The effect of ankle joint manipulation on peroneal and soleus muscle activity in chronic ankle instability syndrome

By

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I, Jason Dicks, 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

I dedicate this dissertation to my family: Alan, Antoinette and Tyron. Thank you for the encouragement and belief in me, I could not have done this without you. Your continual support and sacrifices not only towards this dissertation but my entire academic career has not gone unnoticed. I cannot begin to explain how much I appreciate you.
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Abstract

**Purpose:** Ankle sprains are amongst the most common injury sustained by athletes and the general public. When an ankle is repeatedly sprained it results in chronic ankle instability syndrome (CAIS). This repeated trauma results in disruption of the afferent nerve supply from the injured joint, which affects the motor neuron pool excitability of the peroneal and soleus muscles resulting in arthrogenic muscle inhibition (AMI). Traditional treatment for CAIS focuses on rehabilitation of the affected muscles via strength and proprioceptive training. Recent literature has shown that the addition of ankle joint manipulation resulted in improved clinical outcomes in the treatment of CAIS. The mechanism on how joint manipulation affects AMI is under-investigated especially in extremity joints. Thus this study aimed to determine the immediate effect of ankle joint manipulation on peroneal and soleus muscle activity, by assessing surface electromyography (sEMG) H/M ratio to detect a change in the proportion of the total motor neuron pool being recruited, in participants with CAIS.

**Methods:** The study utilised a quantitative, experimental, pre-test post-test study design. Forty two participants with grade I and II CAIS, aged 18-45 years, were randomly allocated into one of three groups. Group one received a single talocrural joint long axis distraction manipulation, group two received a sham manipulation and group three was the control receiving no intervention. sEMG H/M ratio measurements were taken before and immediately after the intervention using a Biopac wireless emg system.

**Results:** The groups were comparable at baseline for age, gender, body mass index and H/M ratio measurements for the soleus and peroneal muscles ($p < 0.050$). Intra-group analysis of the soleus muscle H/M ratio showed no statistically significant change over time for the manipulation ($p = 0.975$) and sham ($p = 0.056$) groups, with the control group showing a statistically significant ($p = 0.019$) decrease in the H/M ratio. For the peroneal muscle no statistically significant ($p > 0.050$) differences were observed in any of the three groups. Inter-group analysis of the soleus muscle H/M ratio measurements showed no statistically significant differences between the three groups ($p = 0.470; F = 1.010$) over time, with Tukey’s HSD post-hoc test revealing a
statistically significant ($p = 0.028$) difference being observed between the sham and control groups in terms of post soleus muscle H/M ratio measurements.

**Conclusion:** This study failed to show that ankle joint manipulation affects the soleus and peroneal muscles in terms of H/M ratio measurements in participants with CAIS. There may have been a trend of an effect of the sham and manipulation interventions counteracting the muscle fatigue experienced in the control group, however further investigation is required.

**Key indexing terms:** chronic ankle instability syndrome, electromyography, manipulation.
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Definitions

**Ankle joint:** articulation between the distal end of the tibia and fibula and the proximal part of the talus. Also referred to as the talocrural joint or mortise joint (Tortora and Derrickson, 2009; Moore *et al.*, 2010; McKinley and O’Loughlin, 2012).

**Joint manipulation:** a manual treatment technique whereby a high velocity, low amplitude thrust is applied to a synovial or cartilaginous joint within the boundaries of anatomical integrity. It may be accompanied by a crack or pop sound known as a cavitation (Pickar, 2002; Maigne and Vautravers, 2003; Kaur *et al.*, 2014; Cardinale *et al.*, 2015).

**Ligament sprain:** excessive stress placed on a ligament resulting in disruption of its structure, the extent of disruption is dependent on the amount of stress placed on the ligament. It often results in pain, swelling and dysfunction (Hertel, 2002; Bonnel *et al.*, 2010).

**Chronic ankle instability syndrome:** the constant tendency of the ankle to re-sprain following an acute ankle sprain. It is associated with recurrent sprains and the feeling of the ankle “giving way” (Gribble *et al.*, 2013).

**Peroneal Longus:** located on the lateral aspect of the leg and responsible for foot eversion. Also known as the fibularis longus and forms part of the peroneal muscle (Tortora and Derrickson, 2009; Moore *et al.*, 2010; McKinley and O’Loughlin, 2012).

**Peroneal Brevis:** located on the lateral aspect of the leg, originates more distal than the peroneal longus, and is responsible for foot eversion. Also known as the fibularis brevis and forms part of the peroneal muscle (Tortora and Derrickson, 2009; Moore *et al.*, 2010; McKinley and O’Loughlin, 2012).

**Peroneal Tertius:** located on the lateral aspect of the leg but more anterior than the peroneal brevis, is responsible for eversion of the foot. Also known as the fibularis tertius and forms part of the peroneal muscle (Tortora and Derrickson, 2009; Moore *et al.*, 2010; McKinley and O’Loughlin, 2012).

**Mechanoreceptors:** mechanically sensitive neurons found within the joint’s structure and surrounding tissues (Tortora and Derrickson, 2009; McKinley and O’Loughlin, 2012).
Golgi tendon organs: nerve endings located within the tendons near the muscle-tendon junction that are stimulated by tension applied onto the tendon and results in relaxation of the muscle belly (McKinley and O’Loughlin, 2012).

Muscle spindles: nerve endings located within the intrafusal muscle fibres of the muscle belly and are stimulated by stretching of the muscle belly (McKinley and O’Loughlin, 2012).

Surface electromyography: an electrical, non-invasive, accurate method of measuring muscle excitation and activation through the placement of electrodes over the muscle being assessed (Sousa and Tavares, 2012).

H-reflex: an electrically induced spinal reflex that is elicited by direct stimulation of the Ia afferent nerve fibres. It is the equivalent of a mechanically induced spinal stretch reflex however, it bypasses the muscle spindles. It measures the motor neuron pool excitability of the muscle being assessed (Palmieri et al., 2004; Knikou, 2008).

M-wave: a response picked up in the muscle as a result of direct stimulation of the alpha motor neurons. The stimulation does not pass through the spinal cord and therefore is not a true reflex. It gives an indication of the muscles full activation potential and is therefore used in normalization of the H-reflex (Palmieri et al., 2004).

H/M ratio: the H-reflex varies greatly between individuals and therefore requires normalization. A method for achieving this is to display the maximum H-reflex as a ratio of the maximum M-wave ($H_{\text{max}}/M_{\text{max}}$). This can be interpreted as the proportion of motor neuron pool capable of being recruited and is an indication of the activity of the muscle (Palmieri et al., 2004; Tucker et al., 2005).
Abbreviations

CAIS: Chronic ankle instability syndrome
AMI: Arthrogenic muscle inhibition
sEMG: Surface electromyography
H/M ratio: Ratio of maximum H-reflex to maximum M-wave ($H_{\text{max}}:M_{\text{max}}$)
N: Number of participants (total sample)
p: Probability value of statistical significance
>: Greater than
<: Less than
±SD: Standard deviation
Min: Minimum
Max: Maximum
RCT: Randomised controlled clinical trial
T/C: Talocrural
NRS: Numerical pain rating scale
JM: Joint manipulation
HVLA: High velocity low amplitude
VAS: Visual analogue scale
FADI: Foot and ankle disability index
WB: Weight bearing
BBS: Berg balance scale
Ca++: Calcium
Chapter One

1.1 Introduction

Chronic ankle instability syndrome (CAIS) can be defined as the tendency of the ankle to re-sprain following an acute ankle sprain (Caufield, 2000). The patient presents with lateral ankle pain, crepitus, edema, weakness, adhesions, joint restrictions and hyper or hypomobility (Caufield, 2000; Pellow and Brantingham, 2001; Ajis and Maffulli, 2006). Joint restrictions occur when an injury results in altered joint arthrokinematics, in CAIS this most commonly results in a decreased ankle joint dorsiflexion and posterior glide of the talus. Joint restrictions that occur most commonly in CAIS are in the talocrural and distal tibiofibular joints, with talocrural joint restrictions being more prevalent in patients with lateral ankle sprains (Denegar et al, 2002).

