The effect of a unilateral sacroiliac joint manipulation on muscle activity and force output in the posterior oblique sling muscles

By

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I, Aimee Paige McNally, 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 Father, Who created the complexities of the human body, and allowed me the privilege of studying His most intricate and beautifully crafted handiwork. Thank you for instilling within me a passion for health, and blessing me with hands that can facilitate healing. May I never forget where my ability comes from.

To my parents, who have encouraged me hard and loved me harder. Your support not only in my studies, but in the building of my character over these sometimes difficult years, has shaped me into not only the woman, but the Chiropractor I now am. I am forever grateful for your willing sacrifices; I love you endlessly.

When you pass through the waters,
I will be with you;
and when you pass through the rivers,
they will not sweep over you.
When you walk through the fire,
you will not be burned;
the flames will not set you ablaze.
Isaiah 43:2

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ABSTRACT

Objective: The effects of spinal manipulation have been well documented, however, current literature poses a gap regarding the neurophysiological mechanisms responsible for these effects. Further evidence is required in order to uncover the specific neurophysiological mechanisms of spinal manipulative therapy (SMT) and its effect on muscle activity. The study aimed to investigate the immediate effect of a unilateral sacroiliac joint (SIJ) manipulation compared to a control on muscle activity (EMG in mV) and maximum voluntary force output (dynamometer in kg) in the posterior oblique sling (POS) muscles in asymptomatic participants.

Methods: A randomised, controlled, pre-test, post-test design allowed for 34 participants, aged 18-45 years old, with joint dysfunction at the SIJ to be allocated to either a manipulation or a control group. Force output and muscle activity of the gluteus maximus (GM) and latissimus dorsi (LD) muscles were measured before and after the intervention. IBM SPSS was used to analyse the data with significance set at (p=0.05). Independent samples *t*-tests were used to determine significance within, and between, the groups, and Pearson correlation analysis looked for correlations between the muscles in the two slings.

Results: There were no significant differences observed between the control and intervention groups for age (p=0.355), gender (p=0.688), race (p=0.338), BMI (p=0.142), and the side of joint fixation (p=0.473). The intra-group analysis and inter-group analysis showed no significant differences for peak amplitude and mean muscle activity of the muscles of the POS when assessed for intra- or intergroup comparisons. A significant difference between pre and post maximum force output in the right GM for both groups (intervention: p=0.016; control: p=0.030), and in the right LD for the control group only (p=0.032), was noted. However, there was no significant difference between group results for any muscle in terms of force output when assessed for intergroup comparisons.

Conclusion: The results of this study showed no treatment effect of SIJ manipulation on the muscles of the POS in asymptomatic participants. Consideration should be

given to the way SMT is administered, the type of sham intervention used and the protocol followed to elicit maximum voluntary isometric contraction (MVIC).

Key indexing terms: spinal manipulative therapy, muscle activity, force output, posterior oblique sling.

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LIST OF ABBREVIATIONS

Ach	:	Acetylcholine
AP	:	Action potential
ASIS	:	Anterior superior iliac spine
ATP	:	Adenosine triphosphate
BMI	:	Body mass index
CLBP	:	Chronic low back pain
cm	:	Centimeters
CNS	:	Central nervous system
DUT	:	Durban University of Technology
EMG	:	Electromyography
GM	:	Gluteus maximus
GTO	:	Golgi tendon organ
HVLA	:	High-velocity low-amplitude
Hz	:	Hertz
Kg	:	Kilograms
LBP	:	Low back pain
LD	:	Latissimus dorsi
m	:	Meters
mm	:	Millimeters
ms	:	Milliseconds
mVs	:	Microvolts
MA	:	Muscle activity
MEPs	:	Motor evoked potentials
MLBP	:	Mechanical low back pain
MS	:	Muscle spindle
MVIC	:	Maximum voluntary isometric contraction
n	:	Sample number
N	:	Newton’s
NMJ	:	Neuromuscular junction
POS	:	Posterior oblique sling
PSIS	:	Posterior superior iliac spine

PS	:	Paraspinal muscles
RMS	:	Root mean square
ROM	:	Range of motion
SC	:	Spinal cord
SD	:	Standard deviation
sEMG	:	Surface electromyography
SI	:	Sacroiliac
SIJ	:	Sacroiliac joint
SM	:	Spinal manipulation
SMT	:	Spinal manipulative therapy
TLF	:	Thoracolumbar fascia
VC	:	Vertebral column

LIST OF DEFINITIONS

Afferent nerve: A sensory nerve conveying impulses from the periphery to the central nervous system (CNS) (Venes 2017).

Anisomelia: Leg length discrepancy (Kiapour *et al.* 2012).

Asymptomatic: Without symptoms (Venes 2017).

Body mass index (BMI): BMI is an index of body weight in relation to height. It is calculated as follows: weight in kilograms (kg)/height in metres squared (m^2). A BMI between 18.5 kg/m^2 and 24.9 kg/m^2 is defined as a healthy body weight as individuals within this range have the lowest health risks. BMI correlates well with body fat, but it is not actually a measure of body fat (Organization 2000).

Efferent nerve: A nerve conveying impulses from the brain or CNS towards the periphery (Venes 2017).

Facilitation: The enhancement or reinforcement of a reflex or other nervous activity by the arrival of excitatory impulses at the reflex centre (Stedman 2000).

Golgi tendon organs: Nerve endings located within the tendons near the musculotendonous junction that are stimulated by tension applied onto the tendon and results in relaxation of the muscle belly (Haines 2012).

Inhibition: The limitation or depression of reflex activity (Venes 2017).

Isometric contraction: A muscular contraction in which there is a change in muscle tension but not in muscle length (Venes 2017).

Joint fixation/ dysfunction: A biomechanical spine derangement (characterized by joint hypo-mobility) thought to produce clinically significant effects by disturbing neurological function (Henderson 2012).

Joint manipulation/SMT (spinal manipulative therapy): A manual procedure that involves a directed thrust to move a joint past normal physiological range of motion without exceeding the anatomical limit. It is commonly associated with an audible 'crack' or cavitation (Bergmann and Peterson 2010; Hawk 2017) .

Mechanoreceptors: Mechanically sensitive neurons found within the joint's structure and surrounding tissues (Haines 2012).

Motion palpation: A procedure in which the hands are used to assess the active and passive segmental joint range of motion (Bergmann and Peterson 2010)

Motor neuron: Neural structures that cause muscles to contract or glands to secrete by the impulses generated and transmitted by them (Venes 2017)

Motor unit: The cell body and dendrites of a motor neuron, the multiple branches of its axon, and the muscle fibre that it innervates (Konrad 2005).

Muscle inhibition: The inability to fully activate a muscle due to ongoing reflex inhibition (Suter and McMorland 2002a; Venes 2017).

Muscle spindles: Nerve endings and intrafusal muscle fibres of the muscle belly that are stimulated by stretching of the muscle belly (Haines 2012).

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).

Placebo: A method used as an inactive control as a test of a treatment that is suspected of being useful in the treatment of a certain condition (Venes 2017).

Proprioception: Awareness of posture, movement and equilibrium together with understanding of position, weight and the resistance of objects in relation to the human body (Venes 2017).

Sensorimotor integration (SMI): The process by which the nervous system coordinates incoming sensory information from different parts of the body and integrates with the motor system to control movement (Haavik and Murphy 2012).

Sensory Neuron: Neural structures capable of transmission of afferent information or sensation (Venes 2017).

Surface electromyography: An electrical, non-invasive, accurate method of measuring muscle excitation and activation through the placement of electrodes over the muscle being assessed (Criswell 2010). It records myoelectric signals which are formed by physiological variations in muscle fibre membranes (Konrad 2005)

CHAPTER ONE

1.1 Introduction

Joint manipulation is proposed to alter sensorimotor processing, bringing about neurophysiological changes in the joint and surrounding muscles (Haavik and Murphy 2012). Little is understood about the effects and mechanisms of a manipulation, however the three main theories proposed include: biomechanical, neurophysiological and muscular reflexogenic theories; all of which seem to have neurophysiological effects (Keller and Colloca 2000; Bicalho *et al*. 2010; Jones 2014). Spinal manipulative therapy is commonly used in musculoskeletal disorders. Its effect on local muscle tightness is documented well (Harvey and Descarreaux 2013; Nougarou *et al.* 2013), however not much is known in terms of its effect on distal muscles that have a related function such as the POS muscles.

The POS, consisting of the gluteus maximus and contralateral latissimus dorsi muscles and the thoracolumbar fascia between them, plays a major role in the stability of the trunk in extension and rotation during the gait cycle (Page, Frank and Lardner 2010; Shin, Kim and Yoo 2013). It is purported to assist with transmitting forces from the lower limb to the upper limb. This aids in reducing injury in the lumbopelvic hip complex (Shin, Kim and Yoo 2013).

The sacroiliac joint (SIJ) is an integral joint of the pelvis where it functions in load transfer during the gait cycle (Liebenson 2004). It has an intimate relationship with the gluteus maximus, a component of the POS (Vleeming *et al.* 2012). Thus any alterations to the mobility of the SIJ, such as joint dysfunction, can negatively affect the POS. This may result in fatigue of the sling muscles and abnormal recruitment patterns.

Kim, Yoo and Choi (2013) demonstrated that muscle imbalances in the erector spinae and hamstring muscles are linked to low back pain. Therefore, these resultant muscle imbalances may cause a decrease in performance (strength/endurance) of the POS and predispose a person to lower back pain. Mechanical low back pain is one of the most common musculoskeletal conditions, with a lifetime prevalence of 60 to 85% (Krismer and van Tulder, 2007). Much research has been done to find effective

treatment for mechanical low back pain but, due to its high prevalence, it has become one of the most expensive conditions to manage (Dagenais, Caro and Haldeman, 2008; Bell and Burnett, 2009). Therefore, determining ways to improve trunk stability and prevent the initial onset of low back pain has become a high priority in research; hence the use of asymptomatic participants in this study. Haavik and Murphy (2012) said that the relationship between neural markers of altered function, and the development and maintenance of musculoskeletal pain is a “promising area in need of further research”.

To determine the effect on muscle activation attributed to manipulation, this research will investigate the resultant effects of manipulation on muscle activity in the POS pre- and post-manipulative therapy and the related effect on force output in asymptomatic participants.

Surface electromyography was used to monitor muscle activity as it has been shown to be the most valid and reliable measuring apparatus for muscle activity (Biopac Systems Inc, 2015; Cram 2011; Hof 1984) and has been used in a large number of studies (Dunning and Rushton; Keller and Colloca 2000; Niazi et al. 2015). A dynamometer was used to quantify the muscle force output during the MVIC of the POS in order to reduce the effect of the changeable resistance provided by the researcher, which may alter sEMG readings. The dynamometer has been shown to be reliable for testing the strength of both the gluteus maximus (hip extension) and latissimus dorsi (shoulder extension) muscles (Thorborg et al. 2010; Çelik, Dirican and Baltaci 2012).

This study has helped to determine whether spinal manipulation can alter the muscle activity in this group of muscles that are theorised to have a relationship; therefore enhancing the body of knowledge supporting SMT as a valuable tool for the maintenance of the biomechanics of the musculoskeletal system.

1.2 Study aims, objectives and hypotheses

1.2.1 The aim of the study

The aim of this study was to determine the effect of a unilateral SIJ manipulation on the muscle activity (mean and peak amplitude) and force output of muscles in the POS (gluteus maximus and latissimus dorsi) compared to a control intervention in asymptomatic participants.

1.2.2 Study objectives

Objective One
To determine the effect of a unilateral SIJ manipulation on the muscle activity and force output of the POS muscles during maximum voluntary contraction measured before and after the intervention.

Objective Two
To determine the effect of a control on the muscle activity and force output of the POS muscles during maximum voluntary contraction measured before and after the control.

Objective Three
To compare the muscle activity and force output readings of the POS muscles between the control and intervention groups.

1.2.3 Hypotheses

1.2.3.1 Alternative hypothesis

Ho: There will not be a significant difference ($p>0.05$) between the group receiving SMT and the control group, in terms of peak amplitude and mean muscle activity and peak force output of the gluteus maximus and latissimus dorsi muscles.

1.2.3.2 Null hypothesis

Ha: There will be a significant difference ($p<0.05$) between the group receiving SMT and the control group, in terms of peak amplitude and mean muscle activity and peak force output of the gluteus maximus and latissimus dorsi muscles.

1.3 Study Rationale

The POS is biomechanically connected to the SIJ (through the thoracolumbar fascia) (Joseph *et al.* 2014; Feeney *et al.* 2018). Triano (2005) found that dysfunctional joints may cause muscle imbalances through stimulation of associated mechanoreceptors, Thus, if the SIJ is dysfunctional it may affect the POS as fixation of the SIJ may stimulate mechanoreceptors, and cause reflex inhibition or facilitation of the muscles of the POS. Inversely, poor postural habits may result in abnormalities of lumbar musculature (O'Sullivan et al. 2002), resulting in SIJ dysfunction.

Even though this study is observing the effects of SMT on asymptomatic people, it is important to note that Kim, Kang and Oh (2014) demonstrated that women with chronic low back pain (CLBP) had greater activity in the POS than those without CLBP when performing a prone hip extension. Therefore, symptomatic dysfunction may cause increased muscle activity (MA) in the gluteus maximus (GM) and opposite latissimus dorsi (LD). Fryer and Pearce (2012) showed that SIJ SMT applied to asymptomatic participants produced a decrease in corticospinal and spinal reflex excitability, suggesting that SMT could cause reflex inhibition of the muscles associated with the SIJ. Potter, McCarthy and Oldham (2005) discussed that muscle imbalances can predispose the body to pain, therefore muscle imbalances of the POS may predispose a person to low back pain. From this literature one can deduce; bad posture can cause abnormally functioning POS muscles which can cause SIJ dysfunction, and SIJ fixations can result in abnormal POS functioning (thereby leading to CLBP). Therefore, there is importance in finding an appropriate treatment modality that will optimize POS muscle functioning, so as to prevent LBP.

Chiropractors and physiotherapists utilize SMT to bring about tissue healing and recovery from pain episodes (Pickar 2002). Literature to date suggests that there are significant changes in the function of local and distal musculature in the short-term post SMT. Although recent studies discuss the neurophysiological effects of SMT extensively, the mechanisms through which it is achieved are not well understood (Bialosky *et al.* 2009; Haavik and Murphy 2012). The results of this study may provide potential evidence and rationales in understanding SMT and its neurophysiological effects on muscle activity (expressed as motor unit recruitment) and the generation of

force. Thus, understanding how manipulation may affect muscle imbalances will contribute to the growing body of knowledge relative to this treatment modality.

1.4 Delimitations of the study

In clinical practice SMT is seldom used as a “once off” treatment, but rather as a series of treatments applied at various appointments (Hawk 2017). In the context of this research SMT was only applied once in order to determine the effect of a single SMT on the muscle activity and strength in the POS.

This study was conducted on asymptomatic participants to remove the influence of pain on the neuromuscular system, and to observe the changes already occurring in muscles associated with a fixated joint.

The inclusion criteria for this study were limited to participants between the ages of 18 - 45 years whom had a SIJ joint fixation, to allow for a homogenous sample at lower risk for any potential degenerative disorders associated with advanced age.

1.5 Flow of dissertation

Chapter One provides an introduction to the study and outlines the aims, objectives and hypotheses. It provides a rationale for the study based on the literature.

Chapter Two provides a brief review of the related anatomy, followed by a review of the literature relevant to spinal manipulation and how it is proposed that it brings about its effect. The role of the POS and its relationship to back pain will be discussed highlighting the need for the study.

Chapter Three details the methodology used to achieve the aims and objectives of this study. The study design, population and sampling strategy, measurement tools and data analysis techniques and ethical issues will be described.

Chapter Four presents the results of this study in tables and figures.

Chapter Five provides an analysis and discussion of the results in relation to current literature.

Chapter Six will conclude the study, making recommendations for future research and discuss the study limitations.

CHAPTER TWO

2.1 Introduction

Trunk stability and effective load transmission from the spine to the limbs and limbs to the spine is very important in order to protect the lumbopelvic-hip complex from injury (Page, Frank and Lardner 2010). Joint dysfunction in this area can affect the normal kinematics of this region and lead to arthrogenic muscle inhibition. This results in altered muscle relationships and further dysfunction. Joint manipulation can address these abnormal aberrations but further research is required to fully understand its effects (Suter and McMorland 2002a; Niazi *et al.* 2015; Lelic *et al.* 2016).

This chapter will begin by presenting an overview of the anatomy and biomechanics of the SIJ and muscles of the POS. This will be followed by a review of the literature related to the joint manipulation. The following sources were searched for information relevant to the study: Google Scholar, Summon, Ebscohost, PubMed, Medline, eMedicine, and the Durban University of Technology Institutional Repository.

