.

- Each participant had to have presented with at least one fixation in the relevant levels (L2-L5) of the lumbar spine as the purpose of the study was to investigate the effects of a lumbar spine manipulation on the muscle activity and joint position sense of the lower limb. Motion palpation of the lumbar spine was used to determine if there was a fixation in these areas (Bergman and Peterson 2011).

Symptomatic Participants (Lamoth *et al.* 2006; Balagué *et al.* 2012):

- Low back pain for a duration of three or more months.

- Diagnosis of low back pain must be of a mechanical origin.
- Pain must be located in the region below the lower margin of the 12th rib and above the gluteal fold (lumbopelvic hip complex).
- Participants must have reported a minimum of three to a maximum of six on the numerical pain rating scale of the SOAPE note (Appendix K). This was performed to ensure a more homogeneous sample (Younger, McCue and Mackey 2009).

Asymptomatic Individuals (Lamoth *et al.* 2006):

- No history of low back pain or no lower back pain three weeks prior to the study.

3.5.4 Exclusion Criteria

- Participants would not be a part of the study if they had any contraindications to spinal manipulative therapy (SMT) based on a case history and physical exam (Bergman and Peterson 2011). This may include, but was not limited to the following:

  – Neurological conditions (cauda equine syndrome, neuropathies)

  – Malignancy (tumour)

  – Vascular (aortic aneurysm, atherosclerosis)

  – Infections (osteomyelitis)

  – Skeletal conditions (osteoporosis, osteoarthritis, fractures)

- The eligible participant must not have been adjusted four weeks prior to their visit to participate in the study as this may allow for possible effects of previous treatment to alter the outcome of the readings and results.
- Participants who did not present with fixations in the relevant spinal levels (L2-L5).

- Individuals who presented with hip, knee, or ankle pain as a result of trauma or surgery (Hurley, Jones and Newham 1994; Hurley 2003).
- Contra-indications to surface electromyography including, but not limited to:
  - Open wounds and rashes
  - Skin conditions (psoriasis, eczema)
- Participants who had primary or secondary disorders that may compromise normal neurological function (i.e. Diabetes Mellitus) (Eliasson 1964) as this may alter results in the study.
- Participants who were taking medication that may have affected muscle activity (i.e. sedatives and stimulants) (Kruk 2014).
- Participants who had a Body Mass Index (BMI) of 30 or greater as this may affect surface EMG readings.

Symptomatic Participants (Lamoth *et al.* 2006)

- Participants who presented with low back pain due to trauma or structural anomalies.
- Low back pain with neurological deficits (muscle weakness, reflex changes) and radiation into the legs.
- Previous back surgery
- Spinal tumours or infections.

3.5.5 Sample Size

Using G*Power version 3.1.9.2 and assuming a moderate effect size of 0.25 for a repeated measures ANOVA with interaction, significance level of 0.05 and power of 80%, the estimated sample size required was four groups of 12 individuals (48 in total).

The four groups were:

Symptomatic intervention – Group 1

Symptomatic control – Group 2

Asymptomatic intervention – Group 3

Asymptomatic control – Group 4

Participants were randomly allocated to one of each of the four groups (Appendix F) (Esterhuizen 2019).

3.5.6 Sample Allocation

Once the participant was accepted into the study, they were then randomised into the intervention or control group via the 'hat' method. Two boxes, one for the symptomatic group and one for the asymptomatic group, were both filled with 24 pieces of paper. Twelve pieces of paper indicated that the participant was in the intervention group and the other 12 pieces indicated that they were in the control group. Both boxes were mixed thoroughly and, without looking, the participant drew a folded piece of paper. Each participant had an equal opportunity to fall into either group (Kumar 2014; Crano, Brewer and Lac 2015).

Symptomatic box:

- A – Intervention
- B – Control

Asymptomatic box:

- C – Intervention
- D – Control

3.5.6.1 Consort Flow Diagram

The flow diagram below represents the distribution of the participants through the research. After appropriate allocation, it resulted in 24 participants in the symptomatic group and asymptomatic group.

Figure 3.1 Consort Flow Diagram

3.6 Measurement Tools

3.6.1 Surface Electromyography

This equipment provided a non-invasive method of measuring the muscle activity within the lower limbs, specifically in the quadriceps femoris and hamstring muscle groups (Mathur, Eng and MacIntyre 2005; Criswell 2011). The EMG machine that was used in the study had the following specifications:

LabMate Biopac – Bionomadix® complete wireless research system with four channel EMG recording.

Electrodes used:

35mm Round VitaTrode Midi-ACF - Adult ECG electrodes

Surface EMG (sEMG) reading may be influenced or affected by several factors and may have a day to day variance. Readings may vary according to electrode shape, size, placement, and preparation of the skin (Mathur, Eng and MacIntyre 2005). However, all readings using this equipment had been completed in one appointment, which reduced the chance of the readings being influenced by these factors. De Luca (2002) suggested that the accuracy of the EMG readings may be affected by factors such as cross-talk susceptibility, where larger electrodes may pick up signals from adjacent muscles, and signal-to-noise ratio.

The following electrode placement is taken from Criswell (2011):

Vastus Medialis:

Electrodes were placed on the medial aspect of the thigh (approximately at 55° angle) immediately above the patella.

Rectus Femoris:

The electrodes were placed on the medial anterior surface of the thigh, approximately half the distance between the hip and the knee.

Electrodes were placed in a perpendicular plane from the ground so that the electrodes followed the muscle fibres.

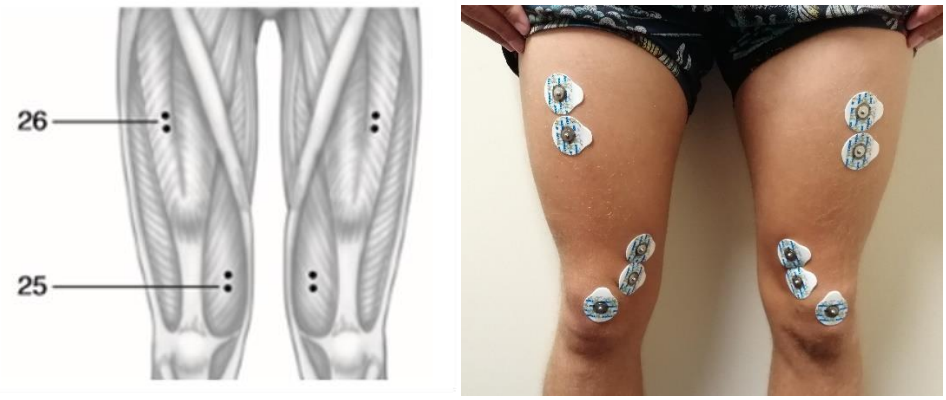

Figure 3.2 Electrode placement for vastus medialis (25) and rectus femoris (26) (Criswell 2011)

Hamstring muscle group:

The electrodes were placed on the medial posterior aspect of the thigh, about half the distance between the hip and the knee.

The electrodes are placed in a vertical plane relative to the ground so that they run parallel to the muscle fibres.

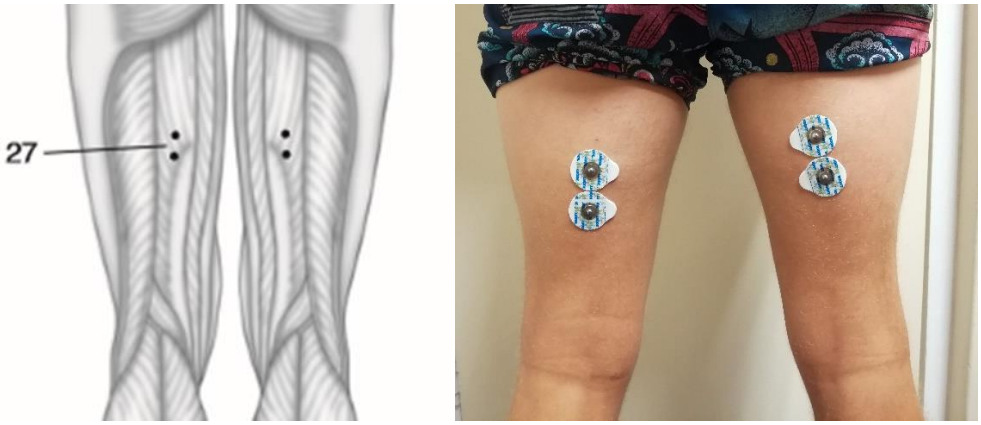

Figure 3.3 Electrode placement for hamstring muscle (27) (Criswell 2011)

Criswell (2011) recommended that the electrodes are placed between two motor points or one motor point and the tendon insertion of the tested muscle. However, the electrodes should not be placed on a tendon as there will be decreased amplitude and an increase in crosstalk (Criswell 2011).