Following a joint injury, the joint may have distension due to oedema or damage may have occurred to the joint structures. This alters the normal neurophysiological functioning of the mechanoreceptors. Afferent neurons send inhibitory information from the disrupted mechanoreceptors to the spinal cord, synapse on the inhibitory interneurons, resulting in decreased activation within the motor neuron pool of the effector muscles. This results in decreased recruitment and rate coding of motor units, decreasing the force of contraction of the involved muscles (Hopkins and Ingersoll, 2000; Rice et al., 2014). This is commonly known as arthrogenic muscle inhibition (AMI) (Hopkins and Ingersoll, 2000; Rice et al., 2014).

Patients who suffer from CAIS experience AMI typically of the peroneal and soleus muscles resulting in reduced force output. This decreases stability of the ankle joint and increases the risk of re-injury (McVey et al., 2005; Sefton et al., 2008; Palmieri-Smith et al., 2009). Traditionally patients with CAIS will be treated with muscle strengthening and proprioceptive retraining (Caufield, 2000; Osborne and Rizzo, 2003; Ajis and Maffulli, 2006; McBride and Ramamurthy, 2006; Lee and Lin, 2008; Holmes and Delahunt, 2009; Chinn and Hertel, 2010; Verhagen and Bay, 2010).

This traditional approach to treating CAIS has been challenged by the literature on ankle joint manipulation. There is a body of research that demonstrates that ankle
joint manipulation is clinically beneficial in the treatment of CAIS (Pellow and Brantingham, 2001; Kohne et al., 2007; Lopez-Rodriguez et al., 2007; Whitman et al., 2009; Joseph et al., 2010; Brantingham et al., 2012; Loudon et al., 2013; Lubbe et al., 2015). Lubbe et al. (2015) demonstrated that the combined effect of ankle joint manipulation and rehabilitation resulted in a statistically significant improvement in pain ($p < 0.002$); pain pressure threshold ($p < 0.002$) and motion palpation findings ($p < 0.001$) in participants with CAIS when compared to rehabilitation alone ($N = 30$). This study showed a synergistic relationship between foot and ankle rehabilitation and manipulation, indicating that the manipulation complimented the rehabilitation in such a way as to improve the treatment of CAIS.

This supports Hopkins and Ingersoll (2000) and McVey et al. (2005) by demonstrating that the affected musculature may only return to optimum functioning once the afferent input has been corrected. These studies assessed clinical outcomes and did not investigate the physiological effect of the manipulation on muscle activity parameters. The neurophysiological mechanisms supporting the clinical benefits of joint manipulation are under-investigated (Evans, 2002; Fryer et al., 2002; Pickar, 2002; Maigne and Vautravers, 2003; Andersen et al., 2003; Bialosky et al., 2009; Brantingham et al., 2009; Ritter, 2014), especially in extremity joints.

When assessing the effect of extremity joint manipulation on AMI, Grindstaff et al. (2011) found that distal tibiofibular joint manipulation had a statistically significant ($p < 0.050$) increase in soleus muscle activity in participants with CAIS ($N = 43$). This study used surface electromyography (sEMG) and assessed changes in maximum H-reflex and M-wave measurements (H/M ratio) pre- and post-manipulation. No effect was observed in the peroneal muscle following distal tibiofibular joint manipulation. This suggests that the afferent input as a result of manipulation of the distal tibiofibular joint may not have an effect on the peroneal muscle and therefore cannot aid in correcting the AMI associated with CAIS, leaving the ankle susceptible to re-injury.

It was recommended that further studies investigate the effect of talocrural joint manipulation in patients with CAIS. Therefore, this study aimed to determine the
effects of talocrural joint manipulation on peroneal and soleus muscle activity in CAIS.

1.2 Study aims, objectives and hypotheses

1.2.1 The aim of the study

The purpose of this study was to compare the effect of talocrural joint manipulation compared to a sham talocrural joint manipulation and a control (no intervention) group on peroneal and soleus muscle activity, to determine maximum H-reflex and M-wave ratio (H/M ratio) in CAIS.

1.2.2 Study objectives

1. To determine the effect of talocrural joint manipulation on peroneal and soleus muscle activity in terms of H/M ratio, in CAIS.

2. To determine the effect of a sham talocrural joint manipulation on peroneal and soleus muscle activity in terms of H/M ratio, in CAIS.

3. To compare the effect of talocrural joint manipulation, sham talocrural joint manipulation and a control on the peroneal and soleus muscle activity in terms of H/M ratio, in CAIS.

1.2.3 Hypotheses

1.2.3.1 Null hypothesis:

The null hypothesis states: The group receiving talocrural joint manipulation will have no statistically significant ($p < 0.050$) improvement in peroneal and soleus muscle activity in terms of H/M ratio, when compared to the sham intervention or the control group, in participants with CAIS.

1.2.3.2 Alternate hypothesis:

The alternate hypothesis states: The group receiving talocrural joint manipulation will show a statistically significant ($p < 0.050$) improvement in peroneal and soleus muscle activity in terms of H/M ratio, when compared to the sham intervention or the control group, in participants with CAIS.
1.3 Flow of dissertation

Chapter one has provided an introduction and rational for the study, together with the aims, objectives and study hypotheses.

Chapter two, the literature review will provide an overview of the anatomy of the ankle joint and the diagnosis and management of chronic ankle instability. This will be followed by a critical analysis of the literature on joint manipulation.

Chapter three gives an explanation of the methodology utilised in this study in order to achieve the aims and objectives. The study design, methods, techniques and instruments are outlined and explained.

Chapter four presents the results of the study. The demographic and anthropometric characteristics of the sample together with the surface electromyography data will be presented using figures and tables.

Chapter five provides the discussion of the results in relation to the current literature.

Chapter six will conclude the study discussing the study limitations and recommendations.
Chapter Two

2.1 Introduction

Chronic ankle instability syndrome (CAIS) is a debilitating condition with a negative impact on daily living (Waterman et al., 2010). It is associated with arthrogenic muscle inhibition (AMI) and resultant weakness of the soleus and peroneal muscles making the ankle susceptible to re-injury (McVey et al., 2005; Palmieri-Smith et al., 2009). Therefore an effective treatment strategy is necessary to prevent long term disability.

This chapter presents an overview of the anatomy and biomechanics of the ankle joint and surrounding structures. This will be followed by the relevant literature related to CAIS, AMI and review of the literature related to the effect of joint manipulation.

The following sources were searched for information relevant to the study: Google Scholar, Summon, Ebscohost, PubMed, Medline, MedNets, OmniMedicalSearch, eMedicine, RefSeek and the Durban University of Technology Institutional Repository.


2.2 Overview of the anatomy of the ankle joint

2.2.1 Bones and ligaments of the ankle joint

The ankle joint, also known as the talocrural or mortise joint, consists of the articulation of three bones, namely; the distal end of the tibia and fibula and the proximal part of the talus. This articular structure is supported by strong medial and
lateral ligaments as seen in Figure 2.1 (Tortora and Derrickson, 2009; Moore et al., 2010; McKinley and O'Loughlin, 2012).

**Figure 2.1:** Lateral (Left) and medial (Right) aspect of ankle joint (McKinley and O'Loughlin, 2012).

### 2.2.2 Movement of the ankle joint

#### 2.2.2.1 Ankle joint range of motion

The ankle joint moves in four ranges of movement, as described in Table 2.1, due to muscular action. The medial and lateral ligaments aid in support of the joint during the four ranges of motion.