Key terms used included: “posterior oblique sling”, “sacroiliac joint manipulation”, “theories of joint manipulation”, “surface electromyography”, “mechanisms of manipulation”, “neurophysiology of manipulation”, “effects of manipulation”, “arthrogenic muscle inhibition”, “gluteus maximus”, “latissimus dorsi”, “dynamometer”, “strength versus muscle activity”, “biomechanics of sacroiliac joint”, “leg length discrepancy”, “neuroanatomy”

2.2 The sacroiliac joint

2.2.1 Bony anatomy, ligaments, and innervation

The pelvis is made up of three bones which are fused together - the ilium, ischium, and pubis. The sacrum is a wedge - or triangular-shaped bone formed by the fusion of five sacral vertebrae. The sacroiliac joint connects the sacrum and ilium as a synovial joint on its anterior portion, and as a syndesmosis on its posterior portion (Moore, Dalley and Agur 2014). This has been termed a ‘diarthro-amphiarthrosis’, i.e. a joint that

possesses characteristics of both an ossified joint (synarthrosis) and a freely mobile joint (diarthrosis) (Vleeming *et al.* 2012).

The auricular surfaces of the synovial joint have irregular but congruent depressions and elevations which interlock – thus limiting mobility within the joint, and hence making the SIJ an atypical synovial joint. This allows the SIJs to play a large role in the transmission of the weight of the body to the hip bones as they form the strong, stable link between the axial and appendicular skeletons (Moore, Dalley and Agur 2014). Analysis of gait mechanics demonstrates that the SIJs provide sufficient flexibility for the intra-pelvic forces to be transferred effectively to and from the lumbar spine and lower extremities (Vleeming *et al.* 2012), highlighting its important role in load transmission. The sacroiliac joint can be a source of low back pain (Center, Springs and Center 2009).

Some of the strongest ligaments in the body are found around this joint, as seen in Figure 1 and detailed in Appendix A.

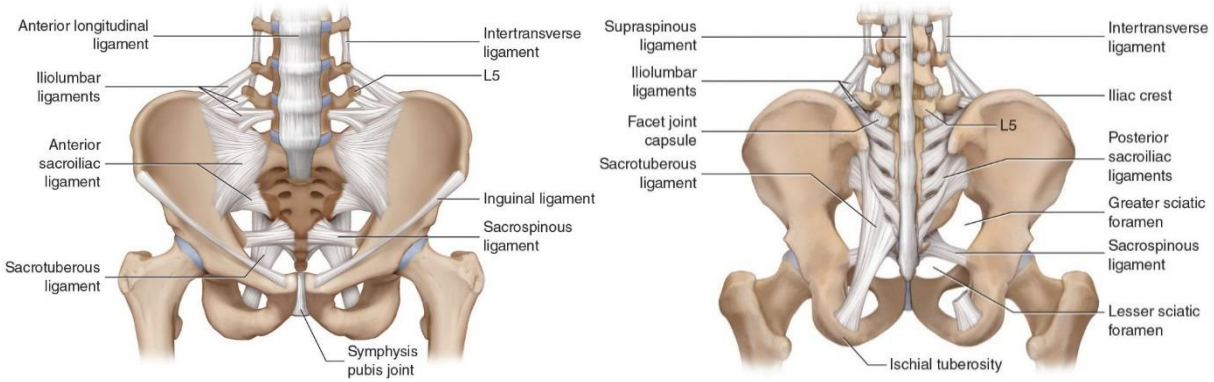


Figure 1 Anterior and posterior ligaments of the lumbro-sacral region (Muscolino 2014)

There is dispute about the innervation of the sacroiliac joint, however several authors (Patel *et al.* 2012; Umimura *et al.* 2012; Vleeming *et al.* 2012) agree that the outer border of the joint receives its innervation from the posterior rami of the lower lumbar and upper sacral segments.

2.2.2 Movements of the SIJ

The SIJ moves up to 1.6mm in translation and up to 4 degrees in rotation (Feeney *et al.* 2018), this movement is termed nutation or counter-nutation. When there is dysfunction in the SIJ, one of these motions can become restricted and can result in pain (Henderson 2012).

2.2.2.1 Nutation

Anterior sacral rotation relative to the ilium has been termed 'nutation' (Vleeming *et al.* 2012). This occurs during trunk flexion or hip extension, whereby the sacral promontory moves anteriorly and inferiorly, as demonstrated in Figure 2 (A) (Lippert 2011). Sacral nutation is increased in load-bearing situations (e.g. sitting/ standing), and is crucial for the optimisation of force closure as it prepares the pelvis to withstand the increasing load (Vleeming and Schuenke 2019). Upon loading, the dorsal SIJ ligaments begin to tense – resulting in the posterior aspects of the ilia being pressed together, increasing SIJ compression (Vleeming and Schuenke 2019).

2.2.2.2 Counter-nutation

Posterior sacral rotation relative to the ilium, as illustrated in Figure 2 (B) has been termed 'counternutation' (Vleeming *et al.* 2012). This occurs during trunk extension or hip flexion, whereby the sacral promontory moves posteriorly and superiorly (Lippert 2011)

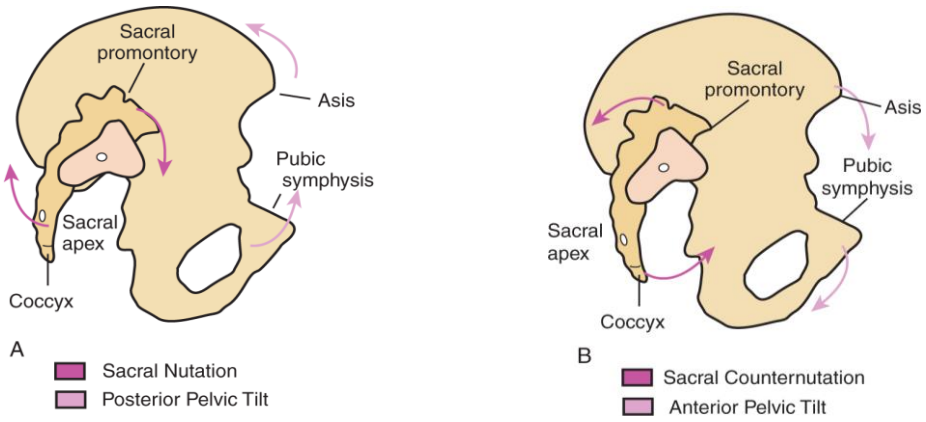


Figure 2 Nutation (A) and counter-nutation (B) (Levangie and Norkin 2000)

Rotation of the ilium posteriorly can result in nutation of the sacrum. This is also true for an anteriorly rotated ilium and counternutation of the sacrum (Levangie and Norkin 2000). A joint fixation can occur at the SIJ at any point in these positions of pelvic obliquity, resulting in joint hypo-mobility (Henderson 2012). This rotation of the pelvis can result in 'Anisomelia' - a condition in which paired limbs are noticeably unequal. This condition affects 40-70% of the population (Kiapour et al. 2012). Hawk (2017) explained that using leg-length as an analysis of pelvic obliquity is considered a functional-based test. Triano (2005) confirmed the validity of leg-length analysis in assessing the pelvic position.

Leg-length discrepancy is directly proportional to the peak load at the SIJ, and it is estimated that a discrepancy as small as 1 cm increases the load across the SIJ by five times (and 3 cm by 12 times), as reported by Kiapour et al. (2012). In this study, this force increase was noted in both SIJs, but was most pronounced on the side with the greater leg length.

According to Al-khayer and Grevitt (2007) the most common reported cause for SIJ pain is SIJ dysfunction due to pregnancy (hypermobility), repetitive minor trauma, or limb length discrepancy. Therefore, a leg length discrepancy in asymptomatic individuals can be thought to precipitate SIJ pain in the future. Hence, it would be beneficial to correctly manage this discrepancy prior to the onset of pain.

2.2.3 Form and force closure

2.2.3.1 Form closure

Form closure refers to a theoretical stable state of the SIJ whereby the close-fitting articular surfaces, create a stable state for the joint during loading and unloading (Zlomislic 2019). If the sacrum were to fit into the pelvis with completely perfect form closure, no lateral compression forces would be needed to maintain stability. However, a design such as this would make mobility impossible.

The components which contribute to form closure include (Vleeming and Schuenke 2019):

- the configuration of the SIJ surfaces, with the ‘wedging’ of the sacrum into the ilia;
- the corresponding architecture of the articular surfaces of the SIJs (resulting in a high coefficient of friction), and
- the integrity of the ligaments surrounding the SIJ, which are some of the strongest in the body.

Although these components create a stable environment in the static state, the practically flat surfaces of the SIJ also make the joints vulnerable to the effects of shear forces (Liebenson 2004; Feeney *et al.* 2018). When a person walks, there is unilateral loading of the legs – this introduces shear forces to the SIJs which can reach up to 4800 N (Feeney *et al.* (2018). Form closure alone is insufficient to create stability to the SIJ when walking; thus force closure is also required for pelvic stabilisation.

2.2.3.2 Force closure

Force closure is the theoretical state where lateral force and friction (resulting in joint compression) are required for the SIJ to withstand vertical loading (Zlomislic 2019). Ligaments, muscles, and the thoracolumbar fascia aid in stabilizing the pelvis by compressing the SIJs (Liebenson 2004). In order for force closure to be effective the surrounding muscles need to work optimally. The three slings which form active components of the pelvic stabilization system are the anterior oblique, longitudinal, and posterior oblique slings (Liebenson 2004).

2.3 The posterior oblique sling

Several authors (Liebenson 2004; Carvalhais *et al.* 2013; Barker *et al.* 2014; Lee *et al.* 2019; Vleeming and Schuenke 2019) propose the concept of muscular slings in the human body. These slings consist of muscles which form part of a muscular chain; where the muscles influence a kinetic pattern of the body (Lee *et al.* 2019). This muscular chain can be composed of synergistic muscles, muscle slings and fascia - with each of them having interdependent relationships with neurons, organs, and joints (Liebenson 2004; Lee *et al.* 2019). The sacrum, spine, pelvis, and their connections to the limbs and head are viewed as an integrated, interdependent and dynamic biological structure working as a kinematic chain (Vleeming *et al.* 2012).

The POS, as illustrated in Figure 3, consists of the gluteus maximus, the contralateral latissimus dorsi muscles, and the thoracolumbar fascia between them. The anatomy of these structures is detailed in Appendix B. It is suggested that this chain of muscles assists in force closure and pelvic stability (Liebenson 2004).

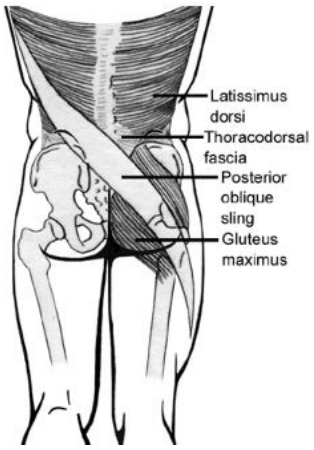


Figure 3 The posterior oblique sling (Diane 2004)

Mooney *et al.* (2001) found that the POS creates a perpendicular compressive force acting on the SIJ increasing SIJ stiffness and stability. This was supported by Barker *et al.* (2014) who found that the gluteus maximus directs 70% of its muscular force to act perpendicularly to the plane of the SIJ. Fourteen percent of this force was dissipated through the muscle's attachment and into the thoracolumbar fascia. Carvalhais *et al.* (2013), in an *in vivo* study, found active and passive tensioning of the latissimus dorsi caused lateral rotation in the contralateral hip, suggesting that there was force transfer between the muscles of the POS.

The POS also aids transmission of forces from the pelvis, over the trunk, to the contra-lateral shoulder (Vleeming *et al.* 1990). Trunk stability and effective load transmission from the spine to the limbs, and limbs to the spine, is important in order to protect the lumbopelvic-hip complex from injury. As one walks, the gluteus maximus muscle contracts during toe-off, while the contralateral latissimus dorsi muscle simultaneously contracts. This creates a mutual gait pattern between the lower and upper extremities (Shin, Kim and Yoo 2013). Similarly, during running and axial movement of the trunk there is this relationship which prevents excessive rotation of the spine during gait.

Hence, alterations to the function of this sling may predispose an individual to spinal injury at various different speeds of walking/ running (Page, Frank and Lardner 2010).

During ground reaction forces the fascia, ligaments and muscles respond creating a perpendicular compressive force across the SIJ in order to adapt and overcome the forces of gravity (Vleeming and Schuenke 2019). A disruption of this mechanism can lead to abnormal movement of the joint, or compensations in kinematics of gait, ultimately predisposing the individual to the onset of pain (Feeney *et al.* 2018).

The TLF, its structure detailed in Appendix C, consists of three layers. Each layer covers the muscles of the lumbar spine, separating them into three compartments. The superficial layer of the posterior component attaches to the latissimus dorsi and gluteus maximus muscles. Through the latissimus dorsi muscle aponeurosis it attaches to the supraspinal ligament and spinous processes of C1 - L4 (Vleeming et al. 1990a). The TLF can help regulate the tension of the muscles of the POS (Stevens *et al*. 2007). It has been shown that the TLF has a rich supply of nociceptors and mechanoreceptors - Schilder *et al.* (2014); Schilder *et al.* (2016) showed that the TLF is greatly nociceptive in nature - implying that it is more sensitive to stimulation than the underlying muscle (Wilke *et al.* 2017), allowing for proprioceptive feedback. Thus the TFL not only has a force-dissipating function but is responsible for providing information about the body's position in space (Snell 2010; Mai and Paxinos 2011; Tortora and Derrickson 2017).

Therefore this intimate relationship between the SIJ and POS plays an important role in load transfer from the spine to the legs and legs to the spine (Van Wingerden *et al.* 2004; Page, Frank and Lardner 2010). In order for the SIJ to contribute to pelvic stability it has to maximise form and force closure (Vleeming *et al.* 2012) and rely on an optimal functioning POS (Feeney *et al.* 2018; Vleeming and Schuenke 2019).

2.4 Relevant Neuroanatomy

The nervous system consists of two parts - the CNS (the brain and SC) and the PNS (all nervous tissue outside of the CNS). The peripheral nervous system consists of two types of nerves - the cranial and spinal nerves (Moore, Dalley and Agur 2014). Nerves

are categorized into sensory, motor and interneurons (see Appendix D for classifications of sensory and motor nerves).

Joint receptors are specialised sensory nerve endings that respond to mechanical, thermal or chemical stimulation (Russo *et al.* 2018). When they are stimulated, an action potential is generated along the sensory nerve with which it is in continuum (Pickar 2002; Mai and Paxinos 2011). Appendix E classifies the various types of receptors in the body, however, the main receptors involved in joints/muscles are muscle spindles, golgi tendon organs, and nociceptors. The relationship between the muscle spindle and GTO is responsible for the tone of a muscle (Haines 2012). Appendix F gives a detailed description of these receptors and their role in the stretch reflex.

Once a receptor has been stimulated, this stimulus is transmitted via the dorsal roots of the sensory nerve to the grey matter of the spinal cord. The grey matter of the dorsal horn is divided into 10 zones known as laminae (Mai and Paxinos 2011). Sensory information terminates here at the various types of laminae, which then ascends via the spinothalamic and spinorecticular tracts to the recticular nuclei in the brain stem. From the ascending tracts the impulse is carried via the medial lemniscus to the thalamus and then to the cerebral cortex.

The information from the brain is transmitted via descending pathways along specific relevant spinal tracts including the corticospinal, vestibulospinal and rubrospinal tracts (Bergmann and Peterson 2010; Mai and Paxinos 2011). The descending tracts terminate on the interneurons in the SC gray matter which then excite the ventral motor neurons that can cause the appropriate reaction (e.g. muscle contraction/relaxation). The motor neurons involved in muscle tone (discussed in Appendix F) receive both inhibitory and excitatory input from neighbouring interneurons. These propriospinal neurons and local reflex circuits assist with motor neuron modulation (Cramer and Darby 2017).

A motor unit is a motor neuron and its muscle fibres that it innervates. Motor neurons combine together to form a motor neuron pool. This motor neuron pool is responsible

for the innervation of one particular muscle group. When stimulated during voluntary contraction, the smallest motor units are recruited first, followed by larger motor units, as the synaptic drive increases (Snell 2010). The number of motor units that are recruited in a muscle contraction determines the strength of the contraction. The firing rate of muscle fibres is directly proportional to the exertional demand of the muscle – hence, the greater the demand, the greater the recruitment of muscle fibres measured by frequencies with electromyography (Criswell 2010). Therefore, the more recruited motor units the greater the muscle contraction (Kaplan 2000).

2.5 Surface electromyography

To measure the muscle activity of the POS muscles the Biopac Bionomadix MP150 Data Acquisition System was used. The sEMG machine measures muscle electrical activity non-invasively during cycles of muscle contraction and relaxation (Merletti and Parker 2004). EMG signal amplitude ranges from 0-10 mV (peak to peak) or 0-15 mV (RMS) and usable energy (i.e. signals with energy greater than the noise level) of the signal ranges from 0-500 Hz (frequency). sEMG signal fidelity can be influenced by the: signal to noise ratio, distortion of the EMG signal, and cross talk susceptibility between adjacent muscles (De Luca 2002). The raw data was processed as root mean square (RMS), as it is considered the most accurate analysis of the signal, providing a good measurement of the power and amplitude of the signal (Suter and McMorland 2002a). Normalization of the sEMG signal was necessary in order to be able to compare EMG activity in the same muscle in different individuals as well as to compare EMG activity between muscles (Halaki and Ginn 2012).