Mathur, Eng and MacIntyre (2005) and Halaki and Ginn (2012) found that the reliability for the amplitude measurements was moderate to high in maximal isometric contractions. Skin preparation may have affected the surface EMG readings, especially in participants with hairy skin; therefore, a method of skin preparation was followed each time an individual participated in the study

- If the skin appeared dry with a layer of dead skin cells, a wet piece of tissue was rubbed over the area where the electrode will be placed.
- Hairy skin was shaved around the area of interest using a disposable razor.
- The skin, where the electrodes were placed, was wiped down with a wet swab.

3.7 Intervention

The intervention that was applied to each participant was based on the group into which they were allocated:

Intervention groups (Group 1 and 3):

The participants in the intervention group received a lumbar spine manipulation dependant on the restriction found during the motion palpation assessment. The Diversified Technique was used (Bergman and Peterson 2011). The lumbar spine segments of L2, L3, L4, and L5 were the only levels where SMT was administered as they have a neurological link to the quadriceps femoris and hamstring muscle groups (Moore, Dalley and Agur 2013; Tortora and Derrickson 2017). If a participant presented with a single restriction at the L4 level, that was the only segment where SMT was applied. In order to ensure a form of consistency, all methods of SMT used, regardless of the direction of the fixation, had the patient in the side-lying position (lateral recumbent position).

The general outline of the procedure (Bergman and Peterson 2011):

Patient position:

- The patient is placed into a lateral recumbent position and the headrest is elevated.

Doctor position:

- The doctor/researcher stands in a square stance.
- For larger patients, a fencer stance may have been used.

The contact point of the researcher's hand and the segmental contact point on the patient changed according to the fixation present.

Control groups (Group 2 and 4):

The control group involved the participants remaining motionless on the chiropractic bed in the supine position for approximately three minutes, which was the average time taken for the setup and manipulation in groups one and three respectively. This group allowed comparison to no intervention and the passing of time as a variable.

3.8 Data Reduction and Analysis

At each consultation, the sEMG readings for muscle activity were acquired during three sets of MVCs that occurred prior to the intervention, post-intervention, and 10-minutes post-intervention. This raw data was processed using root mean squared (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 recorded from each contraction, this value was later divided by three in order to obtain the mean muscle activity of each set of contractions.

Normalisation requires a conversion of the processed data into a scale relative to a known and repeatable value (Halaki and Ginn 2012). This allowed the researcher to be able to compare the EMG activity between different muscles and different individuals. The normalisation of EMG data required that the mean muscle activity acquired from the three contractions was divided by a reference value, a value that should have had high repeatability in the same participant during the one consultation (Rouffet and Hautier 2008; Criswell 2011).

For this study, the process of obtaining peak activation levels during maximal voluntary contractions was used where the highest value of all three contractions was recorded. According to Halaki and Ginn (2012), this was a common method of obtaining both the

peak value and mean muscle activity. The normalised values were displayed as a percentage of the MVC, which allows for a standardised level comparison of the data between participants.

(pre-mean MA ÷ pre-peak amplitude) x 100 = %

(post-mean MA ÷ pre-peak amplitude) x 100 = %

(10-minute post-mean MA ÷ pre-peak amplitude) x 100 = %

IBM SPSS version 26 was used to analyse the data. Repeated measures ANOVA tests were used to compare each outcome between pre- and immediate post-treatment between the intervention and control groups for immediate effects, and pre- and 10-minute post-treatment between the intervention and control groups for short -term effects. This was initially done within the asymptomatic and symptomatic participants separately. In order to compare results in the symptomatic and asymptomatic groups, the effect of the clinical group was used as a between-subject effect for objective 3. In each analysis, three hypotheses were tested: time effect (change from pre to post), group effect (the difference between intervention and control) and time x group interaction effect. The time x group interaction effect measured the effect of the intervention from pre to post, and this was the effect of interest. A significant time x group interaction effect signified that the intervention group showed a difference over time compared with the control group. Profile plots were used to visualise the trend and direction of the intervention effect (Esterhuizen 2019).

3.9 Study Procedure

The participants who met the criteria for the telephonic screening interview presented to the DUT CDC where they received a full verbal explanation of the study and the data collection procedure. They were informed that they were able to withdraw from the study at any time and that it would not affect any further treatments they wished to have at the clinic. Participants were required to read and sign both a letter of informed consent (Appendix L) and a letter of information (Appendix M). The individual was only allowed to participate in the study once all required forms were signed. Each participant would be informed that the consultation would take approximately two hours and a follow-up appointment would not be needed.

The participant would undergo a case history (Appendix H), physical exam (Appendix I), and a regional exam of the lumbar spine (Appendix J). If the participant did not meet the inclusion criteria and/or did not present with a fixated segment in the lumbar spine the participant was thanked but was excluded from the study. If he/she withdrew from the study or a more sinister condition was suspected after the relevant examinations, the participant was later referred to the appropriate practitioner.

It was determined during the consultation and physical examination whether the participant would be excluded from the study or they would be placed into the symptomatic or asymptomatic groups. The participant was informed that they have a chance of being placed into either the intervention or control groups by picking a folded piece of paper out of a box. To determine if the participant had dysfunctional segments, motion palpation of the lumbar spine according to Bergman and Peterson (2011) was used. Motion palpation testing of the lumbar spine and all intervention procedures were performed by the researcher.

Once the participants were placed in their respective groups an explanation of the equipment and procedure was given. Each participant was taught and shown how to perform a maximum voluntary isometric contraction (MVIC) of each muscle being tested. It was vital that each MVIC was as close to identical each time to produce more accurate results (Halaki and Ginn 2012). The skin where the electrodes for the surface EMG were placed was prepared as previously stated. Electrodes were placed at the specific sites for each muscle being tested. At this point, the patient was asked to sit in the designated chair.

For the pre-readings, the strap for the maximal voluntary isometric contraction (MVIC), which was stabilised by the chiropractic bed, was placed 3cm proximal to the lateral malleolus on the posterior surface of the right leg. The participant's leg must have been flexed to approximately 90 degrees. The straps used were adjustable, therefore, they were adjusted in length according to the participant's height to ensure that testing was performed at a 90-degree angle This was the angle where the participant was asked to perform a manually resisted MVIC of the hamstring muscle (resisted flexion of the knee). The participant was required to sit upright in the chair and instructed not to grab the sides of the chair when performing the MVIC. The participant was required to flex their knee for to the best of their ability for five seconds. This was repeated three times

with 30 seconds in between each contraction to prevent fatigue. This procedure was then performed on the left leg for the readings of the left hamstring muscle.

The straps were then attached to the back of the chair which provided a stable anchor for the participant to perform MVIC in extension. The strap was placed 3cm proximal to the lateral malleolus on the anterior surface of the leg and adjusted to ensure that the leg being tested positioned with the knee at 90-degrees. As with the hamstring group, this was performed on the right quadriceps muscle first followed by the left. Three pre-readings would be taken from each muscle when the MICV is performed and recorded. Each MVIC would be separated by 30-second intervals of rest to reduce muscle fatigue.

The participant was then asked to move to the bed where they would undergo the relevant procedure dependent on whether they were placed in the intervention or control groups as well as what fixations were found. Immediately after the intervention or control, three post-readings of each muscle (during a MVIC) were re-evaluated in the same manner as it was performed in the pre-test. These results were then recorded. After a duration of time (10 minutes) after the last MVIC, another three MVIC of each muscle group were re-performed and recorded (Grindstaff *et al.* 2009).

The straps for the MVIC and electrodes of the surface EMG were removed from the patient's skin. The patient was thanked for participating in the study and was issued with a voucher to return to the CDC for one free treatment if they were placed into the symptomatic control group. All readings from the surface EMG and dynamometer were captured on a Microsoft® Excel spreadsheet. The results were then submitted to a biostatistician for analysis. All relevant information was reported back to and signed by the supervising clinician.