**Table 2.1:** Ankle joint ranges of movement (Quinn, 2014).

<table>
<thead>
<tr>
<th>Movement</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inversion</td>
<td>0°-35°</td>
</tr>
<tr>
<td>Eversion</td>
<td>0°-25°</td>
</tr>
<tr>
<td>Dorsiflexion</td>
<td>0°-20°</td>
</tr>
<tr>
<td>Plantarflexion</td>
<td>0°-50°</td>
</tr>
</tbody>
</table>

#### 2.2.2.2 Muscles of the ankle joint

Muscles and ligaments are responsible for movement and stabilisation of a joint (McKinley and O'Loughlin, 2012) they also contain mechanoreceptors which aid in proprioception (Snell, 2010). Table 2.2 describing the soleus and peroneal muscles, with Appendix A describe the rest of the muscles involved in movement of the ankle.
In CAIS these structures may be damaged and contribute towards the pathomechanics of the disorder (Bonnel et al., 2010).

**Table 2.2:** The soleus and peroneal muscles (Moore et al., 2010; Vizniak, 2010; McKinley and O’Loughlin, 2012).

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Origin</th>
<th>Insertion</th>
<th>Innervation</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peroneus tertius</td>
<td>Inferior third of anterior surface of fibula and interosseous membrane</td>
<td>Dorsum of the fifth metatarsal base</td>
<td>Deep fibular nerve (L4, L5)</td>
<td>Dorsiflexes ankle and aids in eversion</td>
</tr>
<tr>
<td>Peroneus longus</td>
<td>Head and superior two thirds of the lateral surface of the fibula</td>
<td>First metatarsal base and medial cuneiform</td>
<td>Superficial fibular nerve (L5, S1, S2)</td>
<td>Everts foot and weakly plantarflexes ankle</td>
</tr>
<tr>
<td>Peroneus brevis</td>
<td>Inferior two thirds of lateral surface of fibula</td>
<td>Tuberosity on dorsal, lateral side of fifth metatarsal base</td>
<td>Superficial fibular nerve (L5, S1, S2)</td>
<td>Everts foot and weakly plantarflexes ankle</td>
</tr>
<tr>
<td>Soleus</td>
<td>Posterior aspect of head of fibula and superior quarter of posterior surface of fibula; soleal line and middle third of medial border of tibia; and tendinous arch between the bony attachments</td>
<td>Posterior surface of calcaneus via calcaneal tendon</td>
<td>Tibial nerve (S1, S2)</td>
<td>Plantarflexes ankle independent of position of knee and steadies leg on foot</td>
</tr>
</tbody>
</table>

In order for a muscle to contract the motor units that make up the muscle need to be intact. A motor unit consists of a motor neuron and all the muscle fibres it innervates, in this way skeletal muscles can be innervated by few or many nerves depending on the muscle’s function (Tortora and Derrickson, 2009; Snell, 2010; McKinley and O’Loughlin, 2012). There are two types of motor neurons involved in skeletal muscle innervation:

1. Large, alpha, myelinated fibres which supply the extrafusal muscle fibres.
2. Small, gamma, myelinated fibres which supply the intrafusal muscle fibres.

Motor neurons terminate at the skeletal muscle fibre in the motor end plates which form the neuromuscular junction. The neurotransmitter, acetylcholine, is released
from the presynaptic membrane of the motor endplates and diffuses across the synaptic cleft. It is then picked up by the receptors on the postsynaptic membrane resulting in depolarisation of the sacrolemma resulting in the release of calcium (Ca^{++}) ions and skeletal muscle contraction (Tortora and Derrickson, 2009; Snell, 2010; McKinley and O’Loughlin, 2012).

2.2.3 Innervation of the ankle joint

The spinal nerves of the lumbar and sacral regions form the lumbosacral plexus, this gives rise to the nerves supplying the pelvic region and lower limb. The sciatic nerve originates from the spinal nerves of L4-S3 (McKinley and O’Loughlin, 2012). The talocrural joint is innervated by the tibial and deep peroneal nerves. These nerves form from the bifurcation of the sciatic nerve just proximal to the popliteal fossa. The common peroneal nerve bifurcates into the superficial and deep fibular nerves (Tortora and Derrickson, 2009; Moore et al., 2010). These same nerves supply the soleus and peroneal muscles. Hilton’s law states that a joint is innervated by the same nerves that innervate the muscles that act on the joint and the skin surrounding the joint (Herbert-Blouin et al., 2013).

A spinal nerve, as illustrated in figure 2.2, is formed in the intervertebral foramen (IVF) and comprises of both afferent and efferent nerves (known as a mixed nerve) and results from the joining of the anterior and posterior roots that arise from the anterior and posterior horns of the spinal cord. Afferent nerve fibres relay sensory information from sensory receptors in the muscles, skin and ligaments, to the posterior horn of the spinal cord via the posterior root which contains the afferent nerve fibres, their cell bodies are located within the posterior root ganglion (Tortora and Derrickson, 2009).

Efferent nerve fibres relay motor information from the anterior horn of the spinal cord to the relevant muscles/effecter organs. The efferent nerve fibres’ cell bodies are located within the anterior horn of the spinal cord (McKinley and O’Loughlin, 2012).

Once the spinal nerve exits the IVF it bifurcates into the anterior and posterior rami, both of which are mixed nerves. The posterior ramus supplies the skin and muscles of the back while the anterior ramus supplies the ipsilateral trunk and limb (Tortora and Derrickson, 2009; Moore et al., 2010; Kieran, 2014).
2.2.3.1 Proprioception of the ankle

Proprioceptors are sensory receptors (mechanoreceptors) that provide information regarding body position, muscle length and tension, joint position, movement and equilibrium. They are located within muscles, tendons, joints and the surrounding tissues (Tortora and Derrickson, 2009; Snell, 2010; McKinley and O’Loughlin, 2012; Dougherty, 2014). There are several different types of proprioceptors:

1) Muscle spindles

Muscle spindles are proprioceptors located within skeletal muscles and measure a muscle’s length (stretch). Muscle spindles consist of intrafusal muscle fibres, which are specialized sensory nerve endings, surrounded by normal skeletal muscle fibres called extrafusal muscle fibres. Stretching of the intrafusal muscle fibre results in

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Figure 2.2: Spinal nerve anatomy (McKinley and O’Loughlin, 2012).
stimulation of the sensory nerve endings. This information is then relayed to the spinal cord via Type Ia and Type II afferent neurons and synapse with alpha and gamma motor neurons in the anterior horn (Tortora and Derrickson, 2009; Snell, 2010).

Gamma motor neurons adjust the tension of the muscle spindle according to muscle length variations. Extrafusal muscle fibres are innervated by alpha motor neurons and when stimulated result in contraction of the extrafusal muscle fibres. Therefore both the alpha and gamma motor neurons participate in the stretch reflex (Snell, 2010; McKinley and O’Loughlin, 2012).

2) Golgi tendon organs

Golgi tendon organs (GTO) are proprioceptors located at musculotendinous junctions and provide information regarding the tension of the muscle (the force exerted by a contracting muscle). An increase in muscle tension results in stimulation of the GTO, the information is relayed to the spinal cord via Type Ib afferent fibres where they synapse with alpha motor neurons in the anterior horn. A negative feedback response follows, inhibiting further muscle contraction. This is known as the tendon reflex and results in a decreased muscle tension, this protects the muscle from over-contracting. The relationship between the muscle spindle and GTO is responsible for the tone of a muscle (Dougherty, 2014).

3) Joint kinesthetics receptors

These receptors, also known as Wyke receptor, are located within a joint’s connective tissue, capsule and surrounding ligaments (Dougherty, 2014; Snell, 2010). There are four different types:

- Small pacinian corpuscles are located within a joint’s connective tissue and capsule and are innervated by Type II afferent fibres. They sense joint movement, particularly direction and velocity.
- Ruffini corpuscles are mainly located within a joint’s capsule and sense static joint position as well as joint movement, direction and speed. They are innervated by Type II afferent fibres.
- Golgi type endings are located within the ligaments surrounding a joint; they sense joint torque and are active at a joint’s end range. They are innervated by Type II afferent fibres.
- Free nerve endings are located within a joint’s capsule, connective tissue, ligaments and musculature. They sense extreme mechanical and chemical irritation and are innervated by Type III afferent fibres.