Electrical noise may come from various sources such as: inherent noise in electronic components of the EMG equipment, ambient noise from electromagnetic radiation, motion artefacts, and the inherent instability of the signal – and was managed where possible in this study. The noise was reduced by using conductive electrolytes (gel electrodes) and removing dead dermis in the areas of electrode placement (using alcohol swabs) to improve adhesiveness to skin (De Luca 2002).

The noise could have been further reduced by using detection surfaces with a large surface area, however crosstalk may have been increased if there was a greater

detection surface area and inter-detection distance, as the electrodes would be more susceptible to detecting signals from muscles adjacent (lateral and below) to the muscle they were wanting to get readings from. A representative sample was obtained by rather using only a small surface area of the muscle as muscle fibres of motor units are spread throughout most of the muscle's cross-section. Taking these points into consideration, a smaller sized electrode was used to minimize crosstalk (De Luca 2002).

Correct electrode placement is essential in order to record an accurate EMG reading (Criswell 2010). According to De Luca (2002), the electrode needed to be placed between two motor points (innervation zones) or between a motor point and the tendon insertion of the muscle (but not on/near the tendon as it will result in a lower amplitude and increased crosstalk). The longitudinal axis of the electrode was positioned parallel to the length of the muscle fibres, along the longitudinal midline of the muscle itself (De Luca 2002; Florimond 2010).

The relationship between the force produced by the muscle and the amplitude of the EMG signal has caused much controversy. Some studies describe a relatively linear relationship, whereas others demonstrate a relative non-linear relationship (with the EMG amplitude signal increasing greater than the force) (De Luca 2002). It is now known that in small muscles the relationship is relatively linear whereas, in larger muscles, the relationship is relatively non-linear. This is because the firing rate of the motor units has a greater dynamic range and motor unit recruitment is limited to the lower end of the force range in small muscles, and in larger muscles the motor unit recruitment continues into the upper end of the force range and the firing rate has a lower dynamic range (De Luca 2002).

2.6 Spinal manipulative therapy

Although chiropractors deliver many forms of treatment, the method most associated with the profession is spinal manipulative therapy (SMT), also known as the "chiropractic adjustment". SMT is delivered to a hypomobile/fixated joint. A joint fixation has been defined by two cardinal biomechanical features - hypomobility and joint 'malposition' (Bergmann and Peterson 2010; Henderson 2012). A joint fixation results

in motion segment dysfunction, which incorporates pathological changes within its connective tissue, nerves, muscles and ligaments (Gatterman 2005). This results in abnormal joint motion, lack of joint play and end feel, and the possible presence of palpable soft tissue changes such as muscle hypertonicity and pain (Haavik and Murphy 2012). The recent literature highlights that the presence of spinal dysfunction has an effect on central neural processing – suggesting that spinal dysfunction may lead to altered afferent input to the CNS (Haavik-Taylor and Murphy 2007). Therefore these biomechanical characteristics are used by clinicians as clinical indicators of spinal dysfunction, but do not necessarily encompass the entirety of its neurological effects (Lelic *et al.* 2016). Sensorimotor integration (SMI) is the coordination process within the nervous system, whereby incoming sensory information from different parts of the body is integrated with the motor system in order to control movement (Haavik-Taylor and Murphy 2007). Haavik and Murphy (2012) proposed that areas of joint fixation demonstrate a state of altered sensory input which may be responsible for perpetual central plastic changes. The abnormal sensory input is received and processed – causing altered SMI of the afferent input, which can then be normalized by SMT, as depicted in Figure 4 (Haavik-Taylor and Murphy 2007; Haavik and Murphy 2012).

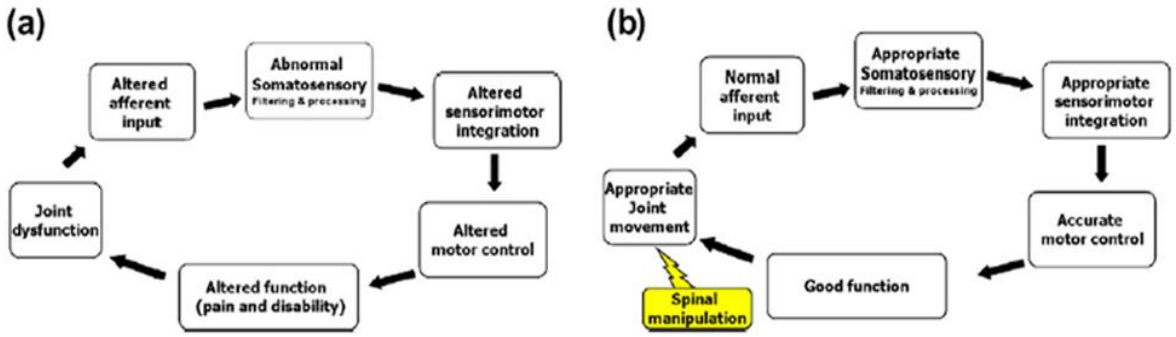


Figure 4 (a) Diagram depicting the proposed effects of spinal fixation, leading to altered SMI (b) Proposed effects of SMT, leading to normalized afferent input and appropriate SMI and restoration of function (Haavik and Murphy 2012)

Altered sensory input from joint fixations can lead to both inhibition or facilitation of neural input to the related muscles (Haavik-Taylor and Murphy 2007). Arthrogenic muscle inhibition (AMI) results when there is a presynaptic, reflex inhibition of the muscles surrounding a joint that is injured, even though the muscles themselves are

not necessarily damaged (Rice *et al.* 2014). It occurs in any condition that involves injury (including surgery) to a joint, as it is the body's natural response to protect the joint from further injury. Although AMI has mostly been associated with muscles that cross the joint, Suter and Lindsay (2001) found that AMI can occur in muscles that are not directly connected to the spine.

AMI has been seen to mainly be caused by the stimulation of mechanoreceptors, and to a lesser degree by free nerve endings and specialised nociceptors (i.e. AMI can exist without the presence of pain) (Rice and McNair 2010). Studies have shown that joint dysfunction can result in an elevation of intra-articular pressure due to joint infusion, which can stimulate mechanoreceptors which are sensitive to stretch. As discussed in the stretch reflex in Appendix F, there is an increase in group II joint afferent discharge (flower-spray sensory nerve endings). These afferents are known to excite group-Ib inhibitory interneurons in the SC, which in turn inhibit alpha motor neurons and restrict full activation of the muscle. Thus, AMI causes a decreased recruitment of motor neurons within the motor neuron pool of the related muscles. The decreased motor neuron pool excitability decreases the ability of the musculature to activate fully, and results in a decrease in the force of contraction of the involved muscles (Suter and McMorland 2002a; Rice *et al.* 2014; Kim *et al.* 2017).

An alteration of muscle recruitment patterns causes a redistribution of loads to auxiliary muscles and ligaments that are not accustomed to the increased stress. This increases the magnitude of coupled motions due to the loss of load bearing efficiency. Thus, there is a heightened likelihood of injury occurrence (Triano 2005). Although AMI of a muscle is a protective mechanism for the particular joint it is associated with, it can predispose injury to other structures of the body due to altered biomechanics. Hence, it is necessary to find a treatment modality that alleviates the cause for muscle inhibition. Treatment of AMI is directed at removing, masking, or overriding the inhibitory interneuron activity; often performed by stimulating the efferent fibres of the PNS, such as with SMT (Parvizi and Kim 2010; Lelic *et al.* 2016).

Motion palpation is the technique used as a palpatory diagnostic tool used to determine the presence of joint dysfunction. It assesses active and passive segmental joint range

of motion – assessing for local pain, tissue hypersensitivity, altered alignment, decreased, increased or aberrant joint movement, altered joint play and end feel, and local palpatory muscle rigidity (Bergmann et al. 2002). Although Bergmann and Peterson (2010) list pain as a major clinical feature for a joint dysfunction, it is not always a pre-requisite for joint dysfunction. Like any disease process, a joint dysfunction may be present in the body for a long period of time before pain is perceived (Haldeman 2004). In much the same way that the asymptomatic patient can still present with joint dysfunction, the cessation of pain does not always indicate the removal or resolution of a dysfunction (Gatterman 2005).

SMT can be delivered via several different techniques. The most common is a low-amplitude, high-velocity dynamic thrust, which is delivered with a controlled velocity and a specific line of drive that does not exceed the anatomical limits of motion. It is delivered at or near the end of the physiological range of motion, into the paraphysiological space (Pickar 2002; Hawk 2017). It is often associated with an audible popping sound, known as a “cavitation.” It is the result of reduced pressure in the joint capsule, resulting in the dissolved gasses from synovial fluid to coalesce and then collapse. It is not necessary for the effects of SMT to be optimal (Cooperstein *et al.* 2001; Gatterman 2005; Hawk 2017). Other types of SMT involve using instruments or devices that do not typically cause cavitation and provide an alternative to conventional techniques. A “drop” adjustment involving a table with a spring-loaded section below the patient is commonly used, and is often gentler and more comfortable for patients (Hawk 2017).

SMT initiates a cascade of neurophysiological events to correct the changes in input from the spine due to joint dysfunction, which over time lead to altered sensorimotor integration of input from the spine and limbs (Haavik and Murphy 2012). It has been shown to have positive benefits in patients with low back pain and other conditions (Hawk 2017). It is unclear as to the exact mechanism through which SMT produces its therapeutic effects, however biomechanical, neurophysiological, peripheral, spinal and supraspinal mechanisms have been suggested (Potter, McCarthy and Oldham 2005; Bialosky et al. 2009).

Many researchers (Evans 2002; Suter and McMorland 2002a; Potter, McCarthy and Oldham 2005; Ernst 2008) have tried to separate the different effects of SMT, however, there is an intimate relationship between the biomechanics, biochemistry and neurophysiology of a joint and its supporting tissues (Bialosky *et al.* 2009). Even though the initial HVLA thrust may be biomechanical, the movement provokes a cascade of biochemical and neurophysiological events (Pickar 2002; Bialosky *et al.* 2009; Cardinale *et al.* 2015a; Lelic *et al.* 2016).

Biomechanical changes to the structures of the joint during manipulation (i.e. stretching the capsule/muscles) stimulates mechanoreceptors within the joints and surrounding tissues (Pickar 2002). This information is then relayed along type I and type II sensory fibres to the dorsal horn of the SC. The sensory neuron synapses with the interneuron which passes on either an excitatory or inhibitory stimulus to the efferent motor neuron. This impulse is transmitted to effector muscles, resulting in an increase or decrease of motor neuron pool excitability (Symons *et al.* 2000; Pickar 2002; Suter and McMorland 2002a). Often, this pathway allows for an increase in joint ROM as there is a reduction of muscle tone (Lehman 2012; Vaseghnia *et al.* 2018).

Maigne and Vautravers (2003); Shearar, Colloca and White (2005) discussed that by reducing the activity of the motor neuron, SMT reduces hypertonicity - and subsequently can reduce muscle spasm and pain, if present. According to Gatterman (2005) the mechanical effects of manipulation include ‘realignment’ of the joint and the correction of dysfunctional joint ROM and, in more recent literature, the effects of manipulation are described to have a more neurophysiological origin (Fryer and Pearce 2012; Harvey and Descarreaux 2013; Lelic *et al.* 2016).

Several investigations have been undertaken to assess the effect of SMT on muscle activity, as seen in Table 1. The studies conducted range from using symptomatic, mostly either low back or neck pain, or asymptomatic participants.

Table 1 The effects of spinal manipulative therapy on muscle activity

Author	**Study Design (n)**	**Outcome Measures**	**Intervention**	**Results**

Herzog, Scheele and Conway (1999)	n = 10 asymptomatic participants Pre-test, post-test	sEMG readings of 16 back and proximal upper limb muscles	1. SMT (11 treatment sessions) 2. Control	Consistent reflex responses in target areas post SMT.
Keller and Colloca (2000)	n =40 LBP participants RCT	MA of PS during trunk MVC extensions	1. Activator SMT 2. Control	Significant increase in PS MA during MVC extension following manually assisted SMT.
Lehman and McGill (2001b)	n =14 LBP participants Analytical cohort	PS and abdominal MA, and lumbo-pelvic kinematics	1. L/S SMT 2. Control	No significant changes following SMT. MA decreased in participants with higher levels of pain or more severe dysfunction
Suter and McMorland (2002b)	n =16 Chronic NP RCT	Active CROM, elbow flexor force, sEMG of elbow flexor and PPT	1. C/S SMT 2. Control	Significant decrease in muscle inhibition and increase in force out, CROM and PPT following SMT of C/S.
Dishman (2003)	n =66 Asymptomatic participates Pre-test, post-test experimental	Changes in flexor carpi radialis and gastrocnemius H-reflex amplitudes before and after SMT	1. C/S SMT 2. L/S SMT	Transient suppression of motor neuron excitability. L/S SMT caused greater attenuation of motor neuron activity than C/S. Therefore, C/S and L/S have different responsiveness levels to SMT
DeVocht, Pickar and Wilder (2005)	n =16 LBP participants RCT	MA of PS (resting)	1. SMT 2. Activator SMT	Significant decrease in MA. Initial increase in MA followed by decrease compared to pre-intervention levels.
Dunning and Rushton (2009)	n =54 Asymptomatic participates RCT	Resting EMG of biceps brachii muscle bilaterally	1. C/S SMT 2. Sham 3. Control	Increase in MA immediately after C/S SMT.
Lalanne, Lafond and Descarreaux (2009)	n =27 Chronic LBP participants RCT	Trunk and pelvic angles, and PS MA during flexion-extension task	1. L/S SMT 2. Control	SMT group had significant decrease in PS MA during full flexion

Fryer and Pearce (2012)	n =14 Asymptomatic participates RCT crossover	sEMG of right gastrocnemius muscle, M-wave and H-reflex, and MEP amplitudes	1. SIJ SMT 2. Control	Significant decrease in corticospinal and spinal reflex excitability after SMT.
Harvey and Descarreaux (2013)	n =60 LBP participants RCT	MA, pain intensity, and lumbopelvic kinematics	1. L/S SMT 2. Control	Control group had increase in PS MA during task compared to SMT group.
Nougarou *et al.* (2013)	n =26 RCT	MA of PS muscles (bilat. T6&8) was recorded during and after SMT, and EMG values were compared across the varying levels of force.	1. 2 trials of 4 different SMT force-time profiles	The thrust phase had an increased PS MA with increased force, but also in the two 250ms time windows after the SMT. Attenuated 500ms post SMT.
Cardinale *et al.* (2015b)	n =27 Asymptomatic participates RCT crossover	Force fluctuation task, modified Sorenson's test, and sit and reach. sEMG of PS and gastrocnemius.	1. L/S SMT 2. L/S Stretching 3. Sham	No significant improvement superior to the other modalities for force output and sEMG parameters post L/S SMT.
Niazi *et al.* (2015)	n =10 Subclinical LBP participants RCT	sEMG V-wave, H-reflex, M-wave, and MVC of ankle plantar flexors	1. L/S SMT 2. Control	Significant increase in motor neuron pool excitability, cortical drive and preventing fatigue.
Pires *et al.* (2015)	n =32 Women chronic NP RCT	MA of SCMs at rest and during isometric contractions for cervical flexion and elevation of the shoulder girdle. NP using visual analog scale.	1. Upper T/S SMT 2. Control	No significant differences were found in the immediate intragroup or intergroup analyses of groups.
Lelic *et al.* (2016)	n =19 subclinical pain RCT	SEPs from 62-channel EEG cap following median nerve stimulation	1. SMT 2. Control	SMT significantly decreased N30 amplitude. Brain source modelling revealed a 4-source model but only the prefrontal source showed significant reduced MA following SMT.

(PPT=pressure pain threshold, PS=paraspinal muscles, MA=muscle activity, NP=neck pain, LBP=Low back pain, SMT=spinal manipulative therapy, SEPs=somatosensory evoked potentials, RCT=randomized clinical trial, sEMG=surface electromyography)

2.6.1 Effect of SMT on MA in asymptomatic and symptomatic individuals

The research conducted by Herzog, Scheele and Conway (1999) first observed the effects of SMT on asymptomatic participants (n=10), whereby they recorded neuromuscular reflexes and MA of 16 back and proximal upper limb muscles, using sEMG. Post SMT, there were consistent reflex responses in target-specific areas. They postulated that the responses to SMT were of multi-receptor origin, and that they were responsible for the resultant functional improvements and inhibition of muscle hypertonicity. This was supported by Dishman (2003), whereby 66 asymptomatic participants received either cervical or lumbar SMT – and were compared according to MA recorded in the flexor carpi radialis and gastrocnemius muscles. There was transient suppression of motor neuron excitability, with L/S SMT causing greater attenuation of MA than C/S SMT. Both studies observed a reduction in muscle hypertonicity following SMT.