3.10 Conclusion

This chapter has described the research protocol utilised in the study and explained the manner in which data was obtained and analysed. The next chapter will present the findings of the study.

CHAPTER FOUR: RESULTS

4.1 Introduction

This chapter presents 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.

4.2 Demographic characteristics of the participants

4.2.1 Age

The sample consisted of 48 participants who were between the ages of 18 to 45 years of age. All participants met the inclusion criteria and were allocated into each clinical group dependent on their symptoms. Table 4.1 displays the mean age of the sample within the clinical groups.

Table 4.1: Mean age per clinical group

Clinical Group				
Group	N	Mean	Std. Deviation	p Value
Symptomatic	24	27.9	7.7	0.058
Asymptomatic	24	24.4	4.2	

Participants that were placed into the symptomatic group had an average of 27.9 years with the asymptomatic group having an average of 24.4 years of age. There was a marginally non-significant difference between the symptomatic and asymptomatic groups (p=0.058) with symptomatic participants being slightly older.

Within each of the symptomatic and asymptomatic clinical groups, each participant was randomly assigned to an intervention or control group. Table 4.2 displays the mean age distribution between the intervention and control groups.

Table 4.2: Mean age per group

Group				
Group	N	Mean	Std. Deviation	p Value
Intervention	24	27.3	6.2	0.243
Control	24	25.1	25.1	

In the intervention group the average age was 27.3 years compared to the control group which had an average age of 25.1 years. However, there was no statistical difference in age between the intervention and control groups (p=0.243).

4.2.2 Gender Distribution

This study included both females and males. Table 4.3 shows the gender distribution across the clinical groups and the experimental groups.

Table 4.3: Gender distribution

		Group			
		Intervention		Control	
Clinical Group	Gender	Count	Column N (%)	Count	Column N (%)
Symptomatic	Male	7	58.3	5	41.7
	Female	5	41.7	7	58.3
Asymptomatic	Male	5	41.7	5	41.7
	Female	7	58.3	7	58.3

Twenty-four symptomatic and 24 asymptomatic participants were randomised equally into intervention and control groups, with 12 participants per group. There was a similar proportion of each gender within the symptomatic and asymptomatic participants of each treatment group, as shown in the table below.

4.3 Mean Muscle Activity

Repeated measures ANOVA tests were used to compare each outcome between pre- and immediate post-treatment between the intervention and control groups for immediate effects, and pre- and 10-minute post treatment between the intervention

and control groups for short-term effects. The effects of lumbar SMT on the relevant muscles; right hamstring (RHAM), left hamstring (LHAM), right rectus femoris (RRF), left rectus femoris (LRF), right vastus medialis (RVM) and left vastus medialis (LVM) in each clinical group have been described in relation to the main objectives of this study.

4.3.1 Objective One

To determine the immediate and short-term effect of lumbar spinal manipulative therapy on the muscle activity on the quadriceps femoris and hamstring muscle groups in asymptomatic participants.

4.3.1.1 Asymptomatic Group – Immediate effects of L/S SMT on muscle activity

Table 4.4 summarises the immediate effects of lumbar SMT on the post readings in the asymptomatic group.

Table 4.4: ANOVA for immediate effect of asymptomatic group

Asymptomatic (Immediate Effects)							
Multivariate Test							
Muscle	Test	Effect	Value	F	Hypothesis DF	Error DF	Sig.
RHAM	Wilks' Lambda	Time	.926	1.754c	1.000	22.000	.199
		Time*Group	.978	.488c	1.000	22.000	.492
LHAM		Time	.990	.214c	1.000	22.000	.648
		Time*Group	.913	2.090c	1.000	22.000	.162
RRF		Time	.958	.966c	1.000	22.000	.336
		Time*Group	.924	1.802c	1.000	22.000	.193
LRF		Time	.918	1.977c	1.000	22.000	.174
		Time*Group	.933	1.577c	1.000	22.000	.222
RVM		Time	.892	2.661c	1.000	22.000	.117
		Time*Group	.999	.014c	1.000	22.000	.907
LVM		Time	.986	.307c	1.000	22.000	.585
		Time*Group	1.000	.011c	1.000	22.000	.919

(RHAM=Right Hamstring, LHAM=Left Hamstring, RRF=Right Rectus Femoris, LRF=Left Rectus Femoris, RVM=Right Vastus Medialis, LVM=Left Vastus Medialis)

Table 4.4 demonstrates that there was no evidence of a treatment effect of the intervention in the asymptomatic group for the RHAM (p=0.492), LHAM (p=0.162), RRF (p=0.193), LRF (p=0.222), RVM (p=0.907) and LVM (p=0.919). For the majority of the muscles, there was also a non-significant time effect and group effect, thus the changes over time were non-significant overall and the group differences (between intervention and control) were also not statistically significant, however, LRF had a borderline significant group effect (p=0.050). Thus, the changes over time were non-significant overall but the intervention group values were higher at both time points than those of the control group.

4.3.1.2 Asymptomatic Group – Short-term effects of L/S SMT on muscle activity

Table 4.5 summarises the short-term effects of lumbar SMT on the post readings in the asymptomatic group.

Table 4.5: ANOVA for short-term effect of asymptomatic group

Asymptomatic (Short term effects)							
Multivariate Test							
Muscle	Test	Effect	Value	F	Hypothesis DF	Error DF	Sig.
RHAM	Wilks' Lambda	Time	.844	1.947c	2.000	21.000	.168
		Time*Group	.849	1.862c	2.000	21.000	.180
LHAM		Time	.788	2.827c	2.000	21.000	.082
		Time*Group	.807	2.503c	2.000	21.000	.106
RRF		Time	.953	.519c	2.000	21.000	.603
		Time*Group	.871	1.558c	2.000	21.000	.234
LRF		Time	.916	.958c	2.000	21.000	.400
		Time*Group	.922	.888c	2.000	21.000	.426
RVM		Time	.869	1.585c	2.000	21.000	.228
		Time*Group	.965	.383c	2.000	21.000	.687
LVM		Time	.981	.208c	2.000	21.000	.814
		Time*Group	.897	1.202c	2.000	21.000	.321

Table 4.5 shows that there was no evidence of a treatment effect of the intervention in the asymptomatic group for the RHAM (p=0.180), LHAM (p=0.106), RRF (p=0.234), LRF (p=0.426), RVM (p=0.687) and LVM (p=0.321). There was also a non-significant time effect and group effect, thus the changes over time from pre, post and 10-minutes

were non-significant overall and the group differences were also statistically insignificant.

4.3.2 Objective Two

To determine the immediate and short-term effect of the lumbar spinal manipulative therapy on the muscle activity on the quadriceps femoris and hamstring muscle groups in symptomatic participants.

4.3.2.1 Symptomatic Group – Immediate effects of L/S SMT on muscle activity

Table 4.6 summarises the immediate effects of lumbar SMT on the post readings in the symptomatic group.

Table 4.6: ANOVA for immediate effect of symptomatic group

Symptomatic Group (Immediate Effects)							
Multivariate Test							
Muscle	Test	Effect	Value	F	Hypothesis DF	Error DF	Sig.
RHAM	Wilks' Lambda	Time	.801	5.472c	1.000	22.000	.029
		Time*Group	.981	.425c	1.000	22.000	.521
LHAM		Time	.662	11.238c	1.000	22.000	.003
		Time*Group	.994	.143c	1.000	22.000	.709
RRF		Time	1.000	.003c	1.000	22.000	.954
		Time*Group	.832	4.439c	1.000	22.000	.047
LRF		Time	1.000	.001c	1.000	22.000	.978
		Time*Group	.895	2.587c	1.000	22.000	.122
RVM		Time	.986	.318c	1.000	22.000	.578
		Time*Group	.911	2.145c	1.000	22.000	.157
LVM		Time	.998	.055c	1.000	22.000	.817
		Time*Group	1.000	.001c	1.000	22.000	.971

Results presented in table 4.6 show that there was no evidence of a treatment effect of the intervention in the symptomatic group for the RHAM (p=0.521), LHAM (p=0.709), LRF (p=0.122), RVM (p=0.157) and LVM (p=0.971). However, there was evidence of a treatment effect for the RRF (p=0.047). Thus, the changes over time were significant by group (difference between intervention and control) which indicated an intervention effect for this outcome in the symptomatic participants.