2.2.3.2 Spinal cord tracts

Afferent neurons originating from joints have projections in both the spinal cord and the supraspinal centres. Therefore supraspinal changes as a result of afferent neuron interruption are likely to occur. Descending spinal pathways have projections to interneurons and motor neurons within the spinal cord thereby having an influence on AMI (Rice and McNair, 2010).

AMI is associated with decreased motor neuron pool excitability but increased corticomotor excitability. It is speculated that the increased corticomotor excitability allows the central nervous system to increase the corticospinal activity to the muscle in an attempt to counteract the decreased motor neuron pool excitability as a result of spinal reflex pathways (Rice and McNair, 2010; Rice et al., 2014).

Rice et al., (2014) assessed the effects of experimentally induced knee joint effusion on AMI motor cortex excitability. They concluded that corticomotor excitability increased post knee joint effusion; however there was no cortical contribution to quadriceps AMI. This suggests that continued spinal reflex inhibition post joint injury is sufficient in explaining the decreased motor neuron pool excitability associated with AMI.

Spinal tracts are bundles of nerve fibres located within the white matter of the spinal cord. Spinal tracts can be divided into ascending, descending and intersegmental tracts (Kieran, 2014).

The ascending tracts are comprised of bundles of ascending neurons that enter the spinal cord. These tracts can link different areas of the spinal cord or link the spinal cord to the brain. Conscious afferent information ascends to the cerebral cortex while unconscious afferent information ascends to the cerebellum. Information originating
from the muscle spindles, GTO and joint receptors ascend via the spinocerebellar tracts (Snell, 2010; McKinley and O’Loughlin, 2012; Kieran, 2014).

The descending tracts are comprised of bundles of efferent neurons originating from the medulla, pons, midbrain and cerebral cortex (Snell, 2010). Only the tracts relevant to this study will be discussed below.

- Corticospinal tracts:
  Efferent neurons originating from the pre central gyrus and post central gyrus enter the spinal cord and descend within the lateral white column. Most corticospinal tract fibres supply alpha motor neurons and gamma motor neurons. This pathway is responsible for performing rapid skilled movements (Snell, 2010; McKinley and O’Loughlin, 2012).

- Reticulospinal tracts:
  Efferent fibres originating from the reticular formation within the brainstem enter the spinal cord and descend within the anterior and lateral white columns. These fibres enter the anterior gray horn of the spinal cord and facilitate or inhibit alpha and gamma motor neurons. Therefore, the reticulospinal tracts are responsible for the control of voluntary movements and reflex activity (Snell, 2010; McKinley and O’Loughlin, 2012).

2.3 Chronic ankle instability syndrome

Ankle sprains are amongst the most common injuries in athletes as well as the general public (Ferran and Maffulli, 2006; Waterman et al., 2010). It is estimated that worldwide there is approximately one acute ankle sprain per 10 000 people per day (Waterman et al., 2010). According to Anandacoomarasamy and Barnsley (2005), 74% of patients who have suffered an acute ankle sprain have at least one persisting symptom one and half to four years after the injury; these symptoms included pain, instability, swelling or weakness.

There are a number of conditions that may mimic CAIS that need to be explored and ruled out before a clinical diagnosis can be made. The following conditions are possible differential diagnoses for CAIS: fractures of the foot and ankle, peroneal tendonitis, osteoarthritis of the ankle joint, Achilles tendon injuries, ankle impingement syndrome, lateral malleolar bursitis, tarsal coalition, posterior tibialis
tendon dysfunction, sinus tarsi syndrome and osteochondritis dessicans (de Bie, et al, 2003; Chan et al., 2011).

Chronic ankle instability syndrome is defined as the constant tendency of the ankle to re-spray following an acute ankle sprain (Caufield, 2000). Clinical features of CAIS include: recurrent ankle sprains (two or more), the feeling of the ankle “giving way”, lateral ankle pain, crepitus, oedema, weakness, adhesions, joint restrictions, hypermobility or hypomobility (Caufield, 2000; Pellow and Brintingham, 2001; Ajis and Maffulli, 2006).

According to the International Ankle Consortium a diagnosis of CAIS (Gribble et al., 2013), requires the following minimal criteria:

1) A history of at least one ankle sprain.

2) A history of the previously injured ankle joint “giving way”.

3) A general self-reported foot and ankle function questionnaire, this is only applicable if the level of self-reported function is important to the research. On the Foot and Ankle Ability Measure questionnaire, a score of <90% for activities of daily living subscale and <80% for the sport subscale. On the Foot and Ankle Outcome Score, a score of <75% in three or more categories is sufficient.

Chronic ankle instability syndrome can be a costly condition economically due to absence from work, socially from an inability to participate in sport, and an increased dependence on a therapist to resume activities of daily living (Ferran and Maffulli, 2006). The high incidence of ankle sprains coupled with the high incidence of residual symptoms suggests that a more effective treatment strategy is required (Anandacoomarasamy and Barnsley, 2005).

2.3.1 Mechanism of injury

The most common mechanism of an ankle sprain involves excessive inversion and supination of the foot and ankle complex. Up to 85% of ankle sprains occur in this way and are called inversion ankle sprains (Ferran and Maffulli, 2006). The excessive stress placed on the lateral ligaments and joint capsule results in disruption of all or some of these structures, depending on the severity of the injury,
together with pain, swelling and joint dysfunction (Denegar and Miller, 2002; Hertel, 2002; Bonnel et al., 2010). To diagnose CAIS the grade of the ankle sprain needs to be determined. CAIS is graded according to the grade of the initial or most recent acute ankle sprain (Pourkazemi et al., 2014).

2.3.1.1 Grading methods for ankle sprains

There are three grading systems, one based on clinical features and two based on ligament integrity (Lynch, 2002). Ligament injuries are normally graded according to the extent of damage to a single ligament, as seen in table 2.3. The criticism of this grading system is that it focuses on a single ligament, when often more than one ligament is involved (Caulfield, 2000; Pellow and Brantingham, 2001; Lynch, 2002).

Table 2.3: Grading method according to the extent of damage to a single ligament (Caulfield, 2000; Pellow and Brantingham, 2001; Lynch, 2002).

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Microscopic damage without any macroscopic damage.</td>
</tr>
<tr>
<td>II</td>
<td>Macroscopic stretching/damage while the ligament remains intact.</td>
</tr>
<tr>
<td>III</td>
<td>Complete tear of the ligament.</td>
</tr>
</tbody>
</table>

This resulted in a grading system being developed based on the number of ligaments involved in a lateral ankle sprain, as detailed in table 2.4. In a clinical setting the drawback to these two grading methods is that the patient is required to undergo objective investigations, such as ultrasound, magnetic resonance imaging (MRI) or arthroscopy to determine the grade (Lynch, 2002).

Table 2.4: Grading method according to the number of ligaments involved (Chan et al., 2011).

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Stretching of the ATFL.</td>
</tr>
<tr>
<td>II</td>
<td>Tearing of the ATFL with/without tearing of the CFL.</td>
</tr>
<tr>
<td>III</td>
<td>Tearing of the ATFL and CFL with a capsular tear or tear of the PTFL.</td>
</tr>
</tbody>
</table>
In response to these drawbacks a grading system was developed based on clinical features, as described in table 2.5. This grading system has been shown to be superior in a clinical setting where objective investigations are not necessary.

**Table 2.5:** Grading method according to the clinical features (Reid, 1992; Caulfield, 2000; Pellow and Brantingham, 2001; Lynch, 2002; Ajis and Maffulli, 2006; Chan et al., 2011).