Although Dunning and Rushton (2009) observed changes in resting biceps brachii MA and Niazi *et al.* (2015) recorded the change in MA in the plantar flexors during MVC, both studies utilised asymptomatic participants, and observed increased motor neuron pool excitability post SMT. Whereas Fryer and Pearce (2012) and Lelic *et al.* (2016) observed decreased spinal reflex excitability after SMT. In contrast, Cardinale *et al.* (2015a) found no significant improvement in muscle activity of the PS and gastrocnemius muscles in the group receiving L/S SMT compared to those receiving L/S stretching or a sham intervention. These contrasting results indicate that, in asymptomatic individuals, the muscle activity responses to SMT are variable and require further investigation.

In studies using symptomatic participants, the results are just as irregular. Keller and Colloca (2000) observed an increase in sEMG readings after SMT (mechanically assisted with the activator device) in PS muscles during MVC of the trunk in extension (n=40 LBP). Thereafter, Suter and McMorland (2002a) and Harvey and Descarreaux

(2013) observed decreased AMI, and a resultant increase in MA in the associated muscles after C/S and L/S SMT respectively.

In a CLBP population (n=27), Lalanne, Lafond and Descarreaux (2009) assessed the effect of L/S SMT on PS activity during a flexion-extension task. There was a significant decrease in PS MA during full flexion. People with LBP fail to reach the relaxation phenomena of forward flexion, therefore this study shows that SMT can modulate these stabilizing neuromuscular responses, and encourage the relaxation phenomena that occurs in healthy individuals in forward flexion. This was further supported by DeVocht, Pickar and Wilder (2005); Nougarou *et al.* (2013) who observed an initial increase in PS MA post SMT, followed by an attenuation of MA post SMT when compared to pre-intervention levels. The authors propose that these findings are consistent with the assumption that muscle hypertonicity is associated with LBP and can be alleviated with SMT.

However, other research demonstrated no significant changes in the associated muscle motor neuron excitability following SMT in symptomatic patients; Lehman and McGill (2001a) observed no significant change in resting MA in PS and abdominal musculature in patients with LBP; and Pires *et al.* (2015) noted no change in myoelectric activity (at rest and during MVIC for C/S flexion and shoulder girdle elevation) of the right and left sternocleidomastoid muscles immediately after SMT and 48-72 hours after the intervention.

An overview of the literature suggests that there is an effect on muscle activity in both symptomatic and asymptomatic individuals. However, the mechanisms through which it is achieved and the outcome expected for each region is still in need of further research. Though some studies have identified a decrease in MA following SMT, increased MA cannot yet be a discriminating factor between asymptomatic and symptomatic populations (Lehman and McGill 2001a). Assessing the effects of SMT on asymptomatic individuals may provide additional knowledge about the neurophysiological muscle response of this treatment modality without the confounding, uncontrolled effects of abnormal MA related to injury(Grindstaff *et al.* 2009).

2.6.2 Effect of SMT on MA in local and distal muscles

Haavik and Murphy (2012) proposed that areas of joint fixation demonstrate a state of altered sensory input which may be responsible for perpetual central plastic changes. The abnormal sensory input is received and processed – causing altered sensorimotor integration (SMI) of the afferent input, which can then be normalized by SMT (Haavik-Taylor and Murphy 2007; Haavik and Murphy 2012). Understanding that the causation and correction of a spinal fixation is that of a neurological origin enables one to understand that SMT has the ability to correct abnormalities in muscles not only mechanically connected to the involved joint, but those at a distant to the joint too.

Evidence suggests that there is increased muscle tone associated with spinal fixation that may be reduced by SMT. However, some reported increases in MA following SMT can be attributed to improved motor fibre recruitment. Therefore, either inhibition or facilitation of a muscle following SMT may be beneficial, considering the state of the muscle prior to SMT. It is possible that the changes in motor recruitment strategies may be as a result of the alteration in corticospinal excitability (Fryer and Pearce 2012).

There have been studies conducted involving various regions of the body – suggesting that SMT may have an effect on both muscles local and distal to the joint receiving the manipulation. It has been suggested that sEMG data is indicative of a reflexive response that originates from muscle spindles receptors within a muscle (Nougarou *et al.* 2013). In a RCT conducted by Nougarou *et al.* (2013) participants (n=26) received two trials of four different SMT force-time profiles. The MA of the PS muscles (local), at the levels of T6 and T8 bilaterally, were recorded during and after SMT. The thrust phase resulted in an immediate increase in PS MA, as well as the two 250ms time windows after the SMT. The MA of the PS muscles then attenuated 500ms post SMT, suggesting that SMT caused a ‘spike’ in MA, and then decreased the MA in the PS muscles. Lumbar manipulation on LBP participants (n=60) has also been demonstrated to result in an increase in PS MA (Harvey and Descarreaux 2013).

Niazi *et al.* (2015) suggested spinal cord effects on MA in the RCT using individuals with sub-clinical LBP (n=10). sEMG, V-wave, H-wave, and M-wave as well as ankle plantar flexor MVC were recorded pre and post SMT. It was reported that L/S SMT

produced significant increases in motor neuron excitability and cortical drive when compared to a control group. There was a resultant improvement in soleus muscle MVC. They suggest that SMT may reduce short term muscle fatigue, suggesting significant clinical relevance of SMT as a treatment modality for sports athletes. There was also a significant decrease in corticospinal and spinal reflex excitability (in relation to the right gastrocnemius muscle) after SIJ SMT delivered to asymptomatic participants (n=14) (Fryer and Pearce 2012).

Lelic *et al.* (2016) conducted a study whereby participants (n=19) attended two sessions, one SMT and one control, in random order. SEPs from 62-channel EEG cap were recorded following median nerve stimulation (1000 stimuli at 2.3 Hz) before and after either intervention. The results revealed that a single session of SMT may alter somatosensory processing at the cortical level, especially within the prefrontal cortex. The prefrontal source showed a 20.2% reduction in activity post SMT.

These studies over the years show that, following SMT, there are changes in the muscles around the spine as well as muscles at a distance from the joint, however, there is a paucity of literature investigating the effect of SMT on muscles that constitute a muscle sling. There is a lack of correlation of sEMG measures and other various clinical outcome measures, hence determining the clinical significance of sEMG changes is difficult. There is conflict in current literature as to the exact effects of SMT on sEMG readings, warranting further investigation into the specifics of these effects (Fryer and Pearce 2012). Further understanding of the neurophysiological responses following SMT will help guide future studies and provide scientific explanation for treatment selections (Grindstaff *et al.* 2009).

2.6.3 Effect of SMT on force output

A reduction in muscle strength and power can be detrimental to a person's functional ability and can predispose an individual to injury. Force output is mediated by neuromuscular mechanisms, and can be studied well by measuring MVC (Christiansen *et al.* 2018). SMT as well as joint mobilization have been shown to alter force output and activation of musculature (Grindstaff *et al.* 2009).

Table 2 Effects of spinal manipulative therapy on force output

Author	Study Design (n)	Outcome Measures	Intervention	Results
Pollard and Ward (1996)	Pre-test, post-test experimental design (asymptomatic) (n=30)	Quadriceps muscle strength	1. L3/4 SMT 2. Control	Statistically significant short-term increase in quadriceps muscle strength in SMT group
Suter and McMorland (2002b)	n =16 Chronic NP RCT	Active CROM, elbow flexor force, sEMG of elbow flexor and PPT	1. C/S SMT 2. Control	Significant decrease in muscle inhibition and increase in force out, CROM and PPT following SMT of C/S.
Learman *et al.* (2009)	Unbalanced randomized controlled crossover design (n=33)	Proprioception, threshold to detect passive motion, direction of motion, and force reproduction	1. L/S SMT, sham 1 week later 2. Sham, L/S SMT 1 week later, sham 1 week later	Group 1 had an effect for TTDPM. Group 2 had an effect for joint position sense. No effect was noted for either DM or force reproduction.
Grindstaff *et al.* (2009)	RCT (asymptomatic) (n=42)	Quadriceps force and activation using the burst-superimposition technique during a seated isometric knee extension task before and at 0, 20, 40, and 60 min following intervention	1. SIJ SMT 2. 1 min lumbar passive ROM 3. Prone extension on elbows for 3 min	All groups had a decrease in force output without changes in activation at all time intervals following intervention. SMT group had increase in force (3%) and activation (5%) immediately following intervention (not present after 20 min).
Cardinale *et al.* (2015b)	RCT crossover design (asymptomatic) (n=27)	Force fluctuation task, modified Sorenson's test, and sit and reach. sEMG of PS and gastrocnemius.	4. L/S SMT 5. L/S Stretching 6. Sham	No significant improvement superior to the other modalities for force output and sEMG parameters post L/S SMT.

Lee and Kim (2016)	Randomized, assessor-blind controlled trial with a pre-test, post-test control group design. (chronic NP) (n=46)	Muscle strength and endurance, pain, neck disability index, and ROM of C/S and T/S	1. T/S SMT with deep craniocervical flexor training 2. deep craniocervical flexor training 3. control (active self exercise)	SMT increase muscle strength, endurance, and C/S and T/S ROM, and decreased pain and neck disability index
Christiansen *et al.* (2018)	Randomized controlled crossover design. (n=11)	Soleus-evoked V-waves, H-reflex and MVC of the plantar flexors; assessed immediate post, post 30 min and post 60 min.	1. SMT 2. Passive movement control	SM increased MVC force over time compared to control.

(PPT=pressure pain threshold, NP=neck pain, LBP=Low back pain, SMT=spinal manipulative therapy, ROM = range of motion, RCT=randomized clinical trial, sEMG=surface electromyography, MVC = maximum voluntary contraction)

Pollard and Ward (1996); Suter and McMorland (2002a); Lee and Kim (2016); Christiansen *et al.* (2018) reported an increase in force output following SMT. Christiansen *et al.* (2018) observed an increase in MVC force over time following SMT (n=11) in a randomized controlled crossover design study. However, this is in conflict with research done by Grindstaff *et al.* (2009) whereby asymptomatic participants (n=42) had an increase in force (3%) and activation (5%) immediately following intervention which was not present after 20 minutes.

Christiansen *et al.* (2018) purported the increase in plantar flexion force to be as a result of increased cortical drive to the muscle. There were no significant H-reflex changes, supporting previous research by Alkjaer *et al.* (2013) suggesting that changes in force output may be caused by supraspinal changes rather than changes in the H-reflex.

However, there are also studies demonstrating no significant effect of SMT on force output (Learman *et al.* 2009; Cardinale *et al.* 2015a). The varied results in the literature suggests that further research into the effects of SMT on force output is necessary.

CHAPTER THREE

Methodology

3.1 Introduction

This chapter describes the methodologies used in this study. It outlines the study design, population, sampling strategies used, measuring tools, manipulative interventions and applied time statistical analysis employed during the study.

3.2 Study design

This was a quantitative experimental study, utilizing a pre-test post-test design. This design is appropriate as it serves the purpose of comparing the control and intervention groups and measures any change resulting from the experimental treatment (Dimitrov and Rumrill Jr 2003).

3.3 Study population

Men and women residing in the eThekwini municipality who were asymptomatic with regards to pain and who met the necessary criteria were invited to participate.

3.4 Location

Permission to conduct the study, and to access staff and students of the DUT, was obtained from the Research Director (Appendix G). Permission to use the Chiropractic Day Clinic for data collection was obtained (Appendix H), as this was the facility used to conduct this study.

3.5 Sampling

3.5.1 Participant recruitment and advertising

Participants were recruited by means of advertisements (Appendix I) at local Durban gyms, as well as via word of mouth at various sports events and wellness days that the current chiropractic students of the Durban University of Technology attended. Permission was obtained prior to displaying the advertisements (Appendix J).

3.5.2 Inclusion and exclusion criteria

3.5.2.1 Inclusion Criteria

1. All participants were required to read and sign the participant information and consent form (Appendix K) prior to being admitted into the study. Consent was received both on paper and verbally.
2. The participants were 18 years of age or older in order to negate the necessity for consent from a parent, but not older than 45 years of age as people older than 45 have an increased prevalence of degeneration as seen on radiographs (Walker and Colledge 2013). Walker and Colledge (2013) said that the increased incidence of pathology in older individuals may also influence the results of the study.
3. The participants had to present with a sacroiliac joint fixation. In order to determine the presence of a SIJ dysfunction, the upper SIJ mobility test was used during the SIJ regional examination, as per the Gillet-Liekens method (Leach 2004). The participant was asked to stand and support his/her self by contacting the wall with both hands. The researcher knelt behind the participant and established thumb contacts on the participant's posterior superior iliac spine (PSIS) and second sacral tubercle. The participant was then instructed to flex the ipsilateral hip to approximately 90 degrees (while keeping their knee bent) - inducing flexion of the ipsilateral hip and SIJ. With normal movement, the researcher's thumbs should've approximated as the PSIS moved posteriorly and inferiorly towards the relatively stationary second sacral tubercle. A SIJ flexion restriction was suspected if the thumbs did not approximate and the pelvis rotated obliquely around the opposite hip. While the researcher maintained the same contact, the participant was then instructed to flex his/her contralateral hip to approximately 90 degrees (while keeping the knee bent) - inducing extension of the ipsilateral hip and SIJ. With normal movement, the researcher's thumbs moved apart as the PSIS moved anteriorly and superiorly away from the second sacral tubercle. A SIJ extension restriction was suspected when the researcher's thumbs did not move apart. This procedure was then repeated on the opposite side to establish if there was a SIJ restriction on the opposite side.

3.5.2.2 Exclusion Criteria

1. Individuals having received a chiropractic manipulation on the lumbar/sacral region four weeks prior to the study. This prevented the possible effects of previous treatment on the outcome of the results and the data collected.
2. Any patient with SIJ syndrome or pain experienced in the SIJs in the three weeks prior to the study (i.e. the participants had to be asymptomatic) (Lamoth *et al.* 2006).
3. Participants who had taken any drugs which have effects on the skeletal muscles (e.g. statins and spasmed) were excluded, as this would affect the results of the sEMG (Kruk 2014).
4. Individuals who presented with contraindications to spinal manipulative therapy, which required further testing, were excluded from the study as per the case history and physical examination (Gatterman 1991). Were any of the above mentioned conditions suspected after carrying out the case history or physical examinations, the participant was sent for the appropriate investigative procedures and/or referred to the appropriate practitioner for treatment.
5. Contraindications to sEMG including, but not limited to: open wounds, rashes, psoriasis, skin irritations, or skin conditions of any kind in the region of electrode placement.
6. Patients with a Body Mass Index (BMI) of 30 or higher (due to the potential influence of adipose tissue on the ability to take muscle activity readings) (Kim, Kang and Oh 2014).
7. Any lower extremity or shoulder pain as any dysfunction in these areas may affect the POS and be unrelated to the SIJ.

3.5.3 Sample size

In consultation with a statistician, a power analysis was conducted using Dowson's time-of-day effects experiment. An 80% power, 1 effect size and a probability of 0.05, resulted in a sample size of 34 participants required for the study (Appendix L) (Matthews, 2018). This allowed for a group of 17 participants in the intervention group and 17 participants in the control group.

3.5.4 Sampling procedure

Individuals who were interested in participating in the study contacted the researcher or were contacted by the researcher telephonically and underwent a telephonic screening interview (Appendix M). Considering all the relevant inclusion and exclusion criteria were met in the telephonic interview, an appointment was then made for the participant to present to the DUT Chiropractic Day Clinic.

3.5.5 Sample allocation

The participants were randomly allocated to one of the two groups using the hat method (Kim and Shin 2014). There were 34 pieces of paper containing 17 of each group written on them (one/ two) that were placed in a tin; the clinic receptionist picked a piece of paper allocating the participant to one of the respective groups. This method aimed to eliminate selection bias (Kim and Shin 2014).

- Group one: Intervention (n=17)
- Group two: Control (n=17)

3.6 Interventions

3.6.1 Group one – intervention

Participants from group one received a toggle-recoil drop piece adjustment applied to the sacroiliac joint as described by Cooperstein *et al.* (2001):
The sacroiliac joint was placed over the drop segment of the table, with the contact hand placed over the posterior superior iliac spine and a force was applied to that joint.

It can best be described as follows:

- Doctor position: Ipsilateral fencer stance
- Patient position: Prone with ASIS over edge of lumbar drop piece
- Contact point on patient: Ipsilateral sacroiliac joint, medial to PSIS
- Contact point on doctor: Reinforced pisiform
- Vector of the thrust: Posterior to anterior and inferior to superior

- Special requirements: The lumbar drop section was raised and the tension in the drop section set so that it would not drop with the patient's body weight, but was able to drop with the addition of minimal force, beyond that of the patient's weight.
- Procedure applied once all the above were in place: contact was taken and skin slack removed. A high velocity, low amplitude with toggle recoil adjustment was then applied to the affected SIJ.

This method of manipulation was chosen as it would impart little stretch on the POS muscles (which occurs during a side-lying diversified manipulation of the SIJ), hence, limiting the effects of stretch on the results observed.