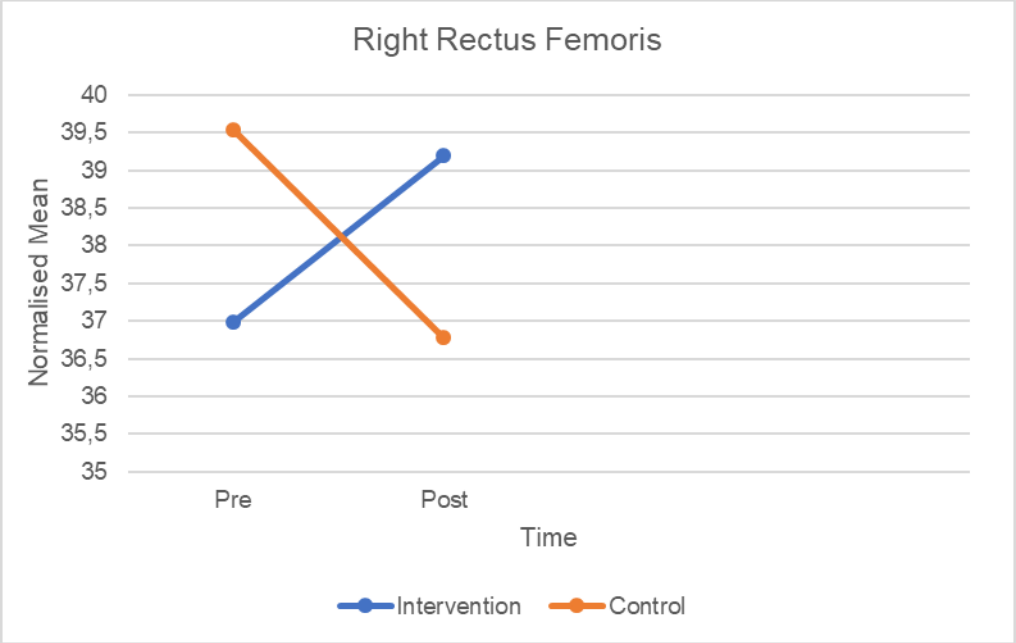

Figure 4.1: Normalised Mean Muscle Activity of RRF

Figure 4.2 depicts the following plot demonstrating the intersecting lines of both experimental groups with the intervention increasing while the control group decreased. In the RHAM and LHAM muscles there was a significant time effect of p=0.029 and p=0.003 respectively, however, both muscles had a non-significant group effect. This indicates that the changes over time were significant overall but the group differences were not statistically significant. The LRF muscle was the only one that resulted in a significant group effect (p=0.006) and a non-significant time effect, thus the changes over time were not significant overall and the group differences were statistically significant. This was not attributed to an effect of the intervention but rather baseline imbalances which persisted.

4.3.2.2 Symptomatic Group – Short-term effects of L/S SMT on muscle activity

Table 4.7 shows the short-term effects of lumbar SMT on the post readings in the symptomatic group.

Table 4.7: ANOVA for short-term effect of symptomatic group

Symptomatic (Short Term Effects)							
Multivariate Test							
Muscle	Test	Effect	Value	F	Hypothesis DF	Error DF	Sig.
RHAM	Wilks' Lambda	Time	.788	2.824c	2.000	21.000	.082
		Time*Group	.930	.785c	2.000	21.000	.469
LHAM		Time	.645	5.785c	2.000	21.000	.010
		Time*Group	.992	.087c	2.000	21.000	.917
RRF		Time	.996	.046c	2.000	21.000	.955
		Time*Group	.832	2.123c	2.000	21.000	.145
LRF		Time	.958	.459c	2.000	21.000	.638
		Time*Group	.838	2.025c	2.000	21.000	.157
RVM		Time	.985	.162c	2.000	21.000	.852
		Time*Group	.899	1.176c	2.000	21.000	.328
LVM		Time	.985	.160c	2.000	21.000	.854
		Time*Group	.922	.883c	2.000	21.000	.428

Table 4.7 shows that there was no evidence of a treatment effect of the intervention in the symptomatic group for RHAM (p=.0469), LHAM (p=0.917), RRF (p=0.145). LRF (p=0.157), RVM (p=0.328) and LVM (p=0.428). As seen above, in the immediate effects of the symptomatic group, the LRF had a significant group effect (p=0.003) and a statistically insignificant time effect, thus the changes over time were non-significant overall but the group differences were significant. This too was attributed to persistent baseline imbalances.

The LHAM muscle displayed a significant time effect (p=0.010) but a non-significant group effect. This indicated that the changes in muscle activity over time were significant overall and the group differences were statistically insignificant.

4.3.3 Objective Three

To compare and correlate the data between the asymptomatic and symptomatic groups.

4.3.3.1 Symptomatic vs. Asymptomatic

Table 4.8: ANOVA for the comparison of the symptomatic and asymptomatic groups

Symptomatic and Asymptomatic							
Multivariate Test							
Muscle	Test	Effect	Value	F	Hypothesis DF	Error DF	Sig.
RHAM	Wilks' Lambda	Time	.853	3.711b	2.000	43.000	.033
		Time*Group	.900	2.387b	2.000	43.000	.104
		Time*CG	.978	.484b	2.000	43.000	.620
		Time*Group*CG	.975	.562b	2.000	43.000	.574
LHAM	Wilks' Lambda	Time	.809	5.087b	2.000	43.000	.010
		Time*Group	.929	1.633b	2.000	43.000	.207
		Time*CG	.860	3.493b	2.000	43.000	.039
		Time*Group*CG	.932	1.574b	2.000	43.000	.219
RRF	Wilks' Lambda	Time	.980	.441b	2.000	43.000	.646
		Time*Group	.873	3.126b	2.000	43.000	.054
		Time*CG	.986	.301b	2.000	43.000	.742
		Time*Group*CG	.973	.598b	2.000	43.000	.555
LRF	Wilks' Lambda	Time	.957	.977b	2.000	43.000	.385
		Time*Group	.933	1.544b	2.000	43.000	.225
		Time*CG	.943	1.311b	2.000	43.000	.280
		Time*Group*CG	.980	.435b	2.000	43.000	.650
RVM	Wilks' Lambda	Time	.933	1.554b	2.000	43.000	.223
		Time*Group	.951	1.117b	2.000	43.000	.336
		Time*CG	.959	.909b	2.000	43.000	.411
		Time*Group*CG	.985	.318b	2.000	43.000	.730
LVM	Wilks' Lambda	Time	.983	.366b	2.000	43.000	.696
		Time*Group	.999	.012b	2.000	43.000	.988
		Time*CG	.997	.055b	2.000	43.000	.947
		Time*Group*CG	.914	2.028b	2.000	43.000	.144

(CG=Clinical Group)

Table 4.8 summarises that there was no difference in the effect of the intervention between the symptomatic and asymptomatic clinical groups for the RHAM (*p*=0.574), LHAM (*p*=0.219), RRF (*p*=0.555), LRF (*p*=0.650), RVM (*p*=0.730) and LVM (*p*=0.144). For the RHAM muscle, there was a significant overall time effect (p=0.033) and a statistically insignificant clinical group effect (p=0.622) therefore it was shown that for this outcome everyone, regardless of clinical group and treatment group, experienced a significant increase to the same degree over time. For the LHAM muscle, there was a significant time x clinical group effect (p=0.039). This indicated that the response over time was dependent on the clinical group and not on the intervention. The LRF muscle displayed that there was no significant overall time effect but there was an overall significant treatment group effect (p=0.001), therefore it can only be concluded that, for this outcome, there were group differences which were not necessarily due to the intervention.

For the RRF, RVM and LVM muscles there was no difference in the effect of the intervention between the symptomatic and asymptomatic clinical groups nor treatment effects overall. It can only be concluded that for this outcome everyone, regardless of clinical group and treatment group, experienced an increase to the same degree over time. A lot of variability in the results and the small sample sizes made it difficult to conclude anything else.