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Mild sprain, mild ligament damage, no haemorrhage or bruising, minimal oedema, point tenderness and no gross instability.</td>
</tr>
<tr>
<td>II</td>
<td>Moderate sprain, partial tearing of the ligaments, minimal haemorrhage and bruising, localised oedema and minimal instability if at all.</td>
</tr>
<tr>
<td>III</td>
<td>Severe sprain, complete rupture of the ligaments, early haemorrhage and bruising, diffuse oedema on both sides of the Achilles tendon, tenderness laterally and possibly medially, and gross instability.</td>
</tr>
</tbody>
</table>

The insufficiencies that may contribute towards the development of CAIS stem directly from the initial effects of the ankle sprain (such as trauma, pain, swelling and joint dysfunction). These insufficiencies can be classified into mechanical and functional and may act alone or together (Hertel 2002; Bonnel et al., 2010).

2.3.1.2 Mechanical insufficiencies leading to CAIS

These are anatomical abnormalities of the bone, ligaments and joints and may be congenital or as a result of injury. Mechanical instability does not respond well to conservative treatment (Bonnel et al., 2010).

1) Pathological laxity
   Disruption and poor healing of the ligaments supporting the ankle following ankle sprain may result in mechanical instability; this is dependent on the extent of the disruption. Instability of the ankle is best demonstrated when placed into vulnerable positions (inversion, supination and plantarflexion) during activities (Hertel, 2002; Bonnel et al., 2010).

2) Arthrokinematic impairments
Disruption of the normal arthrokinematics of any of the joints that make up the ankle joint complex, as a result of joint dysfunction or bony changes, can result in mechanical instability. Hypomobility of the joint, in particular decreased dorsiflexion of the talocrural joint, has been shown to contribute towards CAIS (Denegar et al., 2002; Hertel, 2002; Bonnel et al., 2010).

3) Synovial and degenerative changes
Synovial swelling as a result of inflammation as well as degenerative changes within the ankle joint complex have been shown to result in mechanical instability and may contribute towards CAIS (Hertel, 2002; Bonnel et al., 2010).

2.3.1.3 Functional insufficiencies leading to CAIS
These are postural defects and abnormalities of the muscles and tendons. These defects are commonly a result of injury and respond well to conservative treatment (Hertel, 2002; Bonnel et al., 2010). Functional insufficiencies are often inter-related and may occur in isolation or together (Hiller et al., 2011).

1) Impaired proprioception
Evidence shows that individuals who experience repetitive ankle sprains have impaired proprioceptive sensation. The most likely cause of this impairment is disruption of the mechanoreceptors found within the articular surfaces, ligaments and surrounding musculature of the ankle (Lentell et al., 1995; Konradsen, 2002; Riemann, 2002).

2) Impaired neuromuscular firing
Impaired peroneal muscle response has been found in patients suffering from CAIS. Deficits in peroneal muscle response may be as a result of proprioceptive deficits, decreased nerve conduction velocity or impairments in neuromuscular recruitment (Hertel, 2002; Vaes et al., 2002; Hopkins et al., 2009).

3) Impaired postural control
Postural instability, particularly during single leg standing, has been found in patients with CAIS (Delahunt et al., 2006; Bonnel et al., 2010). Postural control impairments may be attributed to deficits in proprioception and neuromuscular control (Hertel, 2002).

4) Strength deficits and muscle imbalances
A correlation has been made between strength deficits of the peroneal muscles and an increased incidence of repeated ankle sprains however, this correlation can vary between patients (Hertel, 2002; Kaminski and Hartsell, 2002). The exact cause of the strength deficit is unclear; possibilities include muscle damage, atrophy and/or impaired neuromuscular recruitment (Hertel, 2002). A greater link has been found between muscle imbalances and CAIS than strength deficits and CAIS (Kaminski and Hartsell, 2002). The main muscle imbalances found in patients with CAIS are weak ankle evertor muscles and strong or normal invertor muscles (Vizniak and Carnes, 2004).

2.3.2 Arthrogenic muscle inhibition
Arthrogenic muscle inhibition (AMI) is a presynaptic, reflex inhibition of the muscles surrounding a damaged joint even though the muscles themselves are not necessarily damaged. AMI is a natural response to joint injury; its function is to protect the joint from further injury (Hopkins and Ingersoll, 2000) and it is present in any condition that involves arthritis, swelling or injury (including surgery) to a joint. Afferent neurons relay the information from the disrupted mechanoreceptors to the spinal cord where it acts on inhibitory interneurons resulting in decreased recruitment of motor neurons within the motor neuron pool of the related muscles. The decreased motor neuron pool excitability results in a decreased force of contraction of the involved muscles (Hopkins and Ingersoll, 2000; McVey et al., 2005; Rice et al., 2014) otherwise known as AMI.

Arthrogenic muscle inhibition takes a central role in the injury cycle as seen in Figure 2.3. Following joint injury, an individual experiences immobilization or a decreased joint range of motion (ROM) (Hopkins and Ingersoll, 2000; Rice et al., 2014). Decreased (ROM) as a result of swelling, pain, muscle spasm or the inability of the involved muscles to contract maximally (muscle inhibition). This results in muscle
atrophy and weakness thereby increasing the risk of re-injury (Hopkins and Ingersoll, 2000; Rice et al., 2014).

![Diagram of the injury cycle](Image)

**Figure 2.3:** The injury cycle (Hopkins and Ingersoll, 2000)

The neurophysiological response of disrupted joint mechanoreceptors present in AMI plays a role in limiting joint rehabilitation. Strength training and active exercise are necessary in joint rehabilitation; however, AMI decreases the ability of the individual to achieve functional, symmetric, bilateral muscle strength i.e. returning the involved muscles to optimal functioning (Hopkins and Ingersoll, 2000; Rice et al., 2014).

Arthrogenic muscle inhibition of the ankle joint is considered a contributing factor in the development of CAIS. The inhibition of the peroneal and soleus muscles results in a decreased ability of these muscles to exert force and sufficiently stabilise the ankle. This in turn makes the ankle more likely to be re-injured (McVety et al., 2005; Sefton et al., 2008; Palmieri-Smith et al., 2009; Klykken et al., 2011). According to Hopkins and Ingersoll (2000) and McVey et al. (2005) the disruption of afferent input to the nervous systems needs to be corrected in order for the muscles to function optimally. It is theorised that the sudden influx of afferent information as a result of joint manipulation may aid in correcting this and resulting in increased motor neuron pool excitability of the affected muscles (Grindstaff et al., 2011; Maduro de Camargo et al., 2011; Niazi et al., 2015).
2.3.3 Treatment of CAIS

The high incidence of ankle sprains and the development of CAIS demonstrate the need for an adequate and effective treatment protocol (Anandacoomarasamy and Barnsley, 2005; Doherty et al., 2013). Treatment protocols concentrate on reducing residual pain and swelling, improving range of motion, neuromuscular control, proprioception as well as strengthening the effected musculature to prevent re-injury (Denegar et al., 2002; Denegar and Miller, 2002; Hertel, 2002; Kaminski and Hartsell, 2002; Konradsen, 2002; Bonnel et al., 2010). There are a large number of treatment modalities that can be utilised in the treatment of CAIS, they are described below with joint manipulation following in the next section.

2.3.3.1. Bracing and taping

Bracing and taping techniques are recommended as prophylactic measures rather than a treatment option. Delahunt et al. (2010) found that ankle taping does not significantly improve dynamic postural stability in CAIS; however it was noted that ankle taping did result in an increased subjective perception of confidence, stability and reassurance. It is important to note that bracing will not improve muscle strength or proprioception and may result in weakening of the muscles if worn for prolonged periods (Eils et al., 2002; Papadopoulos et al., 2005).