3.6.2 Group two - control

Participants in group two received the control procedure. The above procedure was altered whereby the femur was placed over the drop section of the table, with no contact applied to the SIJ (i.e. neither the contact nor the indifferent hands contacted the patient). The thrust was then applied to the table, and not to the SIJ.

It can best be described as follows:

- Doctor position: Ipsilateral fencer stance
- Patient position: Prone with femur over edge of lumbar drop piece
- Contact point on patient: None
- Contact point on doctor: Pisiform
- Vector of the thrust: Posterior to anterior and inferior to superior
- Special requirements: The lumbar drop section was raised and the tension in the drop section set so that it would not drop with the patient's body weight, but was able to drop with the addition of minimal force, beyond that of the patient's weight.
- Procedure applied once all the above were in place: contact was taken on the drop section of the table and not on the patient – thus there was no subject contact - a high velocity low, amplitude with toggle recoil adjustment was applied to the table to activate the drop piece, with no thrust applied to the participant.

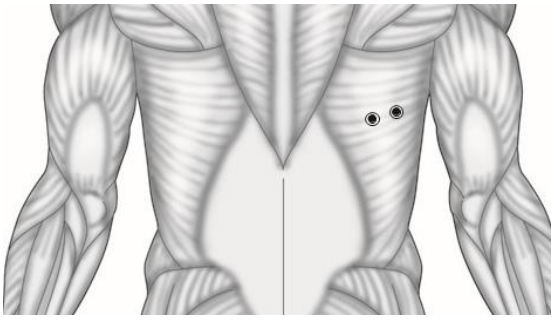
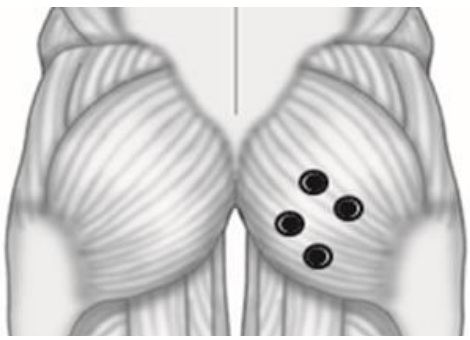
3.7 Measurement tools

To measure the MA of the POS muscles, the Biopac Bionomadix MP150 Data Acquisition System was used. A sampling rate of 2000 Hz was used for the duration of the data collection period. To ensure quality amplification of the sEMG waveform and elimination of any artefact or noise interference, the system has an internal high pass and low pass filter from 5.0 Hz to 500 Hz (Biopac Systems Inc 2015). Data was collected in the same research consultation room for all participants in the study. The room was protected from outside interference and noise by closing the door. To further prevent noise interference, all other electronic devices were stored away from the data acquisition system. All participants followed the same pre-manipulative procedure in order to control the influence of any extraneous environmental factors.

Disposable 35 mm round pre-gelled Ag/AgCl sEMG electrodes were placed on the muscle bellies of the GM and LD bilaterally, according to the technique outlined by Criswell (2010), as depicted in Table 3.

The Biopac - TSD121C Hand Dynamometer was used to quantify the muscle force output (in kilograms) during the maximum voluntary isometric contraction (MVIC) of the POS muscles in this study in order to reduce the effect of the changeable resistance provided by the researcher, which may alter sEMG readings. The dynamometer has been shown to be reliable for testing the strength of both the gluteus maximus (hip extension) and latissimus dorsi (shoulder extension) muscles (Thorborg *et al.* 2010; Çelik, Dirican and Baltaci 2012). The dynamometer was connected to the bottom of the bed by means of a carabiner, with a non-elastic band connected to the other side of the dynamometer in order to create a stable resistive loop for the patient to contract against.

Table 3 Electrode placement and MVIC positioning for the POS muscles (Konrad 2005; Criswell 2010; Thorborg *et al.* 2010; Çelik, Dirican and Baltaci 2012)

Muscle	Electrode placement	MVIC
Latissimus Dorsi	The electrodes (two cm apart) were positioned over the muscle belly (approximately four cm below the inferior angle of the scapula); centered midway between the spine and the lateral edge of the trunk; with a slight oblique angle of 25 degrees from horizontal in an infero-medial direction (measured using a set angle designed with two tongue depressors). 	Elbow placed at 90 degrees flexion and the shoulder is extended maximally. Resistance was applied on the posterior aspect of the upper arm, just proximal to the elbow, and the participant was asked to exert a maximum effort against the dynamometer. The resistance was against shoulder extension. The standardized command by the examiner was ‘‘go ahead-push-push-push-push and relax’’ (lasting 5s).
Gluteus Maximus	There are two possible locations noted; for the upper GM, two active electrodes (three cm apart) were placed midway between the trochanter (hip) and the sacral vertebrae in the middle of the muscle, on an oblique angle at the level of the trochanter or slightly above. For the lower gluteus maximus, placement is in the middle of the muscle below the level of the trochanter, three to five cm (one to two inches) above the gluteal fold. (For the purpose of this study, only the upper placement was used in order to prevent over-exposure of the patient, thereby protecting their privacy). 	Hip extension in the prone position using a long lever shows less measurement variation. The participant was in the prone position, with the hip slightly extended with slightly outward rotated legs. They then held onto the sides of the bed with both hands. They then pushed against a resistance five cm proximal to the proximal edge of the medial malleolus, at the posterior aspect of the lower leg, and the participant was asked to exert a maximum effort against the dynamometer. The resistance was applied against hip extension. The standardized command by the examiner was ‘‘go ahead-push-push-push-push and relax’’ (lasting 5s.)

3.8 Study procedure

Once the participant had been telephonically interviewed and met all the requirements (Appendix M), an appointment at the DUT Chiropractic Day Clinic (CDC) was made that lasted around 3 hours. At the appointment, the participant was given a verbal explanation and a letter of information and informed consent (Appendix K) to sign. He/she was informed that they were free to withdraw from the study at any time, and that this would not jeopardize any of their future treatments at the CDC. The participants were given the opportunity to ask any questions regarding the study, which were then answered by the researcher. The participant underwent a case history (Appendix N), physical (Appendix O), and a lumbro-pelvic-hip examination (Appendix P). When eligibility was determined, the clinic receptionist then allocated the participant to a group via the hat method.

If the participant did not meet the required criteria (i.e. did not have a SIJ dysfunction) or decided to not take part in the study, the researcher thanked them for their time and the participant was free to go. If the participant required referral to another health care practitioner, appropriate referral was given.

In order to determine the presence of a SIJ dysfunction, the upper SIJ mobility test was used during the SIJ regional examination, as per the Gillet-Liekens method (Leach 2004). If the patient had more than one restriction, only the first restriction found was adjusted.

The skin where the surface electrodes were placed was cleaned with alcohol and, when necessary, participants with hairy skin were shaved using a disposable razor (DeVocht, Pickar and Wilder 2005; Bicalho *et al.* 2010; Criswell 2010). The sEMG electrodes were placed bilaterally on the LD muscles and GM muscles of the participant (in the positioning mentioned in Table 3).

The participant was taught how to perform the maximal voluntary isometric contractions (MVIC's) of the latissimus dorsi (LD) and gluteus maximus (GM) muscles

correctly by the researcher, as high repeatability required proper guidance of the participants in order for them to produce identical contractions at maximum effort (Halaki and Ginn 2012). They were then allowed to practise these movements (twice for each movement). According to Halaki and Ginn (2012), there is a lack of consensus as to which MVICs are more effective in generating maximum activity in any given muscle. Thus, the MVICs chosen for this study were chosen due to their prone-lying position in order to minimize movement performed by the participant, as they were already in the prone position for their treatment. First, the activity (sEMG) and force output (dynamometer) of the left LD muscle was measured while the elbow was flexed 90 degrees and the shoulder was extended. The participant exerted their maximum effort for five seconds while the static band with the attached dynamometer resisted shoulder extension (Çelik, Dirican and Baltaci 2012; Escamilla *et al.* 2016). The participant was encouraged continuously to ensure maximum effort (Halaki and Ginn 2012). The right LD was assessed in the same manner.

The participant then held onto the sides of the bed with both hands. The activity (sEMG) and strength (dynamometer) of the left GM muscle was then measured while the hip was in a neutral position. The resistance was applied five cm proximal to the proximal edge of the medial malleolus, at the posterior aspect of the lower leg (resisting hip extension) while the participant exerted their maximum effort against the static band and dynamometer for five seconds. The participant was encouraged continuously to ensure maximum effort (Halaki and Ginn 2012). The right GM was assessed in the same manner. The MVICs that were chosen have been shown to reflect maximal neural activation capacity in the given muscles (Halaki and Ginn 2012).

Three repetitions of each MVIC were performed as a pre-test, with rest periods of 30 seconds between each repetition to reduce any effects of fatigue (Thorborg *et al.* 2010). The three mean values (and absolute maximum value) of muscle activity obtained by the sEMG and the absolute maximum value of muscle strength obtained by the dynamometer were then recorded.

The electrodes remained on the patient throughout the study to avoid any alterations to the results due to a difference in electrode placement between readings (Criswell

2010). The patient was allowed 2 minutes to rest before the intervention (Rouffet and Hautier 2008).

The intervention was applied as pre-determined by the group selection. The participant performed a post-test immediately after the intervention by repeating the same procedure of MVIC of the LD and GM muscles as in the pre-test, in order to see if there was any change in muscle fibre recruitment and force output. The three mean values (and absolute maximum value) of muscle activity obtained by the surface EMG and the absolute maximum value of muscle strength obtained by the dynamometer were then recorded. All data was recorded in an excel spread sheet.

The electrodes were then removed from the patient's skin.

The researcher then allowed the patient a few minutes to rest if they needed to, then thanked them and gave them their free treatment voucher (valid for six months) before they left.

3.9 Data reduction and analysis

3.9.1 Data reduction

At the initial consultation prior to the interventions, three trials of muscle activity were recorded during MVIC of each muscle. The raw data was then processed using root mean squared (RMS) to convert the raw data to a waveform suitable for analysis. The peak amplitude was then obtained by using the maximum peak muscle activity from the three readings, this was recorded per muscle. The mean muscle activity was obtained by using the mean muscle activity for the three trials per muscle, divided by three to obtain the average mean muscle activity obtained during the MVICs, per muscle. This was reported as mean muscle activity. This was then repeated after the intervention/control.

In order to compare between the groups, the peak and the mean muscle activity was normalized. This was done by normalizing to a reference value which, in this study, was the maximum peak amplitude observed from the three trials prior to the

interventions i.e. at baseline. This converted the muscle activity to a percentage (Konrad 2005; Halaki and Ginn 2012).

The baseline peak amplitude readings, through normalization, became 100% by using the following calculation:

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

To normalize the post-intervention peak amplitude data, the following equation was used:

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

The pre- and post-mean muscle activity was calculated using the following equation:

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

At the initial consultation prior to the interventions, three trials of force output were recorded during MVIC of each muscle. The raw force output data was utilized. The maximum force output was obtained by using the maximum force output from the three readings, this was recorded per muscle. The same procedure was followed post intervention/control. The difference in maximum force output between pre-post intervention/control was calculated (i.e. pre – post = difference).

3.9.2 Data analysis

The data was then analysed using the latest version of SPSS (IBM SPSS Statistics 2015). Descriptive statistics were used to describe the numerical data in terms of means and standard deviation, while percentage and count described the categorical data. Inferential statistics were used to determine relationships between the data, using a *p* value of less than 0.05 as statistically significant. Fishers exact test assessed for relationships between categorical data. Normality of the data was assessed and paired student *t*-tests were used to determine pre- and post- within group differences, per

muscle. While independent student *t*-tests determined differences between the groups at baseline and following the intervention, per each muscle of the POS.

Pearson's correlation co-efficient was used to assess for correlation between the two muscles in each of the slings i.e. the left latissimus dorsi and the right gluteus maximus (A) and the right latissimus dorsi and left gluteus maximus (B), as seen in Figure 5. A strong correlation would occur if $r \geq 0.8$.

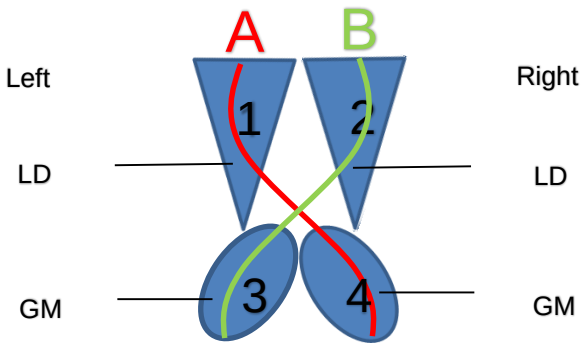


Figure 5 An illustration of the numbering of the muscles and slings for statistical purposes

3.10 Ethical considerations

In this study the ethical pillar of autonomy was adhered to by ensuring that the participants signed a letter of information and informed consent at the initial consultation, as well as confirmation verbally that they understood the procedure. No coercion was used to recruit participants. In addition, participant confidentiality was ensured by allocating codes to the participants, whereby no participant names appeared in the dissertation or any possible publication resulting from this research.

This study poses the possibility that the participant may be a part of an intervention or a control group. In order to not disadvantage the participants in these groups, all participants were offered one free treatment given by the researcher (valid for six months after their research appointment). At the start of the study all participants were informed that they would have a chance of being allocated to any one of the groups. As the patients were asymptomatic, they were not being withheld treatment.

In this study the ethical pillar of justice was adhered to by not being discriminative in participant selection in terms of race, gender, nationality and religion.

One of the inclusion criteria for taking part in this study was that the participants must be asymptomatic. However, if any illness or pathology was diagnosed at the initial consultation (or at any point during the research process) the participant was referred to the appropriate practitioner. The testing of the gluteus maximus and latissimus dorsi muscle activity via MVIC using sEMG has no known risk factors, and was done under the supervision of the researcher. If the participant developed any pain, stiffness or discomfort in the region of these muscles or the SIJ adjusted within 24 hours after the study, they would have been examined and treated by the researcher using conventional chiropractic treatment such as spinal manipulative therapy, mobilisation, dry needling, soft-tissue therapies and electrical modalities (as deemed appropriate by the researcher) – however this was not necessary as there were no reported adverse reactions to the study.

If the participant wished to withdraw from the study, they were free to do so at any point, with no adverse consequences.

Permission was obtained prior to placing advertisements in the locations mentioned (Appendix J). The DUT Institutional Research Ethics Committee granted full ethical approval prior to the commencement of the study – clearance number 187/18 (Appendix Q). The study was registered with PACTR (Appendix R).

In this study the ethical pillar of non-maleficence and benefit was adhered to by protecting the welfare of the participants, as the interventions and machines that were utilised are safe and registered. As mentioned, a free treatment was offered to the participants as compensation for participating in the study and to ensure that all of their treatment needs were met (not only the areas being treated in this study). The research study was conducted at the Chiropractic Day Clinic under permission from the clinic director (Appendix H), and was under the indemnity cover relating to the Chiropractic Day Clinic. All participant data will be kept in the participant's clinical file in the CDC, under a code, with the signed letter of information and informed consent being kept in

the Chiropractic programme. After a period of five years the research data will be shredded. The results of this study will further the understanding of how manipulation results in its biomechanical and neurophysiological effects; resulting in increased scientific support of joint manipulation as a treatment intervention.

CHAPTER FOUR

4.1 Introduction

This chapter presents the results obtained in the study. The data will be presented in the form of graphs and cross tabulations.

4.2 Consort flow diagram

Figure 6 illustrates, in terms of consort flow diagram, the number of participants that responded to the advertisement and were either excluded or completed data collection.