4.4 Conclusion

This chapter summarises that within clinical groups there was evidence of change, with the symptomatic group displaying the majority of the significant change of the effects between the two groups. The symptomatic group was the only one to display evidence of a treatment effect of the lumbar SMT. This change, however did not persist into the short-term readings. There were no differences shown when comparing the symptomatic and asymptomatic groups that indicated that lumbar SMT had a significant effect on the quadriceps femoris and hamstring muscle groups in the immediate post and short-term readings. Therefore, the null hypotheses are retained.

CHAPTER FIVE: DISCUSSION

5.1 Introduction

This chapter involves the discussion of the results presented in Chapter Four, which include the demographic data and the results from the statistical analysis of sEMG readings of the muscle activity in the quadriceps femoris and hamstring muscle groups following lumbar spine manipulation. This chapter will discuss the results of this study in relation to the current literature that was summarised in Chapter Two.

5.2 Demographic Data

5.2.1 Age

This study included age as an inclusion criterion and was limited to individuals between the ages of 18 to 45 years. This age range was chosen to negate the necessity for parental consent and limit the chance of an individual with degenerative changes from participating in the study as the incidence rate of osteoarthritis increases exponentially in the lumbar spine and knee joints from the sixth and seventh decade of life (Yochum and Rowe 2005; de Schepper *et al.* 2010). Furthermore, the presence of degenerative changes in the lumbar spine and the knee joint can alter muscle activation of surrounding musculature, thus affecting the quality and reliability of the readings of muscle activity obtained during testing (Ding *et al.* 2005; Billot *et al.* 2010; Ansari *et al.* 2018). By reducing the sample population to the age ranges of 18 to 45 years it reduced the risk of obtaining poor sEMG readings.

When reporting on the mean age within the clinical groups, the symptomatic and asymptomatic group, Table 4.1 demonstrated that the mean age was 27.9 years and 24.4 years respectively. There was a marginally non-significant difference between the symptomatic group and asymptomatic group (p=0.058) as participants that were placed into the symptomatic group were slightly older than those in the asymptomatic. However, when reporting on the mean age within the experimental groups, the intervention group had a mean age of 27.3 years and the control 25.1 years. There was no significant difference in age (p=0.243) found between the intervention and control groups. This was comparable to studies conducted by Niazi *et al.* (2015) and

Grindstaff *et al.* (2009) where the mean age was 27.6 years and 28.3 years respectively. The results suggest that the four clinical sub-groups were homogenous in nature with negligible difference, thus making the effect of the intervention comparable.

5.2.2 Gender

The ability to perform repetitive tasks does not differ between genders, but it has been observed that the sEMG readings obtained during these tasks may differ between women and men (Arjunan *et al.* 2011). However, Liebensteiner *et al.* (2012) reported contradicting evidence that suggested that there were no significant differences in sEMG readings between genders when assessing the activation levels of various muscles, including the quadriceps femoris and hamstring muscle, during voluntary contractions. Therefore, participants of both genders were included in this study. There were no significant differences found in the distribution of the women and men between the symptomatic and asymptomatic groups, making the groups comparable. In the sample population, there was a greater percentage of female participants (54%) compared to male participants (46%). This is similar to previous studies that had the inclusion of both genders with a higher percentage in female participants (Suter *et al.* 2000; Dunning and Rushton 2009; Grindstaff *et al.* 2014). However, this varied from other studies where the percentage of male subjects was higher or the only gender included in the sample population (Herzog, Scheele and Conway 1999; Hillermann *et al.* 2006; Niazi *et al.* 2015).

5.3 Muscle Activity

5.3.1 Asymptomatic Group

Theories suggest that when SMT is applied to fixated joints it causes an alteration of inflow of sensory information that result in neurophysiological changes (Henderson 2012; Pickar and Bolton 2012). Dunning and Rushton (2009) reported that there was an increase in biceps brachii muscle activation following manipulation of the cervical spine. However, Fryer and Pearce (2012) found that lumbosacral manipulation resulted in a decrease in spinal excitability in an asymptomatic population when compared to the control group.

In this study, lumbar SMT did not result in any significant changes in the immediate or short-term readings of muscle activity of the quadriceps femoris and hamstring muscles in the asymptomatic participants. In previous studies, it had been observed that SMT of fixated spinal segments that share a neurological link to a muscle that is not directly attached to the spine could significantly alter muscle activity levels (Dunning and Rushton 2009; Fryer and Pearce 2012). It was anticipated that due to the neurological connection that the quadriceps femoris and hamstring muscles have to the L2-L5 levels of the lumbar spine, that applying SMT to any fixated segments in the relevant levels would lead to changes in the activation of these muscles. However, the findings of the current study contrast findings of previous studies but are rather in accordance with Cardinale *et al.* (2015) who observed that there were no significant differences in the sEMG readings of the paraspinal and gastrocnemius muscle in asymptomatic participants following SMT applied to the level of L5/S1.

Asymptomatic participants were included in the study to assess the effects of SMT without the added physiological consequences of pain. Grindstaff *et al.* (2014) suggested that the response of symptomatic participants to SMT would differ due to the neurophysiological consequences of pain which may include hyperesthesia, hypertonic muscles, muscle imbalances and increase fatigability of muscles. The use of an asymptomatic population was aimed at providing further research and evidence into the effects of SMT on fixated joints as well as providing a better understanding of how joint restrictions can affect normal neurophysiological functioning.

Grindstaff *et al.* (2009) suggested that when assessing the effects of SMT on individuals who are free from joint pathology, therefore considered asymptomatic, it is possible that alterations in the motor neuron excitability may not occur to the same extent as in studies that included symptomatic populations. As the use of an asymptomatic population in this study is based on findings that show that fixations can cause alterations in muscle activity, it would be important to consider other regions than just the lumbar spine. A limitation of this study is that no regional examination was performed of the hip and knee overlooking the possible effects of fixations within those regions on the muscle activity in the quadriceps and hamstring muscles due to their attachment proximity.

5.3.2 Symptomatic Group

The results of the study indicate that lumbar SMT may have an effect on quadriceps and hamstring muscle activity in symptomatic participants. Statistically significant time effects were identified within the symptomatic group in the RHAM (p=0.029) and LHAM (p=0.003) when comparing the intervention group to the control. However, after the 10-minute reading, the results show that the significant time effect only persisted in the LHAM (p=0.010). There was evidence of a treatment effect for the RRF (p=0.047) as there was a significant increase in muscle activity in the intervention compared to the decreasing muscle activity in the control group. However, this treatment effect in the RRF was no longer present after the 10-minute period.

Although there were no statistically significant differences in the other quadriceps femoris muscles, there was clear evidence of a clinical trend between the participants who received the intervention compared to the control group. The participants that received the intervention had an increase in muscle activity at the immediate post and 10-minute readings when compared to pre-intervention levels which occurred bilaterally. However, the control group showed an overall decrease in muscle activity readings in the RRF, LRF, RVM and LVM bilaterally. This decrease in activity occurred at the immediate post reading and persisted till the 10-minute reading. The current results are similar to those in studies performed by Suter *et al.* (2000) and Suter and McMorland (2002).

The improvement or increase of sEMG readings of the quadriceps muscle in the intervention group may indicate that lumbar SMT may reduce the effects of AMI. As the control did not receive any form of intervention, the effects of AMI may have persisted in the form of decreased activity and increased fatigability.

5.3.3 Comparison of The Symptomatic and Asymptomatic Groups

The results of this study have shown that lumbar SMT resulted in no statistically significant differences between the symptomatic and asymptomatic groups. However, the RHAM muscle had a significant time effect (p=0.033) and an insignificant group effect suggesting that everyone, regardless of whether they were in the symptomatic or asymptomatic group, showed a significant increase in muscle activity at immediate post and 10-minute readings. An overall significant treatment group effect (p=0.001)

was observed in the LRF muscle but with no significant overall time effect the significant differences identified between the symptomatic and asymptomatic clinical groups could not be definitively proven to be caused by the lumbar SMT. The results from comparing the clinical groups are similar to the findings of Currie *et al.* (2016) where it was observed that there were changes in muscle activity in both of the symptomatic and asymptomatic populations but the differences were not significant when comparing the findings between the two groups.