2.3.3.2. Non-steroidal anti-inflammatory drugs

Non-steroidal anti-inflammatory drugs (NSAID’s) have been widely used for their analgesic and anti-inflammatory effects. NSAID’s are indicated in the majority of musculoskeletal conditions, benefits include reduction in pain, swelling and a decreased return to activity time (Ziltener et al., 2010; Bruno et al., 2014). Adverse effects of NSAID’s are related to the gastrointestinal tract, the cardiovascular system, the renal system and the liver. Adverse effects are a result of frequent prolonged use therefore prescriptions should be kept to the minimal dosage for the shortest period of time (Ong et al., 2007; Bruno et al., 2014).

2.3.3.3. Ultrasound therapy

The use of ultrasound therapy for the treatment of musculoskeletal conditions is a controversial topic. The benefits of ultrasound therapy include: pain relief, decreased swelling and collagen mobilisation (Turner and Merriman, 2005). There is limited
evidence demonstrating that therapeutic ultrasound is effective in treating musculoskeletal conditions of the lower limb (Shanks et al., 2010). Zammit and Herrington (2005) found no statistically significant benefit for ultrasound compared to a placebo in the treatment of CAIS (N = 34).

2.3.3.4. Surgery
Surgical treatment for CAIS is reserved for cases when there is marked mechanical instability that corresponds to a grade III ankle sprains as described above (Chan et al., 2011; de Vries et al., 2011).

2.3.3.5. Rehabilitation
Rehabilitation has an important and integrative role in the treatment of CAIS (Chan et al., 2011; de Vries et al., 2011). Rehabilitation includes muscle strengthening, proprioception and balance training as well as regaining neuromuscular control (Caufield, 2000; Ajis and Maffulli, 2006; McBride and Ramamurthy, 2006; Lee and Lin, 2008). Functional rehabilitation with early mobilisation has proven to be the most effective method (de Vries et al., 2011). According to Webster and Gribble (2010) a rehabilitation program of four to six weeks with exercises performed three to five times a week will improve dynamic measures of postural control and self-reported outcomes.

2.4 Joint manipulation
Manipulation is defined as a manual treatment technique whereby a high velocity, low amplitude thrust is applied to a synovial or cartilaginous joint. Manipulation results in a biomechanical change to the joint within the boundaries of anatomical integrity. Manipulation is often accompanied by a crack or popping sound known as a cavitation however, the cavitation is not necessary for the manipulation to be successful (Pickar, 2002; Maigne and Vautravers, 2003; Kaur et al., 2014; Cardinale et al., 2015).

Manipulation is most commonly applied to the spine, however it can be used on any synovial joint (Pickar, 2002). The biomechanical benefits of manipulation can be attributed to releasing trapped meniscoids, breaking down adhesions and restoring
normal joint position, resulting in optimum joint mobility and joint play (Maigne and Vautravers, 2003; Kaur et al., 2014).

It is theorised that the biomechanical changes to the structures of the joint during manipulation results in a stimulation of the mechanoreceptors within the joints and surrounding tissues (Pickar, 2002; Haavik and Murphy, 2012; Pickar and Bolton, 2012; Kaur et al., 2014; Cardinale et al., 2015). This information is then relayed along type I and type II afferent fibres to the dorsal horn of the spinal cord. The afferent neuron synapses with the interneuron which relays an excitatory or inhibitory effect to the motor neuron; this information is then relayed to the appropriate muscles resulting in an increase or decrease of motor neuron pool excitability (Suter and McMorland, 2002; Dunning and Rushton, 2009; Haavik and Murphy, 2012; Pickar and Bolton, 2012; Cardinale et al., 2015).

Ankle joint manipulation has shown to be clinically beneficial in the treatment of CAIS (Pellow and Brantingham, 2001; Kohne et al., 2007; Lopez-Rodriguez et al., 2007; Whitman et al., 2009; Joseph et al., 2010; Brantingham et al., 2012; Loudon et al., 2013; Lubbe et al., 2015). Manipulation increases range of motion by reducing restrictions, improves proprioception and muscle functioning and may decrease the subjective level of pain (Lindsey-Renton, 2005; Whitman et al., 2009; Grindstaff et al., 2011; Loudon et al., 2013; Lubbe et al., 2015).

Loudon et al. (2013) performed a systematic review of eight articles pertaining to the treatment of lateral ankle sprains by manual joint techniques; five of the eight articles fell under the heading subacute/chronic ankle sprains. The review concluded that manual joint techniques aid in the treatment of subacute/chronic ankle sprains by restoring range of motion (ROM) (especially dorsiflexion), pain reduction as well as increasing foot and ankle function. The neurophysiological mechanisms of these clinical effects remain unclear and under investigated (Courtney et al., 2010; Grindtsaff et al., 2011; Grindstaff et al., 2014). Table 2.6 presents studies investigating the clinical effect of ankle joint manipulation on CAIS.
Table 2.6: Clinical studies investigating Talocrural joint manipulation in the treatment of CAIS.

<table>
<thead>
<tr>
<th>Author</th>
<th>Sample Size</th>
<th>Study Design</th>
<th>Intervention</th>
<th>Outcome Measures</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joseph et al., 2010.</td>
<td>N=40,</td>
<td>RCT</td>
<td>1: T/C HVLA JM. 2: T/C mobilisation</td>
<td>One leg standing test, NRS.</td>
<td>Both groups significant improvement in balance, ROM and function and pain.</td>
</tr>
<tr>
<td>Kohne et al., 2007.</td>
<td>N=30</td>
<td>RCT</td>
<td>1: Single T/C JM. 2: Six T/C joint manipulations</td>
<td>Proprioception, ROM and point tenderness.</td>
<td>Significant increase in proprioception and dorsiflexion ROM in group two. Both groups improved however; group two demonstrated a greater improvement.</td>
</tr>
</tbody>
</table>

(RCT = randomised controlled clinical trial, T/C = Talocrural, NRS = numerical pain rating scale, JM = joint manipulation, HVLA = high velocity low amplitude, VAS = visual analogue scale, FADI = foot and ankle disability index, WB = weight bearing, BBS = berg balance scale)

These studies all support the use of ankle joint manipulation in the management of either subacute or chronic grade I and grade II ankle sprains or CAIS. The clinical benefits range from improved ROM, function, pain pressure threshold and balance, with greater improvements occurring after more than one treatment.
Lubbe et al. (2015) demonstrated that the combined effect of ankle joint manipulation and rehabilitation resulted in significant improvements in pain (visual analogue scale: \( p < 0.002 \)); pain pressure threshold (\( p < 0.002 \)) and motion palpation findings (\( p < 0.001 \)) in CAIS when compared to rehabilitation alone (\( N = 30 \)). This study showed a synergistic relationship between foot and ankle rehabilitation and manipulation, indicating that the manipulation complemented the rehabilitation in such a way as to improve the treatment of CAIS. This supports Hopkins and Ingersoll (2000) and McVey et al. (2005) by demonstrating that the affected musculature may only return to optimum functioning once the afferent input has been corrected.

2.4.1 Measuring the neurophysiological effects of manipulation with electromyography

Electromyography (EMG) is an electrical method of measuring muscle excitation and activation, particularly surface electromyography (sEMG) which is a convenient, accurate and non-invasive method of measuring muscle activity and muscle patterns (Sousa and Tavares, 2012). sEMG has often been used in assessing muscle activity in patients suffering from ankle sprains (Beckman et al., 1995; Cordova and Ingersoll, 2003; Grindstaff et al., 2011; Klykken et al., 2011). There are a number of studies where sEMG has been used to assess changes in muscle activity before and after spinal or extremity manipulation (Colloca and Keller, 2000; Suter and McMorland, 2002; Noska, 2006; Dunning and Rushton, 2009; Murray, 2009; Grindstaff et al., 2011; Niazi et al., 2015).