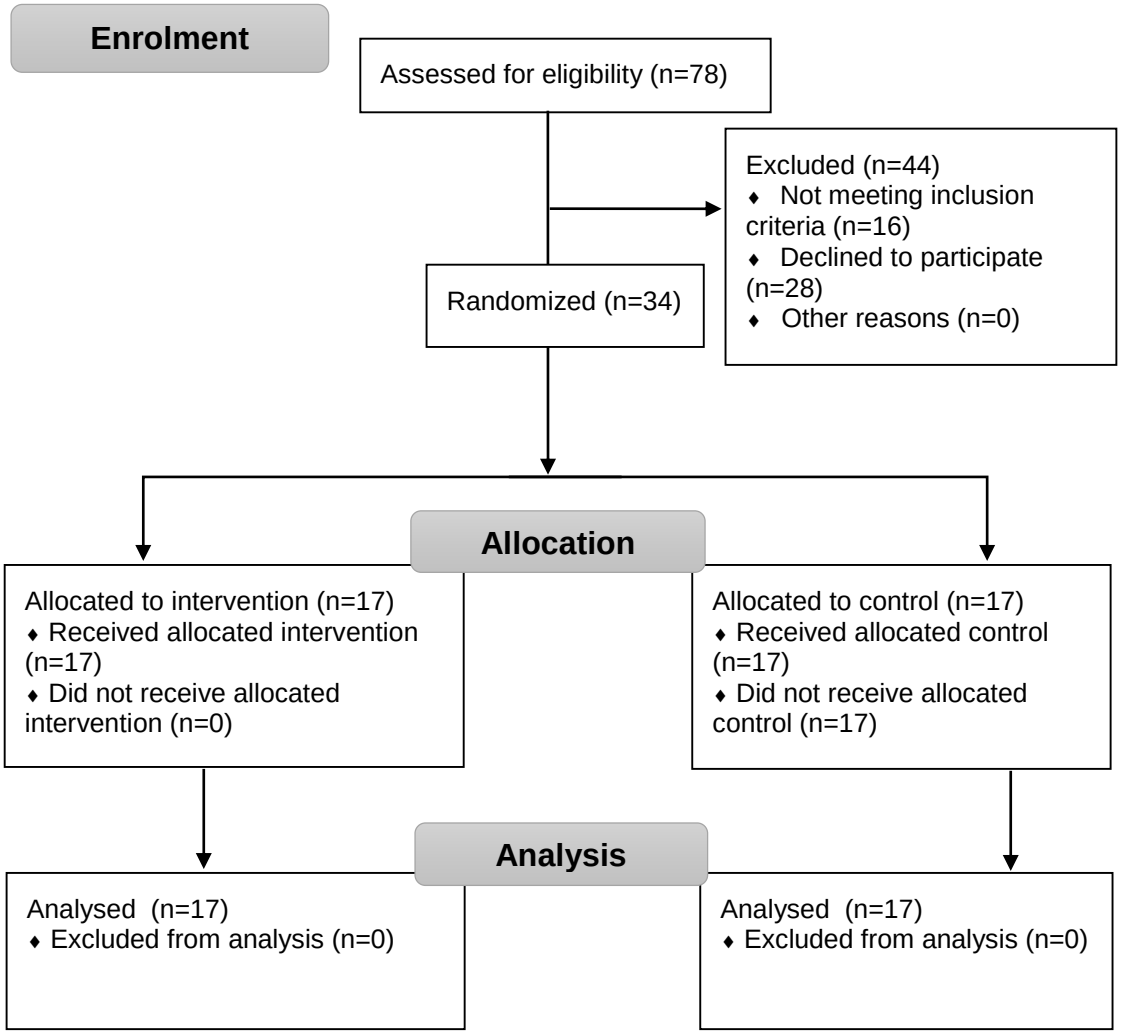


Figure 6 Consort flow diagram

4.3 Demographic characteristics

4.3.1 Age

The mean age of all the participants was 23.15 (± 5.29), with a range from 18 – 45 years. Those in the intervention group had a mean age of 24.0 (±6.92) with those in the control group having a mean age of 22.29 (±2.85). There was no statistically significant difference between the two groups for age (p = 0.355; independent samples t-test).

4.3.2 Race and gender

The majority of participants were from the white populace and were female, as seen in Table 4. There were no significant differences found between the groups for race (p = 0.338; Chi-squared) or gender (p = 0.688; Fisher's exact test).

Table 4 Race and gender distribution per group

		Total	Group		*p* value
			Intervention	Control	
		n (%)	n (%)	n (%)	
Race					
	Black	5 (14.7)	3 (17.6)	2 (11.8)	0.338
	Indian	5 (14.7)	1 (5.9)	4 (23.5)	
	White	24 (70.6)	13 (76.5)	11 (64.7)	
	Total	34 (100)	17 (100)	17 (100)	
Gender					
	Female	26 (76.5)	14 (82.4)	12 (70.6)	0.688
	Male	8 (23.5)	3 (17.6)	5 (29.4)	
	Total	34 (100)	17 (100)	17 (100)	

4.4 Anthropometric data

Table 5 shows the height, weight and BMI of the participants per group. Using an independent samples t-test there are no significant differences between groups for any of these measures (p>0.05).

Table 5 Group statistics for height, weight, and BMI

	Group	Mean (±SD)	*p*-value
Height	Intervention	1.63 (±0.08)	0.104
	Control	1.67 (±0.05)	
Weight	Intervention	64.09 (±11.23)	0.830
	Control	63.32 (±9.41)	
BMI	Intervention	24.10 (±3.03)	0.192
	Control	22.69 (±3.12)	

4.5 SIJ fixation

Table 6 shows the side of SIJ fixation per group. There were no significant differences (p=0.473, Fischer's Exact test) found between the groups for side of SIJ fixation. Therefore, although both groups presented with more left SIJ fixations than right, there is no difference in distribution of SIJ fixation across groups.

Table 6 Group statistics for side of SIJ fixation

SIJ Fixation	Total n (%)	Intervention n (%)	Control n (%)
Right	12 (35)	5 (29)	7 (41)
Left	22 (65)	12 (71)	10 (59)
Total	34 (100)	17 (100)	17 (100)

4.6 Muscle activity

4.6.1 Peak amplitude

Table 7 shows the muscle activity in terms of peak amplitude (mV) measured in the muscles of the POS, assessed before and after the intervention. All the muscles except the left latissimus dorsi showed a trend of increased peak amplitude following both interventions. There were no significant intra or inter-group differences found for peak amplitude in any of the four muscles of the POS.

Table 7 The peak amplitude (mV) measured pre and post intervention for the muscles of the POS

Muscle	Group	Pre % (±SD)	Post % (±SD)	*With-in group p* value*	*Between group p* value**
L LD	Intervention	100 (±0)	95.67 (±18.06)	0.337	0.777
	Control	100 (±0)	97.78 (±24.57)	0.714	
R GM	Intervention	100 (±0)	110.43 (±63.68)	0.509	0.592
	Control	100 (±0)	123.74 (±98.31)	0.334	
R LD	Intervention	100 (±0)	107.63 (±36.47)	0.401	0.759
	Control	100 (±0)	107.90 (±36.88)	0.390	
L GM	Intervention	100 (±0)	105.24 (±24.23)	0.386	0.715
	Control	100 (±0)	101.77 (±30.35)	0.813	

(L=left; R=right; LD=Latissimus dorsi; GM=Gluteus Maximus)
**p* value paired t-test (intra-group)
***p* value independent samples t-test (inter-group)

4.6.2 Mean muscle activity

At baseline there were no significant differences found between the groups for mean muscles activity in the four muscles of the POS; left LD (p=0.984), right GM (p=0.479), right LD (p=0.716), left GM (p=0.091). There is a trend of effect that the control group increased MA with relatively no change in intervention for the GM muscles; and left LD mean MA decreased in both groups, and mean MA in the right LD increased in both groups. However, Table 8 shows that there were no significant differences found for the intra-group and inter-group comparisons of mean muscle activity per muscle.

Table 8 Pre and post intervention mean muscle activity (mV) for the muscles of the POS

Muscle	Group	Pre % (±SD)	Post % (±SD)	*With-in group p* value*	*Between group p* value**
L LD	Intervention	38.24 (±5.27)	37.58 (±9.37)	0.691	0.679
	Control	38.28 (±6.16)	36.58 (±9.74)	0.378	
R GM	Intervention	41.74 (±34.31)	41.81 (±25.85)	0.994	0.871
	Control	35.68 (±6.28)	37.53 (±19.83)	0.671	
R LD	Intervention	38.73 (±4.41)	42.75 (±16.86)	0.262	0.278
	Control	38.12 (±5.12)	38.13 (±6.00)	0.996	
L GM	Intervention	41.44 (±27.45)	39.75 (±9.48)	0.825	0.493
	Control	29.67 (±4.74)	33.43 (±11.73)	0.124	

(L=left; R=right; LD=Latissimus dorsi; GM=Gluteus Maximus)
**p* value paired t-test (intra-group)
***p* value independent samples t-test (inter-group)

4.6.3 Correlations between the two muscles within each sling

There were no significant correlations found when the muscles within each of the POS slings were assessed for peak amplitude and mean muscle activity as shown in Table 9.

Table 9 Correlation between peak amplitude and mean muscle activity of the two muscles within each of the POS slings within each group

	Sling	Group	Pearson Correlation	*p* value
Peak amplitude	**A**	Intervention	0.336	0.188
		Control	0.230	0.374
	B	Intervention	0.246	0.342
		Control	0.135	0.607
Mean	**A**	Intervention	0.014	0.959
		Control	0.063	0.810
	B	Intervention	0.101	0.699
		Control	0.287	0.264

(Sling A = Left LD and right GM; Sling B = Right LD and left GM)

4.7 Force output

There was no difference in baseline values between groups for each muscle: left LD (p=0.123); right GM (p=0.935); right LD (p=0.352); left GM (p=0.849). Table 10 indicates that there was a significant difference between pre and post maximum force output in the right GM for both groups, and in the right LD for the control group only. However, there was no significant difference between group results for any muscle. The difference in maximum force output between pre-post intervention/control was calculated (i.e. pre – post = difference), indicating that the force output was raised in bilateral LD in both groups, and was decreased in bilateral GM in both groups.

Table 10 Maximum force output (kg) for each muscle pre-post intervention/control

	Group	Pre (kg)	Post (kg)	Difference (±SD)	*With-in group* ***p* value***	*Between group* ***p* value****
L LD	Intervention	7.16	7.69	0.53 (±1.42)	0.141	0.690
	Control	9.16	9.91	0.75 (±1.65)	0.081	
R GM	Intervention	9.30	8.63	-0.67 (±1.02)	0.016	0.807
	Control	9.27	8.69	-0.58 (±1.00)	0.030	
R LD	Intervention	7.97	8.31	0.33 (±0.93)	0.156	0.068
	Control	9.25	11.23	1.98 (±3.48)	0.032	
L GM	Intervention	9.42	9.25	-0.18 (±0.71)	0.325	0.548
	Control	9.38	9.02	-0.35 (±0.96)	0.151	

(L=left; R=right; LD=Latissimus dorsi; GM=Gluteus Maximus)
*p value paired t-test (intra-group)
**p value independent samples t-test (inter-group)

CHAPTER FIVE

5.1 Introduction

This chapter discusses the results of this study in the context of the current literature.

5.2 Demographic and anthropometric characteristics

The age range of the participants in this study was restricted to between 18 and 45 years of age. This was done to negate the necessity for parental consent and to limit the likelihood of having participants in the study with spinal degeneration and pathology. It has been reported that these factors are associated with increased age (Walker and Colledge 2013). Age-related loss in neuromuscular function can change maximal motor unit firing rate, force steadiness, agonist muscle activation and antagonist muscle co-activation, leading to an impairment of muscle performance and decreases in maximal muscle strength (Boccia *et al.* 2015). Resulting in the elderly requiring a higher level of muscle activity to produce the identical force produced by a younger individual (Billot *et al.* 2010). It has been recommended that age should be carefully considered when performing sEMG studies. Further supporting why this study limited age range. When the two groups were compared at baseline there were no significant differences (p=0.355) found between them for age, thus mitigating the effect age could have had on the results.

Gender has also been reported to affect sEMG readings, as women and men have been observed to differ in the ability to perform repetitive tasks at high-force levels (Arjunan *et al.* (2011). Although this study placed no restriction on gender, it is unlikely that it influenced the result as there were no differences (p = 0.688) between the groups in terms of gender. South Africa is a multicultural country, as seen by the diverse race groups represented in this study. Although, there was a predominant representation of White individuals with few Black or Indian individuals. There is a scarcity of literature with regards to the effect of race on sEMG readings, however, no differences between the groups were observed (p = 0.338).

Another important consideration when using sEMG is subcutaneous fat, as it acts as an insulator between the electrodes and the muscle. Therefore, sEMG amplitudes may be higher in a thin individual compared to an individual with a thicker subcutaneous fat layer as there is a negative correlation between skinfold thickness and sEMG amplitude (Criswell 2010; Cooper et al. 2014). Patients with a Body Mass Index (BMI) of 30 or higher were excluded from the study to mitigate this effect (Kim, Kang and Oh 2014). Although BMI was regulated, it would be recommendable to measure skin fold thickness as it can vary between individuals and was not controlled in this study. In order to mitigate this the data was normalized to reduce the variability of skin fold thickness differences on muscle activity (Nordander et al. 2003). The impact that skin fold thickness had on these results is undetermined.

5.3 Muscle Activity

Spinal manipulation is theorised to alter the inflow of sensory signals from the affected joint and its associated tissues in a manner that improves physiological function (Pickar 2002). Dishman (2003) showed that manipulation of the cervical and lumbar spine decreased muscle activity in the flexor carpi radialis and gastrocnemius muscles respectively. However, DeVocht, Pickar and Wilder (2005) showed that during the five to ten minutes after the treatment protocol of manipulation of the lumbar or low thoracic spine, both increases and decreases in sEMG levels were observed. Thus, joint dysfunction can result in athrogenic muscle inhibition or facilitation, and manipulation of a joint appears to cause a restoration of the optimal muscle activity (i.e. a decrease in athrogenic muscle inhibition/facilitation) (Pickar 2002; Rice and McNair 2010).

In this study, SIJ manipulation did not result in any significant changes in muscle activity in the muscles of the POS. It was anticipated that the gluteus maximus muscle, especially due to its proximity to the SIJ, would show altered muscle activity readings post SMT. This prediction was made due to previous studies on asymptomatic individuals where there has either been an increase in motor neuron pool excitability (Dunning and Rushton 2009; Niazi *et al.* 2015), or a decrease in spinal reflex excitability after SMT (Fryer and Pearce 2012; Lelic *et al.* 2016). However the results of the current study are in contrast with these studies and are line with Cardinale *et al.* (2015a) who reported no significant improvement in the group receiving L/S SMT group

compared to L/S stretching or sham intervention for muscle activity of the paraspinal and gastrocnemius muscles in asymptomatic participants.

Studies (DeVocht, Pickar and Wilder 2005; Lalanne, Lafond and Descarreaux 2009; Nougarou *et al.* 2013) on symptomatic populations demonstrated that paraspinal muscles become hypertonic when a local joint is dysfunctional and, following the application of manipulative therapy, there is a reduction of MA. In the SIJ it has been shown that, when there is joint dysfunction present, there is hyperactivity of the ipsilateral gluteus maximus and the contralateral latissimus dorsi muscles - resulting in a muscle imbalance (Mooney et al. 2001); these results were supported by Bashir et al. (2019) in symptomatic populations. Women with chronic low back pain (CLBP) have been found to have greater muscle activity in the POS than those without CLBP when performing a prone hip extension task (Kim, Kang and Oh 2014).

Our study aimed to assess the effect of SMT on MA using asymptomatic participants in order to remove the effect that pain may have on muscles and their function. Grindstaff *et al.* (2009) suggested that symptomatic populations that have higher levels of pain and dysfunction may respond differently to SMT than those without/lower levels of pain. From the literature presented this is evident. Using asymptomatic participants in studies like these, aims to add to the literature in terms of the effect of SMT and its effect on muscles that do not have reactive muscle spasm. This study has illustrated that SIJ manipulation has little to no effect on the muscles of the POS.

The manipulation utilised in this study was the toggle-recoil drop piece adjustment applied to the fixated SIJ, and a drop piece sham procedure as the control – whereby there was no force applied to the patient. It is possible that the table dropping away may have provided a therapeutic effect, eliminating the chance of a significant difference in effect of the intervention. Further research should assess the therapeutic effects of a non-contact SIJ drop adjustment. The results may have been different if manual SMT had been utilised as it has been shown to have notable effects on MA in individuals with sub-clinical pain in studies performed by Niazi *et al.* (2015); Lelic *et al.* (2016). Future studies assessing effect of SMT on POS MA should consider using manual SMT techniques.

Another consideration is the side of joint dysfunction. This study included participants with fixations on either the left or right. There were no significant differences between the groups for side of fixation, but this may have affect the results. It would have been worthwhile to do a subgroup analysis to see if the side of fixation influenced the outcomes however, due to the small sample size, it was not possible. Although there were more left SIJ fixations than right, there was no significant difference in distribution of the side of SIJ fixations between groups; therefore, this study may indicate that the theory behind the proposed relationship of the muscles that constitute the POS may be flawed. Future studies should separate the groups further into side of SIJ SMT, or limit SMT to one side, as this could possibly effect the correlative result.

Although the results of this study demonstrate that SIJ manipulation has no effect on the muscles of the POS, SMT has previously been observed to initiate a cascade of neurophysiological events to correct the changes in input from the spine due to joint dysfunction and the effects of sensory motor integration (Haavik and Murphy 2012). Biomechanical changes to the structures of the joint during manipulation (i.e. stretching the capsule/muscles) stimulates mechanoreceptors within the joints and surrounding tissues (Pickar 2002), relaying information along the sensory neuron which synapses with the interneuron - passing on either an excitatory or inhibitory stimulus to the efferent motor neuron. Studies including Symons *et al.* (2000); Pickar (2002); Suter and McMorland (2002a) observed that this impulse may be transmitted to effector muscles, resulting in an increase or decrease of motor neuron pool excitability. It is unclear as to the exact mechanism through which SMT produces its therapeutic effects, however biomechanical, neurophysiological, peripheral, spinal and supraspinal mechanisms have been suggested (Potter, McCarthy and Oldham 2005; Bialosky et al. 2009). Further research is needed in order to understand how these effects may come about, and why there are so many variations in results across studies observing changes in muscle activity.