Pain is a complex outcome that is complicated to measure due to its subjective nature. There is an increasing need for more reliable outcome measures of pain to demonstrate the efficacy of various treatments. Currently, there are no methods for accurately quantifying an individual's perspective of their pain (Younger, McCue and Mackey 2009; Reed and Van Nostran 2014). In the current study, low back pain participants were included to allow for a comparison between different clinical groups. However, this study did not assess the subjectivity of the pain but rather focused on the physiological consequences that were present in the symptomatic participants. To include a more homogeneous sample, symptomatic participants had to rank their pain from a minimum of three to a maximum of six on the numerical pain rating scale. Therefore, symptomatic participants included in this study had similar pain ratings. This avoids including symptomatic participants that reported minimal pain and participants who may have reported intense pain with a high rating on the pain scale.

Body Mass Index (BMI) is an important factor to consider with sEMG readings. In this study, participants with a BMI of higher than 30 were excluded as it has been suggested that subcutaneous fat acts as an insulator between the electrodes and the muscles, thus altering the signal (Criswell 2011; Minetto *et al.* 2013). Therefore, subjects with a lower BMI that have a smaller distribution and thickness of subcutaneous fat may have higher readings than subjects with a BMI that's over 30. Nordander *et al.* (2003) used ultrasound imaging to determine the distance between the skin (where the sEMG electrodes were placed) and the desired muscle and found that participants with a higher BMI had a thicker layer of subcutaneous fat resulting in an increased distanced between the electrodes and muscle. This resulted in decreased EMG amplitude readings during both submaximal and maximal contractions. BMI can provide a rough estimate of the amount of subcutaneous fat but

skinfold measurement may allow for a more reliable way to quantify subcutaneous fat. The impact of skinfold thickness in this study is unknown but future studies should consider skinfold measurement as a form of exclusion criterion (Minetto *et al.* 2013).

5.4 Conclusion

This study demonstrated that lumbar SMT is able to affect muscle activity within the quadriceps femoris and hamstring muscles bilaterally. There were no statistically significant differences in the sEMG readings of the symptomatic and asymptomatic groups as both showed a similar increase in muscle activity to the same degree over time. Majority of the changes were noted in the symptomatic group with the results revealing a positive clinical trend that occurred specifically the quadriceps femoris muscles. However, with a larger population size of both clinical groups a significant positive effect may be seen. Thus, further investigation is warranted.

CHAPTER SIX: CONCLUSIONS, LIMITATIONS AND RECOMMENDATIONS

6.1 Conclusion

This study aimed to determine and compare the effects of a lumbar spine manipulation on the muscle activity in the quadriceps femoris and hamstring muscle groups in asymptomatic and symptomatic participants. Forty-eight participants were recruited for the study and were allocated into either the symptomatic or asymptomatic clinical group. Each clinical group consisted of 24 participants that were randomly allocated to an intervention or control group. The intervention group received lumbar spinal manipulation.

The results of this study show there were significant differences found when observing the clinical groups individually. However, the differences observed could not be definitively proven to be caused by, or be a direct result, of the intervention. The only significant treatment effect that was demonstrated occurred in the symptomatic group at the immediate post-reading and it was observed that this effect did not persist in the ten-minute reading. There was clear evidence of a clinical trend that presented in the quadriceps femoris muscle (rectus femoris and vastus medialis) of the symptomatic group, as the overall muscle activity of the participants who received the intervention increased bilaterally at the immediate post and ten-minute readings compared to the control group whose muscle activity decreased. However, these changes were not statistically significant. Although the majority of the significant differences and a significant treatment effect were identified in the symptomatic group, there was no evidence of a significant difference when comparing the two clinical groups. As a result, the null hypotheses were retained.

6.2 Limitations

The following limitations were identified during the course of this study:

1. The subcutaneous fat layer thickness above the quadriceps femoris and hamstring muscles in participants should be measure using other methods such us ultrasound imaging or with the use of Lean Thigh Volume test (LTV).

2. Concerning the sEMG electrode placement, although much effort was taken to ensure reproducibility, the exact sEMG electrode placement between participants could not be standardised or verified. 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.

6.3 Recommendations

- A larger sample size should be used in future studies in order to further examine the possible trend found in the symptomatic group of this study.
- Skin fold thickness should be controlled for in future studies and should be included as part of the participant inclusion criteria.
- Future studies should make use of a research assistant to deliver the intervention or conduct the sEMG study in order to remove any potential researcher bias.

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APPENDICES

Appendix A: Permission to use the DUT Chiropractic Day Clinic

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 : 23.08.2019

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

---

Permission is hereby granted to:

Ms Jenna-Leigh Parkes (Student Number: 21414969)

Research title: "The effect of lumbar spine manipulation on the muscle activity of the quadriceps femoris and hamstring muscle groups".

Ms Parkes, 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.

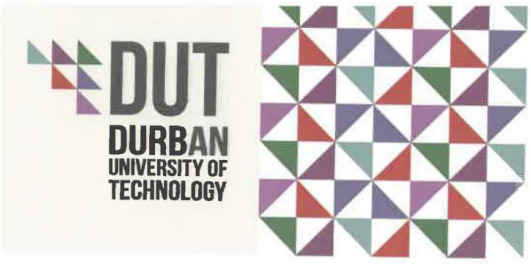
Kind regards

Dr L O'Connor
Head of Department:
Chiropractic

Dr Desiree Varatharajullu
Clinic Director: Chiropractic Day
Clinic: Chiropractic

Cc: Mrs Linda Twiggs: Chiropractic Day Clinic
Dr A. Abdul-Rasheed: Supervisor
Dr A. van der Meulen: Co-Supervisor

Appendix B: IREC Approval



Institutional Research Ethics Committee
Research and Postgraduate Support Directorate
2nd Floor, Berwyn Court
Gate 1, Steve Biko Campus
Durban University of Technology

P O Box 1334, Durban, South Africa, 4001

Tel: 031 373 2375
Email: lavishad@dut.ac.za
http://www.dut.ac.za/research/institutional_research_ethics

www.dut.ac.za

6 September 2019

Ms J-L Parkes
P.O. Box 838
Southport
4230

Dear Ms Parkes

The effect of lumbar spine manipulation on the muscle activity of the quadriceps femoris and hamstring muscle groups
Ethical Clearance number IREC 113/19

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

Dr M A Sathar
Deputy Chairperson: IREC

Appendix C: Pan African Clinical Trial Registration

27 January 2020

To Whom It May Concern:

RE: The effect of lumbar spine manipulation on the muscle activity of the quadriceps femoris and hamstring muscle groups

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 **PACTR202001586284982**.

Please be advised that your trial is registered under an initiative within our system that allow us to capture data of trials that are already in progress or completed. As such, your trial registration may not adhere to the mandates set forth by the International Committee of Medical Journal Editors for registration requirements, and it is your duty to be transparent to any journal that may ask about the retrospective status of your registration.

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.

Please note you are responsible for updating your trial, or for informing us of changes to your trial. 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 epienaar@mrc.ac.za should you have any questions.

Yours faithfully,

Elizabeth D Pienaar
www.pactr.org Project Manager
+27 021 938 0835

The South African Medical Research Council
Cochrane South Africa | PO Box 19070, Tygerberg, 7505
Tel: +27 (0)21 938 0438 | Email: cochrane@mrc.co.za | Web: www.southafrica.cochrane.org

Appendix D: Permission to conduct research at DUT

Directorate for Research and Postgraduate Support
Durban University of Technology
Tromso Annexe, Steve Biko Campus
P.O. Box 1334, Durban 4000
Tel.: 031-3732576/7
Fax: 031-3732946

21st August 2019

Ms Jenna-Leigh Parkes
c/o Department of Chiropractic
Faculty of Health Sciences
Durban University of Technology

Dear Ms Parkes

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 effect of lumbar spine manipulation on the muscle activity of the quadriceps femoris and hamstring muscle groups" at the Durban University of Technology.