2.4.1.1 H-reflex and M-wave

The Hoffmann Reflex (H-reflex) is one of the most widely researched reflexes in neurophysiological literature (Misiaszek, 2003; Knikou, 2008). The H-reflex is an electrically induced reflex equivalent to a mechanically induced spinal stretch reflex, the only difference being that the H-reflex bypasses the muscle spindles (direct stimulation of the Ia afferents) (Palmieri et al., 2004; Knikou, 2008). The H-reflex measures the motor neuron pool excitability (MNPE) of the involved muscles and can therefore be used to assess various neurological conditions, musculoskeletal injuries and the effect of therapeutic modalities (Palmieri et al., 2004; Knikou, 2008).
At low levels of stimulation the afferent neurons are preferentially stimulated as a result of their larger diameter. The stimulation travels along the afferent neurons to the spinal cord where they synapse with alpha motor neurons, the stimulation is then relayed to the muscle where the reflex contraction is picked up by the sEMG, this reflex contraction is the H-reflex, the peak-to-peak amplitude is recorded. (Palmieri et al., 2004).

Increasing the stimulation intensity past that required to elicit the maximum H-reflex ($H_{\text{max}}$) results in direct stimulation of the alpha motor neurons which are smaller in diameter than the afferent fibres. This stimulation is relayed directly to the muscle and does not pass through the spinal cord; it is therefore not a true reflex and is referred to as the muscular response (M-wave) (Palmieri et al., 2004).

The M-wave is elicited at the same time as the H-reflex only at higher stimulus intensity. As the alpha motor neurons are stimulated they block impulses coming from the Ia afferents via the spinal cord, once the stimulation intensity is great enough to directly stimulate all of the alpha motor neurons it results in a complete block of information from the Ia afferents and absence of the H-reflex, this is known as antidromic collision.

Figure 2.4 summarises this process. The stimulus intensity is increased until the M-wave peak-to-peak amplitude fails to increase further. This is the maximal M-wave ($M_{\text{max}}$) and is an accurate indicator of the muscles full activation (Tucker et al., 2005).
Figure 2.4: Summary of the process of eliciting the H-reflex and M-wave (Palmieri et al., 2004). A. Electrical stimulation only stimulates the Ia afferent fibres, this is relayed, via the spinal cord, to the muscle and presents as the H-reflex curve on the sEMG. B. Slightly greater electrical stimulation stimulates the Ia afferents as well as some alpha motorneurons, this appears as an H-reflex curve (on the descending part) as well as an M-wave curve (on the ascending part). C. Greater electrical stimulation results in stimulation of all of the alpha motor neurons, antidromic collision blocks all impulses from the Ia afferents therefore only the maximum M-wave curve appears on the sEMG.

2.4.1.2 Processing of sEMG signal

The amplitude of the raw sEMG signal is not an accurate method for comparisons between different individuals; the signal is variable and is dependent on a number of factors. In order to compare muscle activity between different individuals and different muscles the sEMG signal needs to be normalized (Sousa and Tavares,
2012). The representation of the H-reflex and M-wave as a ratio is used to normalise the sEMG data. This is done by taking the maximal H-reflex and maximal M-wave and expressing it as an H/M ratio (Palmieri et al., 2004; Tucker et al., 2005). The H/M ratio is an indication of the proportion of the total motor neuron pool that is capable of being recruited. This can be construed as a measure of the muscle’s activity. This is based on the premise that the $M_{\text{max}}$ amplitude is a stable value between pre and post-intervention measurements. It has been recommended that the $M_{\text{max}}$ be reported on and that no differences were detected. This ensures that the change in H/M ratio is a result of change in the $H_{\text{max}}$ as opposed to the $M_{\text{max}}$ (Palmieri et al., 2004).

### 2.4.2 Research into the neurophysiological effects of manipulation

The exact neurophysiological mechanism of spinal manipulation remains unclear, with the effect being either excitatory (Colloca and Keller, 2000; Haavik and Murphy, 2012; Niazi et al., 2015) or inhibitory (Lelanne et al., 2009; Fryer and Pearce, 2012) varying between studies (Pickar and Bolton, 2012). Table 2.7 describes studies that assess the effects of spinal manipulation on muscle activity.

<table>
<thead>
<tr>
<th>Author</th>
<th>Sample size</th>
<th>Study design</th>
<th>Intervention</th>
<th>Outcome measures</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardinale et al., 2015</td>
<td>N=27, asymptomatic</td>
<td>RCT crossover</td>
<td>1: L/S SM 2: Lumbar stretching. 3:Sham</td>
<td>Force fluctuation task, modified Sorensen’s test and sit and reach. PS and gastroc muscles sEMG.</td>
<td>L/S SM did not show a significant improvement superior to the other modalities for force output and sEMG parameters.</td>
</tr>
<tr>
<td>Niazi et al., 2015</td>
<td>N=10, subclinical low back pain.</td>
<td>RCT</td>
<td>1: L/S SM 2: Control</td>
<td>sEMG V-wave, H-reflex, M-wave and max MVC of ankle plantarflexors</td>
<td>Significant increase in motor neuron pool excitability, cortical drive and preventing fatigue.</td>
</tr>
</tbody>
</table>

Table 2.7: The effects of spinal manipulation on muscle activity.
<table>
<thead>
<tr>
<th>Study</th>
<th>Participants</th>
<th>Study Design</th>
<th>Intervention</th>
<th>Outcome Measures</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grindstaff et al., 2014.</td>
<td>N=75, history of knee joint injury and current quadriiceps inhibition.</td>
<td>RCT</td>
<td>1: Lumbopelvic SM 2: SM positioning (no thrust) 3: Grade IV patella mobilization 4: Grade I patella mobilization 5: Control</td>
<td>sEMG H-reflex of quadriceps over time (pre, post 0, 30, 60, 90 min)</td>
<td>No significant differences in H-reflex between groups across time.</td>
</tr>
<tr>
<td>Harvey and Descarreaux, 2013.</td>
<td>N=60, participants with low back pain.</td>
<td>RCT</td>
<td>1: L/S SM 2: Control</td>
<td>sEMG activity of PS muscles, Kinematics, Pain intensity.</td>
<td>No significant differences between the groups.</td>
</tr>
<tr>
<td>Lelanne et al., 2009.</td>
<td>N=27, participants with chronic low back pain.</td>
<td>RCT</td>
<td>1: L/S SM. 2: Control</td>
<td>Trunk and pelvic angles and sEMG activity of PS muscles in trunk flexion-extension.</td>
<td>Significant decrease in sEMG of PS muscles at full flexion following SMT.</td>
</tr>
<tr>
<td>Colloca and Keller, 2000.</td>
<td>N=40, participants with low back pain.</td>
<td>RCT</td>
<td>1: Manually assisted SM 2: Control.</td>
<td>sEMG of PS muscles during trunk extension maximum voluntary contraction.</td>
<td>Significant increase in sEMG activity of PS muscles post manually assisted SM.</td>
</tr>
</tbody>
</table>

(SM= spinal manipulation, L/S= lumbar spine, HVLA = high velocity low amplitude, RCT = randomised controlled trial, PS = paraspinal, Gastroc= gastrocnemius muscle, MVC = maximum voluntary contraction)

Lelanne et al. (2009) demonstrated a significant decrease in paraspinal muscle EMG activity following lumbar SM in participants with chronic low back pain. This result is
supported by Fryer and Pearce (2012) who also demonstrated a decrease in motor neuron activity following lumbar SM. Similarly Harvey and Descarreaux (2013) showed that lumbar SM has an inhibitory effect on the paraspinal muscles, they concluded that lumbar SM reduces fatigue and sensitization of the paraspinal muscles in participants with low back pain. In contrast to these studies Niazi et al., (2015) found that lumbar SM significantly increased the motor neuron excitability and cortical drive of the soleus muscle when compared to control in subclinical low back pain individuals.