5.4 Force Output

Studies done by Learman *et al.* (2009); Cardinale *et al.* (2015a) observed SMT to produce no significant effect in terms of force output. However, SMT as well as joint

mobilization have been shown, in other research, to alter force output and activation of musculature (Keller and Colloca 2000; Grindstaff *et al.* 2009). In this study, inter-group analysis found that there was a significant decrease in force output in the right GM in both groups, and an increase in the right LD for the control group pre and post intervention. However, there was no significant difference found between the groups for any of the muscles.

Fatigue of a muscle can be expressed as a reduction in the muscle's maximal voluntary contraction (MVC) (Palomino *et al*. 2018). This research utilized three maximum voluntary isometric contractions (MVICs) as tests before and after the intervention/control. The 'force-fatigability relationship' implies that the greater the force exerted by a muscle, the quicker it will fatigue and reach its failing point (i.e. it will be unable to maintain the force required) (Palomino *et al*. 2018). The decreased force output noted within the GM muscles after repeating the MVICs could be as a result of fatigue. However, one would have expected the fatigue effect to be bilateral, yet the decrease was only observed on the right side. In addition, the lack of significant differences between the groups in terms of side of fixation indicate that it is unlikely that this would have influenced this finding. A long-lever MVIC was utilised to activate the GM. This procedure also allowed for activation of the hamstring muscle and did not purely isolate the GM. Although the research tried to ensure that the participants kept their leg straight during the MVC's it is possible that participants may have flexed the knee when fatigue of the GM set in, decreasing the force output readings as the hamstring began to compensate.

The difference in maximum force output between pre-post intervention/control indicates a trend of effect in that the force output was raised in bilateral LD in both groups, and was decreased in bilateral GM in both groups. Christiansen *et al.* (2018) suggested that force output is mediated by neuromuscular mechanisms, and can be studied well by measuring MVC. Therefore, the method used to obtain force output has been verified. Due to the fact that both groups experienced a similar response to the respective interventions, it possibly indicates a therapeutic effect of both the SMT, and the non-contact drop protocol used in the control group. This study indicates a need for further research into the therapeutic effects of a non-contact drop protocol.

This study's results show no statistically significant difference between the groups in terms of force output, indicating that SIJ SMT may not have an effect on POS strength in healthy pain free individuals. Although the result of this study refutes the literature observing an effect of SMT on force output, it is possible that, with a larger sample size, a positive effect may have been observed.

CHAPTER SIX

Conclusion and Recommendations

6.1 Conclusion

The aim of this study was to determine the effect of a unilateral SIJ manipulation on the muscle activity (mean and peak amplitude) and peak force output of muscles in the POS (gluteus maximus and latissimus dorsi) compared to a control intervention in asymptomatic participants. The study used a pre-test post-test design. The results of this study failed to provide evidence to support the null hypothesis that stated that there would be a significant difference (p<0.05) between the group receiving SMT and the control group, in terms of peak amplitude and mean muscle activity and peak force output of the gluteus maximus and latissimus dorsi muscles. Consideration should be given to the way SMT is administered, the type of sham intervention used and the protocol to elicit MVC.

6.2 Limitations

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

1. The sample size (n=34) was small, it was possible the study could have shown a different treatment effect had the sample size been larger.
2. The results of the study may not be applicable to symptomatic populations as it was conducted using asymptomatic participants.
3. The subcutaneous fat layer thickness above the GM and LD muscles was not controlled between participants, which may have affected sEMG data as it can act as an insulator between electrode and muscle.
4. Although much effort as was taken to ensure reproducibility - the exact sEMG electrode placement between participants could not be standardised or verified.
5. Although the same researcher delivered the SIJ SMT to all participants, the magnitude of the thrust could not be standardised between participants.
6. The position and technique of MVIC was demonstrated thoroughly to each participant, however MVIC is a subjective measure, therefore the researcher

could not control whether or not the participant was truly contracting at their maximum capacity.

7. The resistive straps used to perform the MVIC were not adjustable, therefore the resistance may have varied between individuals.

6.3 Recommendations

1. Future research should consider observing the MA changes that occur in the POS muscles in individuals with an asymptomatic SIJ fixation prior to SMT, in order to better quantify the changes observed in this study (i.e. post SMT).
2. A larger sample size should be used in future studies in order to further examine the possible trend found in this study.
3. Future studies should consider performing this protocol on a symptomatic population which may produce different, yet more clinically relevant results.
4. Skin fold thickness should be controlled for in future studies and should be included as part of the participant inclusion criteria.
5. A research assistant may be used in future studies in order to remove any potential bias; to deliver the intervention or conduct the sEMG study.
6. Adjustable straps should be used in future studies to ensure repeatability of MVIC.
7. A study may be performed using alternative manipulative techniques, which may produce significant results. This should be considered due to the varying effects of different grades of mobilization.
8. Future studies should research the therapeutic effects of a non-contact drop intervention.
9. The study may be redone using only participants with either a left or right fixation, or otherwise separate right and left into subgroups within the treatment and control groups, in order to better quantify the correlation of the muscles within each sling.
10. This research only considered asymptomatic vs symptomatic participants. There is a third possible option of subclinical subjects, which should be considered in future research.

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APPENDICES

Appendix A: Ligaments of the sacral region

Ligaments of the sacral region (Bogduk 2005; Bergmann and Peterson 2010)

Ligament	Attachments	Function
Interosseous sacroiliac ligament	Lies between the sacrum and ilium. Deep layer attaches medially to the three fossae on the lateral aspect of the posterior sacral surface, and laterally to the iliac tuberosity. Superficial layer is a fibrous sheet which attaches to the lateral crest at S1 and S2 and the medial aspect of the iliac crest.	Primary stabilizer of S1 joint strongly binding the sacrum and the ilium. Provides multidirectional structural stability of the joint, and transfers weight between axial and inferior appendicular skeleton.
Iliolumbar ligament	L4 and L5 TVP to the iliac crest.	Primary stabilizer of S1 joint reinforcing the anterior portion of the joint. Strengthens the lumbosacral joints. Resists extension, lateral flexion, and axial rotation of L5-S1.
Anterior sacroiliac ligament	Thickening of the anterior and inferior aspects of the joint capsule. Covers anterior aspect of the SIJ. Consists of numerous long, transversely orientated fibres extending from the ala and anterior surface of the sacrum to the anterior surface of the ilium.	Weakest ligament in this group. Binds the ilium to the sacrum and prevents anterior diastasis of the SIJ.
Posterior sacroiliac ligament	Lies posterior to the interosseous ligament. Long posterior SI ligament consists of long fibres attaching medially to the lateral crest of S3 and S4, and laterally to the PSIS and inner lip of the iliac crest, mixing with the sacrotuberous ligaments.	Primary stabilizer of S1 joint reinforcing the posterior portion of the joint. Restrains counternutation of the sacrum and assists in force closure of the SIJ. Short posterior longitudinal ligament and interosseous ligament prevent posterior diastasis of the SIJ.
Sacrospinous ligament	Triangular shaped. Has a broad origin medially from the lower lateral edge of the sacrum below the SIJ, and on the upper edge of the coccyx. Laterally the apex of the triangular ligament attaches to the ischial	Secondary stabilizer of the SIJ. Restrains nutation of the sacrum.

	spine. Proximally the fibres blend with the joint capsule of the SIJ.	
Sacrotuberous ligament	Arises from the PSIS, and blends with the long posterior SI ligaments; from the transverse tubercles of the lower spinal segments and lateral margin of the sacrum; where it blends with the sacrospinous ligament. It then narrows and broadens again to attach to the medial margin of the ischial tuberosity. Composed of three large fibrous bands: Lateral band attaches to the PSIS and ischial tuberosity. Medial band attaches to S3, S4 and S5 transverse tubercles and the lateral margin of the lower sacrum and coccyx, and runs anteroinferolaterally to the ischial tuberosity. Superior band runs superficially to the interosseous ligament and attaches to the PSIS and coccyx.	Secondary stabilizer of the SIJ. Restrains nutation of the sacrum.
Anterior longitudinal ligament	Long band which covers and connects anterior and lateral aspects of the VBs and IVDs. Extends from the pelvic surface of the sacrum to the anterior tubercle of C1 vertebra and the occipital bone anterior to foramen magnum	Prevents hyperextension of the spine, therefore maintains stability of the joints between the VBs
Posterior longitudinal ligament	Narrower, weaker band. Extends from C2 to the sacrum. Runs within the vertebral canal along posterior aspects of the VBs, and expands laterally to attach to the IVDs.	Weakly resists hyperflexion of the spine, and prevents or redirects posterior herniation of the nucleus pulposis.

Appendix B: Muscles of the posterior oblique sling

Muscles of the POS (Muscolino 2016)

Muscle	Proximal Attachment	Distal Attachment	Action	Segmental Innervation	Distal Innervation
Gluteus Maximus	Iliac crest to posterior gluteal line, lateral surface of ilium, thoracolumbar fascia, dorsal surface of sacrum and coccyx, sacrotuberous ligament	Iliotibial tract (that inserts into lateral condyle of tibia), greater trochanter and gluteal tuberosity of femur	Extends and laterally rotates hip	L5, S1-2	Inferior gluteal nerve
Latissimus Dorsi	Spinous processes of T7-L5, thoracolumbar fascia, iliac crest, ribs 8-12	Inter-tubercular sulcus of humerus	Extends, abducts, and medially rotates humerus	C6-7-8	Thoraco-dorsal nerve

Appendix C: Structure of the thoracolumbar fascia

Layers of the thoracolumbar fascia (Bogduk 2005)

Layers of the TLF	Attachments
Anterior	Is thin, covering the anterior aspect of the quadratus lumborum muscle. It attaches medially to the anterior aspects of the lumbar transverse processes.
Middle	Lies posteriorly to the quadratus lumborum muscle, attaching medially to the tips of the lumbar transverse processes, and laterally forms the aponeurosis of the transverse abdominus muscle.
Posterior	Covers the back muscles and infuses with the other layers of TLF along the lateral border of the iliocostalis lumborum, while attaching medially to the lumbar spinous processes

Appendix D: Classification of sensory and motor nerve fibres

Classification of sensory nerve fibres (Pickar 2002; Leach 2004; Mai and Paxinos 2011)

Type	Size (μm) and conduction velocity (m/s)	Myelin	Characteristics	Associated receptor
Ia	12-20; 80-120	Yes	Responds to rate of length change in a muscle	Muscle spindle
Ib	12-20; 80-120	Yes	Responds to tension changes in a muscle	Golgi tendon organ
II	5-15; 35-75	Yes	Stretch receptor, non-adapting	Secondary receptors of muscle spindles, all cutaneous mechanoreceptors
III	1-5; 3-35	Thin	Responds to pain	Free nerve endings for touch and pressure, nociceptors of neospinothalamic tract and cold receptors
IV	0.2-1.5; 0.5-2	No	Responds to pain	Nociceptors of paleospinothalamic tract and warmth receptors

Classification of motor nerve fibres (Pickar 2002; Leach 2004; Bergmann and Peterson 2010; Mai and Paxinos 2011)

Type	**Size and conduction velocity**	**Myelin**	**Function**
Alpha (α)	Largest (8-20 μm) and fastest (35-120 m/s)	Yes	Innervate the slow (for posture) and fast (for **movement**) twitch fibres of the **extrafusal** muscle fibres.
Beta (β)	Medium size and velocity.	Yes	Innervate the slow (for posture) twitch fibres of the extrafusal muscle fibres, and intrafusal fibres of muscle spindles
Gamma (γ)	Smallest (2-8 μm) and slowest (10-50 m/s)	Yes	Innervate the **intrafusal** muscle fibres and together with the muscle **spindles** give **proprioceptive** feedback

Appendix E: Classification of receptors

Classification of receptors and their functions (Pickar 2002; Leach 2004; McGlone and Reilly 2010; Muscolino 2016)

Receptor type		Location	Sensations	Adaption rate
Mechano-receptors	**Meissner corpuscles**	Hairless skin	Fine touch, pressure & slow vibrations	Rapid
	Merkel discs	Epidermis	Fine touch & pressure	Slow
	Type I: Ruffini corpuscles	Deep in the dermis, in ligaments & tendons, periosteum and superficial layer of the joint capsule	Stretching of skin, static joint position. Active and passive joint movements	Static and dynamic, low threshold, slow adapting
	Type II: Pacinian corpuscles	Dermis, subcutaneous layer, submucosal tissues, joint capsule and articular fat pad, periosteum & some viscera	Pressure, fast vibrations. Active and passive joint movements	Dynamic, low threshold, rapid adapting
	Muscle spindles	Within most striated skeletal muscles	Muscle length	Slow
	Type III: Golgi tendon organs	Ligaments and tendons	Muscle tension	Dynamic, high threshold, slow adapting
Pain receptors/ nociceptors	**Type IV 'free nerve endings'**	Joint capsule, joint fat pads and blood vessels walls. Free nerve endings – intrinsic and extrinsic ligaments	Pain	High threshold, non-adapting

Appendix F: The stretch reflex

Receptor types

1. Muscle spindles

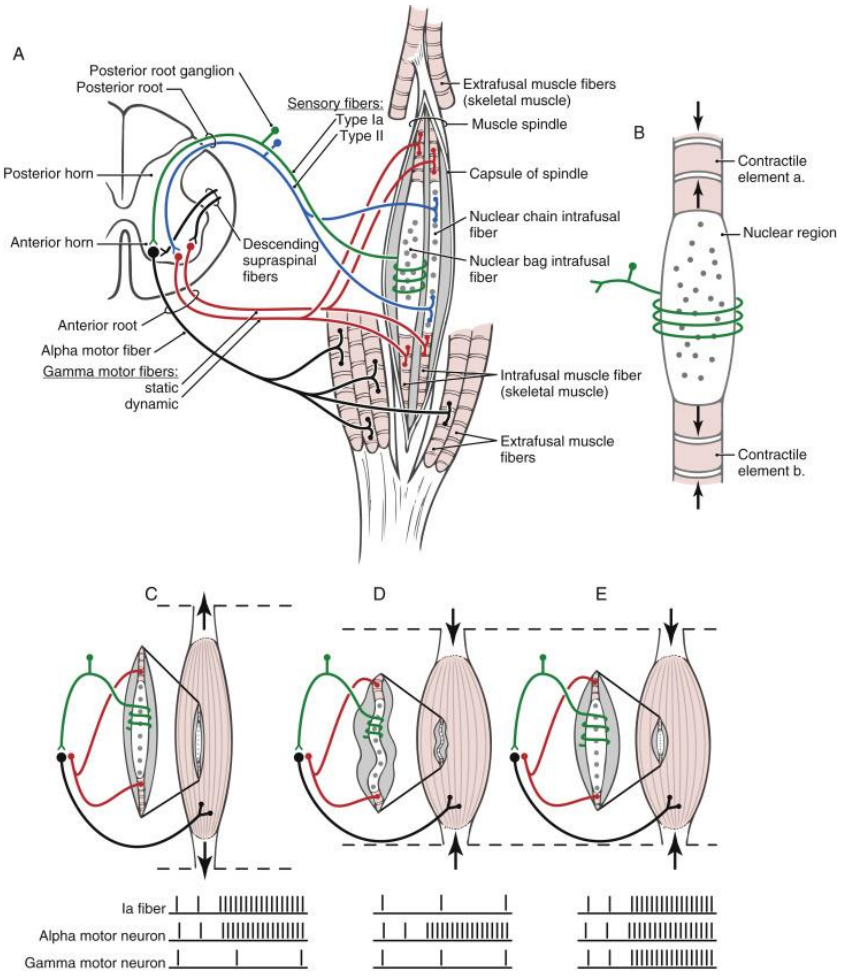
Muscle spindles are located within skeletal muscles, and are responsible for measuring a muscle's length (stretch). Each spindle is composed of two to ten intrafusal muscle fibres encapsulated by a thin connective tissue capsule. These intrafusal muscle fibres are stretch sensitive and are located within the belly of the muscle. The intrafusal fibres lie parallel to and attach to the surrounding extrafusal muscle fibres (see fig. 8).

There are two types of sensory nerve endings in the muscle spindle: primary nerve endings located at the centre of the intrafusal fibres, consisting of a single, large nerve fibre that branches out - referred to as annulospiral endings; and secondary endings that exist on nuclear chain fibres near the annulospiral endings, consisting of smaller nerve fibres that branch out in a different pattern to the annulospiral endings - known as flower-spray endings. These annulospiral and flower-spray sensory nerve endings both respond to stretch, while the annulospiral nerve endings also respond to the rate of the muscle lengthening (Parvizi and Kim 2010; Haines 2012).

Stretching of the spindle initiates stimulation of the sensory nerve endings, which is then relayed to the SC via Type Ia (annulospiral) and Type II (flower-spray) afferent neurons which synapse with alpha and gamma motor neurons in the anterior horn.