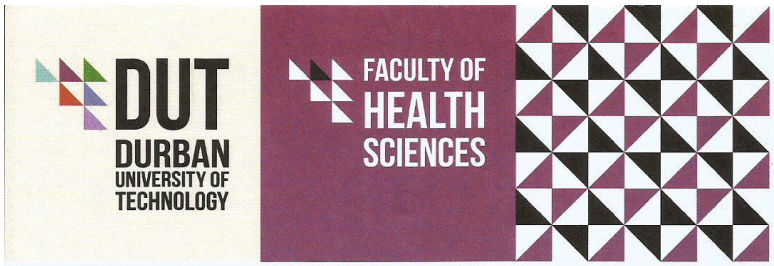
The DUT may impose any other condition it deems appropriate in the circumstances having regard to nature and extent of access to and use of information requested.

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

PROF KEVIN DUFFY
ACTING DIRECTOR: RESEARCH AND POSTGRADUATE SUPPORT DIRECORATE

Appendix E: Treatment Voucher



Department of Chiropractic
Chiropractic Clinic
Faculty of Health Sciences
Ritson Campus
Durban University of Technology

11 Ritson Road, Berea, Durban 4001

P O Box 1334, Durban, 4000, South Africa

Tel: (031)373 2205

www.dut.ac.za

Treatment Voucher

Date: _____

This is to certify that _____ , I.D. number _____

May receive one free treatment at the Durban University of Technology Chiropractic Day Centre.

Thank you for participating in the research.

Researcher: Jenna Parkes

_____

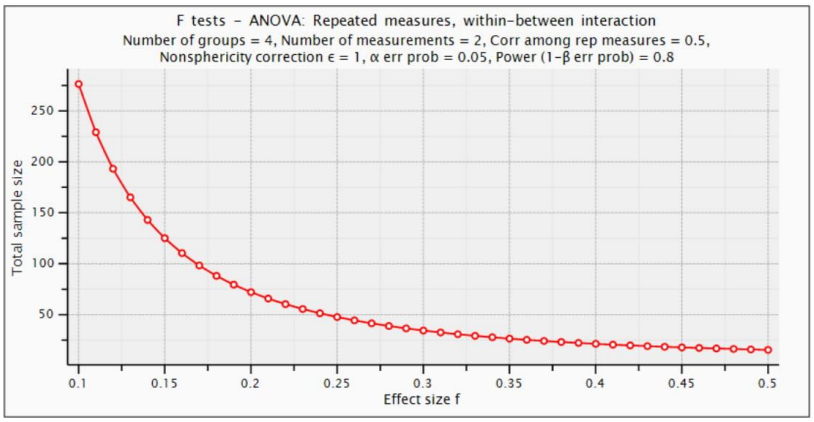
This voucher will be valid from the _____ to _____

DUT Chiropractic Clinic Stamp

Appendix F: Statistical analysis

Statistical Analysis

Using G*Power version 3.1.9.2 and assuming a moderate effect size of 0.25 for a repeated measures ANOVA with interaction, significance level of 0.05 and power of 80%, the estimated sample size required is four groups of 12 individuals (48 in total). The four groups are symptomatic intervention, symptomatic control, asymptomatic intervention, and asymptomatic control. Participants will be randomly allocated to one of each of the four groups.



Appendix G: Advertisement

DO YOU WANT TO TAKE PART IN EXCITING RESEARCH??

ARE YOU BETWEEN THE AGES OF 18 – 45?

A LOCAL RESEARCH STUDY IS LOOKING FOR PARTICIPANTS WITH AND WITHOUT LOWER BACK PAIN

YOUR LOWER BACK WILL BE ASSESSED AND YOU MAY RECEIVE TREATMENT!

IF YOU WOULD LIKE TO PARTICIPATE IN A STUDY THAT LOOKS AT CORRECTING MUSCLE IMBALANCES PLEASE CONTACT THE FOLLOWING:

JENNA PARKES: 0732971602

DUT CHIROPRACTIC DAY CLINIC: 031 373 2205

Appendix H: Case history

DUT DURBAN UNIVERSITY OF TECHNOLOGY — DEPARTMENT OF CHIROPRACTIC

CHIROPRACTIC DAY CLINIC CASE HISTORY

Patient: _____ Date: _____

File #: _____ Age: _____

Gender: _____ Occupation: _____

Student: _____ Signature _____

FOR CLINICIANS USE ONLY:

Initial visit

Clinician: Signature:

Case History:

Examination:
Previous: Current:

X-Ray Studies:
Previous: Current:

Clinical Path. lab:
Previous: Current:

CASE STATUS:

PTT:	Signature:	Date:

CONDITIONAL:
Reason for Conditional:

Signature: Date:

Conditions met in Visit No:	Signed into PTT:	Date:

Case Summary signed off: Date:

Student's Case History:

1. **Source of History:**

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

3. **Present Illness:**

	Complaint 1(principle complaint)	Complaint 2 (additional or secondary complaint)
Location		
Onset : Initial:		
Recent:		
Cause:		
Duration		
Frequency		
Pain (Character)		
Progression		
Aggravating Factors		
Relieving Factors		
Associated S & S		
Previous Occurrences		
Past Treatment		
Outcome:		

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 life-style:

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

	Noted	Family member		Noted	Family member
Alcoholism			Headaches		
Anaemia			Heart Disease		
Arthritis			Kidney Disease		
CA			Mental Illness		
DM			Stroke		
Drug Addiction			Thyroid Disease		
Epilepsy			TB		
Other (list)					

8. Psychosocial history:

Home Situation and daily life
Important experiences
Religious Beliefs

Appendix I: Physical examination

DUT DURBAN UNIVERSITY OF TECHNOLOGY

DEPARTMENT OF CHIROPRACTIC AND SOMATOLOGY

CHIROPRACTIC PROGRAMME

PHYSICAL EXAMINATION: SENIOR

Patient Name: _____			**File no:** _____	**Date:** _____
Student:		**Signature:**		
VITALS:				
Pulse rate:			Respiratory rate:	
Blood pressure:	R	L	Medication if hypertensive:	
Temperature:			Height:	
Weight:	Any recent change?	Y / N	If Yes: How much gain/loss	Over what period

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

SYSTEM SPECIFIC EXAMINATION:
CARDIOVASCULAR EXAMINATION
RESPIRATORY EXAMINATION
ABDOMINAL EXAMINATION
NEUROLOGICAL EXAMINATION
COMMENTS
Clinician: **Signature:**

Page 1

Appendix J: Lumbar regional exam

DUT DURBAN UNIVERSITY OF TECHNOLOGY

DEPARTMENT OF CHIROPRACTIC AND SOMATOLOGY

CHIROPRACTIC PROGRAMME

REGIONAL EXAMINATION
LUMBAR SPINE AND PELVIS

Patient: _____ File#: _____ Date: _____

Student: _____ Clinician: _____

STANDING:

Posture– scoliosis, antalgia, kyphosis
Body Type
Skin
Scars
Discolouration

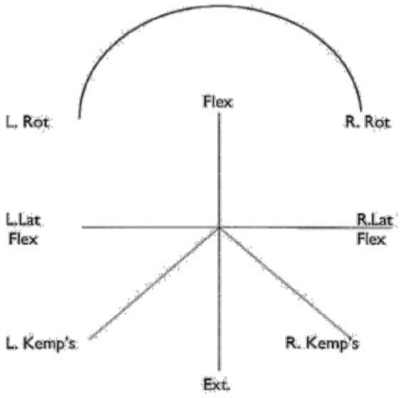
Minor's Sign
Muscle tone
Spinous Percussion
Schober's Test (6cm)
Bony and Soft Tissue Contours

GAIT:

Normal walking
Toe walking
Heel Walking
Half squat

ROM:

Forward Flexion = 40-60° (15 cm from floor)
Extension = 20-35°
L/R Rotation = 3-18°
L/R Lateral Flexion = 15-20°



Which movement reproduces the pain or is the worst?

- Location of pain
- Supported Adams: Relief? (SI)
- Aggravates? (disc, muscle strain)

SUPINE:

Observe abdomen (hair, skin, nails)
Palpate abdomen\groin
Pulses - abdominal
- lower extremity
Abdominal reflexes

		Degree	LBP?	Location	Leg pain	Buttock	Thigh	Calf	Heel	Foot	Braggard
SLR	L										
	R										

	L	R
Bowstring		
Sciatic notch		
Circumference (thigh and calf)		
Leg length: actual -		
apparent -		
Patrick FABERE: pos\neg – location of pain?		
Gaenslen's Test		
Gluteus max stretch		
Piriformis test (hypertonicity?)		
Thomas test: hip \ psoas \ rectus femoris ?		
Psoas Test		

SITTING:

Spinous Percussion
Lhermitte

Valsalva

Page 1 of 4

TRIPOD SI, +, ++		Degree	LBP?	Location	Leg pain	Buttock	Thigh	Calf	Heel	Foot	Braggard
	L										
	R										

SLUMP 7 TEST											
	L										
	R										

LATERAL RECUMBENT:	L	R
Ober's		
Femoral n. stretch		
SI Compression		

PRONE:	L	R
Gluteal skyline		
Skin rolling		
Iliac crest compression		
Facet joint challenge		
SI tenderness		
SI compression		
Erichson's		
Pheasant's		

MF tp's	Latent	Active	Radiation
QL			
Paraspinal			
Glut Max			
Glut Med			
Glut Min			
Piriformis			
Hamstring			
TFL			
Iliopsoas			
Rectus Abdominis			
Ext/Int Oblique muscles			

NON ORGANIC SIGNS:

Pin point pain
Trunk rotation
Flip Test
Ankle dorsiflexion test
Axial compression
Burn's Bench test
Hoover's test
Repeat Pin point test

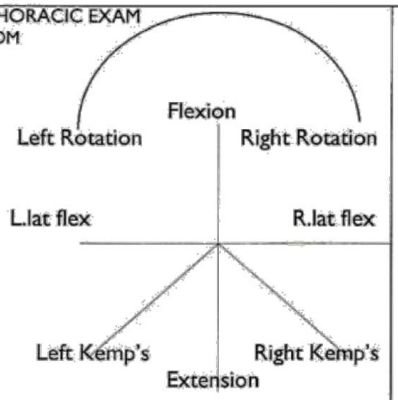
NEUROLOGICAL EXAMINATION

Fasciculations						
Plantar reflex						
level	Tender?	Dermatomes		DTR		
		L	R		L	R
T12				Patellar		
L1				Achilles		
L2						
L3				Proprioception		
L4						
L5						
S1						
S2						
S3						

MYOTOMES

Action	Muscles	Levels	L	R	
Lateral Flexion spine	Muscle QL				
Hip flexion	Psoas, Rectus femoris				5+ Full strength
Hip extension	Hamstring, glutes				4+ Weakness
Hip internal rotation	Glutmed, min, TFL, adductors				3+ Weak against grav
Hip external rotation	Gluteus max, Piriformis				2+ Weak w\o gravity
Hip abduction	TFL, Glut med and minimus				1+ Fascic w\o gross movt
Hip adduction	Adductors				0 No movement
Knee flexion	Hamstring,				
Knee extension	Quad				W - wasting
Ankle plantarflexion	Gastrocnemius, soleus				
Ankle dorsiflexion	Tibialis anterior				
Inversion	Tibialis anterior				
Eversion	Peroneus longus				
Great toe extensor	EHL				

BASIC THORACIC EXAM
Passive ROM



History :

Orthopedic assessment:

BASIC HIP EXAM

History
ROM: Active
Passive: Medial rotation: A) Supine (neutral) If reduced
- hard \ soft end feel
B) Supine (hip flexed):
- Trochanteric bursa

Appendix K: SOAPE Note

DUT DURBAN UNIVERSITY OF TECHNOLOGY | DEPARTMENT OF CHIROPRACTIC AND SOMATOLOGY

CHIROPRACTIC PROGRAMME

Patient Name:	***File number:*** ***Page:***
Date: ***Visit:*** ***Student:***	
Attending Clinician:	***Signature:***
S: *Numerical Pain Rating Scale (Patient)* *Least* ***0 1 2 3 4 5 6 7 8 9 10*** *Worst*	*Student Rating* ☐ ***A:***
0:	***P:***
	E:
Special attention to:	***Next appointment:***
EBCC References:	
Date: ***Visit:*** ***Student:***	
Attending Clinician:	***Signature:***
S: *Numerical Pain Rating Scale (Patient)* *Least* ***0 1 2 3 4 5 6 7 8 9 10*** *Worst*	*Student Rating* ☐ ***A:***
O:	***P:***
	E:
Special attention to:	***Next appointment:***
EBCC References:	

Appendix L: Informed consent

Informed Consent

Statement of Agreement to Participate in the Research Study:

• I hereby confirm that I have been informed by the researcher, _____(name of researcher), about the nature, conduct, benefits and risks of this study - Research Ethics Clearance Number:

• 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**

I, Jenna-Leigh Parkes (name of the researcher) herewith confirm that the above participant has been fully informed about the nature, conduct and risks of the above study.

_____	_____	_____
Full Name of Researcher	**Date**	**Signature**
_____	_____	_____
Full Name of Witness (If applicable)	**Date**	**Signature**
_____	_____	_____
Full Name of Legal Guardian (If applicable)	**Date**	**Signature**

Appendix M: Letter of information

LETTER OF INFORMATION

Title of the Research Study: The effect of lumbar spine manipulation on the muscle activity within the quadriceps femoris and hamstring muscle groups.

Principal Investigator/s/researcher: Jenna-Leigh Parkes (B: Tech Chiropractic)

Co-Investigator/s/supervisor/s: Dr Ashura Abdul-Rasheed (M: Tech Chiropractic)

Brief Introduction and Purpose of the Study: The aim of this study is to determine the effects of a lower back manipulation on the activity within the quadriceps femoris and hamstring muscles in symptomatic and asymptomatic participants. Forty-eight people will be required to complete this study.

Outline of the Procedures:

If you have agreed to participate in the study, you will be required to sign an informed consent form. The researcher will take a case history followed by a physical examination and lumbar spine regional exam. This will help the researcher to determine if you meet the inclusion criteria and are eligible to take part in the study. If you meet the necessary requirements, you will be placed into your relevant group (i.e. participant with back pain will out into the symptomatic group, and a participant with no back pain will be placed in the asymptomatic group). Once placed in to a group you will be placed into either the treatment or control subgroup. You will be required to remove clothing that may cover the lower back and upper legs, however appropriate clothing will be provided (shorts and robes). For the EMG reading it may be required that the areas where the electrodes will be placed may need to be shaven if there is hair. Baseline measurements for the surface EMG will need to be obtained by the researcher. The treatment/procedure will then be administered according to which subgroup you were placed. EMG readings will then be taken again immediately after treatment and then again after ten minutes.

Risks or Discomforts to the Participant: After treatment you may experience slight, transient stiffness, but this should improve after 24 hours. If the stiffness continues, please contact me.

Benefits: You may receive a voucher for one free treatment at the DUT Chiropractic Day Clinic which will need to be used within three months from the date of participation.

Reason/s why the Participant May Be Withdrawn from the Study: If you do not present with a fixation in the lumbar spine you may be withdrawn from the study. If you do not meet the required inclusion criteria or have received manipulative therapy within four weeks prior to the research appointment then you may be excluded from the study. If there are reasons to which the researcher

cannot apply the necessary equipment to your skin (i.e. open wound, rash, or any other contra-indication) then you be withdrawn from the study. If at any point you would like to withdraw from the study, you may do so with no questions asked.

Costs of the Study: As a participant you will not be expected to contribute to any cost of the study. The only requirement will be to attend one appointment that may take up to two to three hours.

Confidentiality: Any information given by you will only be available to the researcher, the supervisor and the co-supervisor.

Research-related Injury: If there are any adverse reactions the researcher will report this to IREC.

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

Please contact the researcher (0732971602), the supervisor Dr Ashura Abdul-Rasheed (031 373 2102) or the Institutional Research Ethics administrator on 031 373 2375. Complaints can be reported to the DVC: Research, Innovation and Engagement Prof S Moyo on 031 373 2577 or moyos@dut.ac.za.

Appendix N: Permission for advertisement placement

Permission for Advert Placement

Dear Sir/Madam

I am currently registered at the Durban University of Technology, working on my Master's degree in Technology Chiropractic. I would like permission to place an advertisement on your premises in order to recruit participants for my research project.

Please find advertisement attached.

Yours sincerely,

Jenna-Leigh Parkes

Researcher

I, _____give permission for an advert to be placed on the premises.

Signed _____ Date _____