The central part of the intrafusal muscle fibre has no actin or myosin and, hence, it does not contract. The spindle is primarily for sensory perception. However, the end portions of the intrafusal fibre are able to contract when stimulated by gamma motor neurons (Parvizi and Kim 2010; Snell 2010; Tortora and Derrickson 2017). Extrafusal muscle fibres are innervated by the alpha motor neurons - stimulating contraction of the extrafusal muscle fibres, resulting in shortening of the muscle. This reflex that protects the muscle from being overstretched or torn is known as the stretch reflex.

Gamma motor neurons adjust the tension of the muscle spindle according to muscle length variations. So, while there is a contraction of the extrafusal muscle fibres, the gamma motor neurons are also firing; which causes the ends of the spindle to contract – resultantly pulling on the non-contractile central portion, making it taut and increasing its sensitivity to stretch and, therefore, more easily triggering the stretch reflex. This cycle is known as 'the gamma loop' whereby Ia sensory neurons transmit a change in muscle length to the SC resulting in activation of the alpha motor neurons and a resultant change in muscle length.



Neuroanatomy of the stretch reflex (Haines 2012)

All muscles/muscle systems have agonistic/antagonistic relationships. Therefore, to enhance the effectiveness of the stretch reflex, axon collaterals from the muscle spindle neurons simultaneously excite inhibitory interneurons too, producing an inhibitory effect on the alpha motor neurons of the opposing muscle group. This causes

relaxation of the opposing muscle group, known as reciprocal inhibition (Muscolino 2014).

2. Golgi tendon organs

Golgi tendon organs (GTO) are located at musculotendinous junctions and provide information about the force exerted by a contracting muscle (i.e. the tension in a muscle). The GTO relays this information as part of a protective mechanism to prevent a muscle from over-contracting – this is called the tendon reflex.

As a muscle contracts there is a pull on its tendon, causing the tension to increase, resulting in GTO stimulation. The stimulus is sent to the SC via Type Ib afferent sensory fibres. At the SC, they synapse with alpha motor neurons in the anterior horn. A negative feedback response follows, inhibiting further muscle contraction. The product is decreased muscle tension, protecting the muscle from over-contracting (Muscolino 2014; Muscolino 2016).

The relationship between the muscle spindle and GTO is responsible for the tone of a muscle (Haines 2012).

Appendix G: Permission to conduct research from research director

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

15th March 2019

Ms Aimee Paige McNally
c/o Department of Chiropractic and Somatology
Faculty of Health Sciences
Durban University of Technology

Dear Ms McNally

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 a unilateral sacroiliac joint manipulation on muscle activity and force output in the posterior oblique sling muscles" at the Durban University of Technology.

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

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

Kindest regards.
Yours sincerely

PROF CARIN NAPIER
DIRECTOR (ACTING): RESEARCH AND POSTGRADUATE SUPPORT DIRECORATE

Appendix H: Permission from clinic director to use DUT CDC

MEMORANDUM

To : Prof Adam
Chair: IREC

From : Prof A Ross
Deputy Dean: Faculty of Health Sciences

Dr Desiree Varatharajullu
Clinic Director Chiropractic Day Clinic: Chiropractic

Date : 12.03.2019

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

---

Permission is hereby granted to :

Miss Aimee Paige McNally (Student Number: 21417296)

Research title : "The effect of a unilateral sacroiliac joint manipulation on muscle activity and force output in the posterior oblique sling muscles"

Miss McNally, is requested to submit a copy of her FRC / IREC approved proposal along with proof of her M.Tech:Chiropractic registration to the Clinic Administrators 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

Prof AHA Ross
Deputy Dean
Faculty of Health Sciences

Dr Desiree Varatharajullu
Clinic Director: Chiropractic Day Clinic:
Chiropractic

Cc: Mrs Linda Twiggs: Chiropractic Day Clinic
Dr A. Abdul-Rasheed: Research co-ordinator
Dr L. O'Connor and Dr A. Abdul-Rasheed: Research supervisors

Appendix I: Advertisement

Treatment even if you're PAIN- FREE!

Did you know?

Muscle imbalances can predispose you to various injuries,

BUT…

Chiropractic treatment has been shown to have a positive effect on muscle activity!

If you are between the **ages of 18 and 40**, and would like to take part in a study aimed at correcting abnormal muscle activity (preventing your body from injury), please contact:

The DUT Chiropractic Day Clinic : 031 373 2205
Aimee McNally : 081 010 8517
Or email aimeemcnall@gmail.com

Appendix J: Permission to place advertisement

Letter requesting permission to place advertisements

To whom it may concern

This letter is to request permission to place advertisements regarding participant recruitment for a research study.

Title of study: The effect of a unilateral sacroiliac joint adjustment on muscle activity and strength in the posterior oblique sling muscles.

Brief Introduction and Purpose of the Study: The surface electromyography (sEMG) machine will be used to assess the electrical activity latissimus dorsi and gluteus maximus muscles, while the strength output will be measured using a dynamometer. You will be required to attend an appointment at the Durban University of Technology during which you will be randomly allocated to either the test, sham, or control group to allow unbias allocation. A full patient history, physical exam and regional evaluation of the lower back (sacro-iliac joints and lumbar spine) will be performed. Contraindications to sacroiliac joint (SIJ) manipulation will also be evaluated for. You will then be screened in an effort to identify any fixations in their sacroiliac joints. The researcher will then take the time to explain to you how the sEMG and dynamometer works. You will be allocated time to practise the required maximum voluntary isometric contraction. Three pre-tests for all muscles will then be taken. The intervention will then be performed and three post-tests for each muscle will be done. The data will be recorded and securely filed.

Statement for permission to place advertisements:

I..... (Full name), ID number ....., have read this document in its entirety and understand its contents. Any questions have been answered and explained to me sufficiently by..... I hereby grant permission for advertisements to be placed at..... (Full name of facility/location).

Name.....

Signature..... Date.....

Researcher's name.....

Researcher's signature..... Date.....

Witness' name.....

Witness' signature..... Date.....

Appendix K: Letter of information and consent

LETTER OF INFORMATION

Dear Participant

Thank you for taking the time to join this research project

Title of the research study: The effect of a unilateral sacroiliac joint manipulation on muscle activity and force output in the posterior oblique sling muscles.

Researcher: Aimee Paige McNally, B. Tech Chiropractic

Supervisor/s: Dr Laura O'Connor M. Tech Chiropractic and Dr Ashura Abdul-Rasheed M. Tech Chiropractic

Brief introduction and purpose of the study: The aim of this study is to determine the effect of a lower back manipulation on the activity and strength within the gluteus maximus and opposite latissimus dorsi muscles. The surface electromyography (sEMG) machine will be used to assess the electrical activity (excitability) of the muscles, while the strength will be measured using a dynamometer.

Outline of the procedures: You will be required to attend an appointment at the Durban University of Technology during which you will be randomly put into one of two groups (either the control, sham, or intervention group.) A full history, physical exam and regional evaluation of your lower back will be performed by the researcher. Anything found that contraindicates treatment will be noted by the researcher, and you will be excluded from the study. If no contraindications are found, you will then be screened in an effort to identify any fixations in the sacroiliac joints. The researcher will then take the time to explain to you how the sEMG and dynamometer works. You will be allocated time to practice the movements you will be asked to perform. Three pre-tests for all muscles will then be taken. The intervention will then be performed and three post-tests for each muscle will be done. The data will be recorded and securely filed.

Risks or discomforts to the participant: You may experience transient muscle pain in your lower back region following treatment, however this will subside within 24 hours – if it fails to do so please contact me.

Benefits: You will be given one free treatment following the completion of the research procedure. Due to the nature of the research investigating the effects of SIJ manipulation on the posterior oblique sling muscles, the intervention may prevent lower back pain as well as

other injuries that have been associated with abnormal walking patterns.

Reason/s why the participant may be withdrawn from the study: If you do not present with a SIJ fixation you may be withdrawn from the study. You may not receive any form of chiropractic manipulative treatment on your lower back for four weeks before the study (including chiropractic students.) If you do, you will be withdrawn from the study. If you present with SIJ syndrome or pain experienced in your lower back in the three weeks prior to the study, you will be excluded from the study. If you present with contraindications to the usage of the surface electromyography machine (sEMG) including, but not limited to: open wounds, rashes, psoriasis, skin irriattions, or skin conditions of any kind in the region of electrode placement (the researcher will point out these locations); you will be excluded from the study. If you are taking any drugs which have effects on the skeletal muscles, you will be excluded from the study.

Remuneration: There will be no payment for your participation in this study. However, you will be issued with a voucher for one free treatment at the DUT Chiropractic Day Clinic (valid for 3 months after the date of your appointment.)

Costs of the Study: You will not be expected to contribute to any of the costs of the study. The only requirement will be that you give up your time to attend one appointment – that should last about two hours.

Confidentiality: Any information given by you will only be accessible to the researcher, the supervisor and the co-supervisor for data collection purposes.

Research-related Injury: Should there be any adverse reactions (such as an allergic reaction to the electrodes, or prolonged pain in response to the manipulation), the researcher will report this to IREC.

Persons to Contact in the Event of Any Problems or Queries: Please contact the researcher (0810108517), my supervisor, Dr Laura O'Connor (084 848 0620) or the Institutional Research Ethics administrator (Ms Lavisha Deonarain) on 031 373 2375 or lavishad@dut.ac.za. Complaints can be reported to the DVC: Prof C Napier on 031 373 2326/2577 or carinn@dut.ac.za.

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 / Right Thumbprint**

I, _____ (name of 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**

2. How is it calculated?

The effect size is just the standardised mean difference between the two groups. In other words:

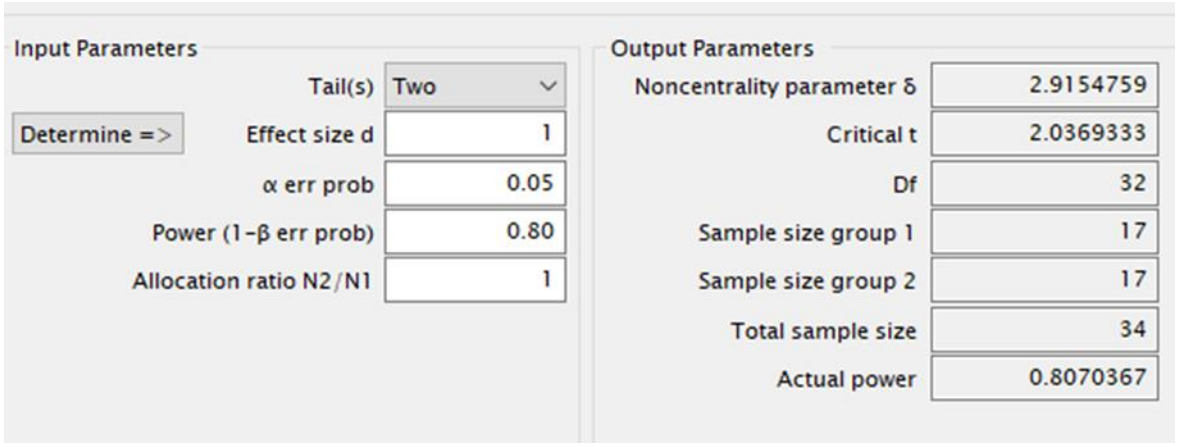
$$\text{Effect Size} = \frac{\text{[Mean of experimental group]} - \text{[Mean of control group]}}{\text{Standard Deviation}}$$

Equation 1

If it is not obvious which of two groups is the 'experimental' (i.e. the one which was given the 'new' treatment being tested) and which the 'control' (the one given the 'standard' treatment - or no treatment - for comparison), the difference can still be calculated. In this case, the 'effect size' simply measures the difference between them, so it is important in quoting the effect size to say which way round the calculation was done.

The 'standard deviation' is a measure of the spread of a set of values. Here it refers to the standard deviation of the population from which the different treatment groups were taken. In practice, however, this is almost never known, so it must be estimated either from the standard deviation of the control group, or from a 'pooled' value from both groups (see question 7, below, for more discussion of this).

In Dowson's time-of-day effects experiment, the standard deviation (SD) = 3.3, so the effect size was (17.9 - 15.2)/3.3 = 0.8.



Appendix M: Telephonic screening interview

Question	Yes	No	Comment
1. How old are you?			
2. Do you have any lower back pain (specifically the SIJ)?			
3. Have you ever had surgery or trauma to the lower back (specifically the SIJ)?			
4. Do you know your height and weight (BMI)?			
5. Are you on any pain medication?			
6. Have you had lower back chiropractic treatment in the last 4 weeks?			
7. Are you pregnant?			

Appendix N: Case history

DUT DURBAN UNIVERSITY OF TECHNOLOGY | DEPARTMENT OF CHIROPRACTIC AND SOMATOLOGY

CHIROPRACTIC PROGRAMME

CHIROPRACTIC DAY CLINIC
CASE HISTORY

Patient: _____ Date: _____

File #: _____ Age: _____

Sex: _____ 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

9. Review of Systems (please highlight with an asterisk those areas that are a problem for the patient and require further investigation)

General

Skin

Head

Eyes

Ears

Nose/Sinuses

Mouth/Throat

Neck

Breasts

Respiratory

Cardiac

Gastro-intestinal

Urinary

Genital

Vascular

Musculoskeletal

Neurologic

Haematological

Endocrine

Psychiatric

Appendix O: 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:**			

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Appendix P: Lumbro-pelvic regional examination

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

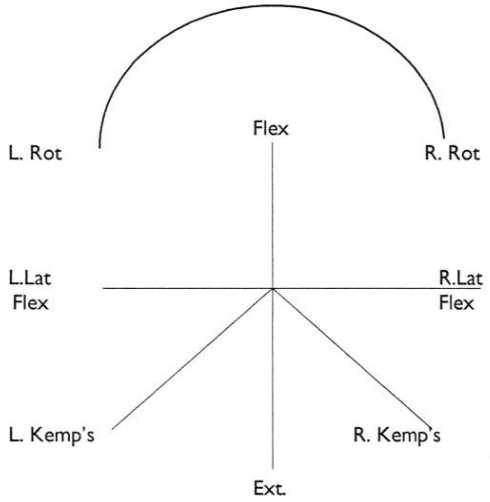
GAIT:

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

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

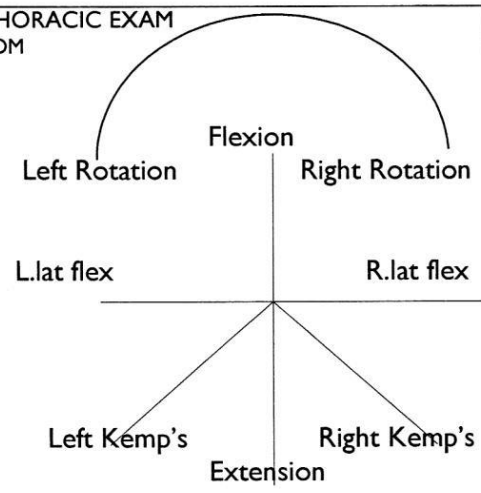
NEUROLOGICAL EXAMINATION

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:

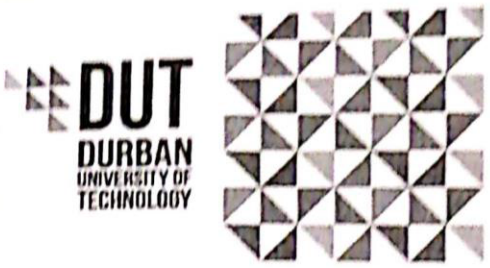
BASIC HIP EXAM

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

B) Supine (hip flexed):
- Trochanteric bursa

MOTION PALPATION AND JOINT PLAY	L	R
Thoracic Spine		
Lumbar Spine		
Sacroiliac Joint		

Appendix Q: IREC approval



Institutional Research Ethics Committee
Research and Postgraduate Support Directorate
3rd Floor, Berarys Court
Gate 1, Steve Biko Campus
Durban University of Technology

P O Box 1334, Durban, South Africa, 4001

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

www.dut.ac.za

26 March 2019

Miss A P McNally
567 Currie Road
Morningside
4001

Dear Miss McNally

The effect of a unilateral sacroiliac joint manipulation on muscle activity and force output in the posterior oblique sling muscles

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

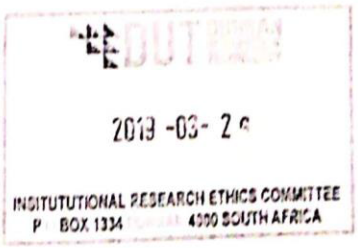
Please note that FULL APPROVAL is granted to your research proposal. You may proceed with data collection.

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

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

Yours Sincerely

Professor J K Adam
Chairperson: IREC



Appendix R: Trial registration

04 July 2019

To Whom It May Concern:

RE: The effect of a unilateral sacroiliac joint manipulation on muscle activity and force output in the posterior
oblique sling muscles

As project manager for the Pan African Clinical Trial Registry (www.pactr.org) database, it is my pleasure to inform you that your application to our registry has been accepted. Your unique identification number for the registry is **PACTR201907862005869**.

Please be advised that 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

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