Cardinale et al. (2015) conducted a pilot study on the immediate neuromuscular changes in the gastrocnemius and soleus muscles following lumbar SM in asymptomatic individuals, they found that lumbar SM did not result in any statistically significant changes in sEMG parameters when compared to other treatment techniques, the authors concluded that further studies are needed to assess and explain the effects of SM on neuromuscular function. Parallel with the results of Cardinale et al. (2015) Grindstaff et al. (2014) concluded that manual therapies directed at the knee and lumbopelvic region do not significantly affect quadriceps neuromuscular excitability. Grindstaff et al. (2014) compared the effects of manipulation and mobilization of the lumbopelvic joint and patella on quadriceps neuromuscular excitability in participants with previous knee joint injury and current quadriceps inhibition, they found no significant differences in quadriceps H-reflex, presynaptic or postsynaptic excitability between groups across time.

There have been limited investigations on the neurophysiological effects of extremity joint manipulation and its effects on the surrounding musculature in symptomatic or asymptomatic individuals (Courtney et al., 2010; Grindstaff et al., 2011; Grindstaff et al., 2014).

The stimulation of mechanoreceptors within the extremity joints and their surrounding tissues, should have similar neurophysiological responses as those seen in the spine (Haavik and Murphy, 2012; Pickar and Bolton, 2012).

Grindstaff et al. (2014) found no significant influence of patella mobilization on quadriceps neuromuscular excitability in participants with knee pain. This manual technique did not incorporate a thrust which is typical of manipulation. When assessing the effect of extremity manipulation on AMI, Grindstaff et al. (2011) found
that distal tibiofibular joint manipulation had a statistically significant ($p < 0.05$) increase on soleus muscle activity in patients with CAIS ($N = 43$). This study used sEMG and assessed changes in H/M ratio pre- and post-manipulation. No effect was observed in the peroneal muscle following distal tibiofibular joint manipulation. Grindstaff et al. (2011) concluded that distal tibiofibular joint manipulation has an excitatory effect on the soleus muscle with no effect noted on the peroneal muscle. This suggests that the afferent input as a result of manipulation of the distal tibiofibular joint did not have an effect on the peroneal muscles and therefore cannot aid in correcting the AMI associated with the CAIS. Denegar et al. (2002) found that distal tibiofibular joint restrictions as well and talocrural joint restrictions are present in patients with CAIS with talocrural joint restrictions being more prominent. Grindstaff et al. (2011) failed to manipulate the more prominent restriction which may explain the absence of an effect noted in the peroneal muscles. Therefore this study aimed to assess the effect of talocrural joint manipulation on soleus and peroneal muscle activity in CAIS.

2.5 Sham manipulation

The challenge of a sham manipulation procedure is that it needs to fit the participants’ perception of a “real” manipulation while ensuring that the sham manipulation is therapeutically inert. The participants should have no doubt that the intervention they are receiving is an active intervention (Vernon et al., 2005). The sham intervention should resemble the active intervention (Chaibi et al., 2015) therefore detuned laser or ultrasound and instrument manipulation would not have served as an appropriate sham intervention for this study.

Chaibi et al. (2015) utilised a placebo intervention for lumbar SM that consisted of placing a broad, no-specific contact over the lumbar area and delivering a low-velocity, low amplitude push manoeuvre with no thrust, in a non-intentional and non-therapeutic direction. A post intervention questionnaire revealed that participants were unable to say whether they received the active intervention or the placebo. Placebo methods utilised in this manner result in stimulation of the afferent receptors and may influence the results of the participant, therefore the placebo and active intervention cannot be used on the same participant (Chaibi et al., 2015).
The sham manipulation in the current study consisted of contacting the participants’ foot and ankle complex and passively placing it in the same position as for the talocrural joint manipulation, however no thrust was applied. Caution was taken to maintain adequate soft tissue and joint slack so no joint cavitation occurred. This procedure was adequate enough to serve as the sham manipulation in the current study according to guidelines set by Vernon et al. (2005) and Chaibi et al. (2015).

2.6 Conclusion

Chronic ankle instability syndrome is often a result of inadequate initial treatment and rehabilitation and can result in a decreased quality of life (Ferran and Maffulli, 2006). The literature shows that one of the main contributing factors to the development of CAIS is AMI, particularly of the peroneal and soleus muscles (Caufield, 2000; McVey et al., 2005; Setton et al., 2008; Palmieri-Smith et al., 2009; Klykken et al., 2011). Rehabilitation forms a major part of the treatment protocol for CAIS. However, optimum muscle functioning cannot be achieved without correcting the afferent input to the spinal cord from the involved joint leaving the ankle susceptible to re-injury (Hopkins and Ingersoll, 2000; McVey et al., 2005).

The literature demonstrates that ankle joint manipulation is clinically beneficial in the treatment of CAIS (Pellow and Brantingham, 2001; Lindsey-Renton, 2005; Lubbe et al., 2015) however, the neurophysiological mechanisms supporting these clinical benefits are under-investigated (Evans, 2002; Fryer et al., 2002; Pickar, 2002; Maigne and Vautravers, 2003; Andersen et al., 2003; Bialosky et al., 2009; Brantingham et al., 2009; Ritter, 2014).

Grindstaff et al. (2011) found that distal tibiofibular joint manipulation resulted in a statistically significant increase in soleus muscle activity without an increase in peroneal muscle activity in CAIS. This suggests that the afferent input as a result of manipulation of the distal tibiofibular joint did not have an effect on the peroneal muscles and therefore cannot aid in correcting the AMI associated with CAIS. Distal tibiofibular joint and talocrural joint restrictions occur in lateral ankle sprains however, talocrural joint restrictions are more prominent (Denegar et al., 2002).
Therefore this study aimed to demonstrate the neurophysiological effect of talocrural joint manipulation on peroneal and soleus muscle activity in CAIS, in order to contribute to the knowledge on the neurophysiological effect of manipulation.
Chapter Three

3.1 Introduction

This chapter will describe the methodology utilised in this study along with the ethical considerations that were taken to ensure participant safety and well-being.

3.2 Study design

The study was done in the quantitative paradigm using an experimental, pre-test post-test study design. This design allowed for the random allocation of participants to two or more groups, where all groups were tested prior to the administration of the intervention, then following the intervention, all groups were re-tested in order to determine the effect of the independent variable (Kirk, 2003).

The dependant variable in this study was the H/M ratio and the independent variable was talocrural joint manipulation.

3.3 Location of study

The study was conducted at the Chiropractic Day Clinic (CDC) at the Durban University of Technology (DUT) Ritson campus. A letter granting permission to use the premises was obtained from the Clinic Director (Appendix B). Ethical approval to conduct the study was obtained from the DUT Institutional Research Ethics Committee (IREC 116/15: Appendix C).

3.4 Participant recruitment

Participants were recruited from the eThekwini municipal area. Advertisements (Appendix D) were placed on the DUT campus, other local universities and various gyms and sports clubs. Permission was obtained from the premises prior to the placement of the advertisements (Appendix E). Prospective participants were also recruited by word of mouth.

3.5 Population

Prospective participants contacted the researcher (using the contact information supplied on the advertisement or by word of mouth) where the researcher provided
additional information regarding the study. Those that wished to participate thereafter were asked the following qualifying questions, detailed in table 3.1:

**Table 3.1: Telephonic questions and answers.**

<table>
<thead>
<tr>
<th>Question</th>
<th>Required answer for participation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Are you willing to answer some questions with regard to participating in this research study?</td>
<td>Yes.</td>
</tr>
<tr>
<td>2. How old are you?</td>
<td>Between the ages of 18 and 45 years.</td>
</tr>
<tr>
<td>3. When did you first sprain your ankle?</td>
<td>Longer than three months prior to the consultation.</td>
</tr>
<tr>
<td>4. How many times have you sprained your ankle?</td>
<td>Twice or more.</td>
</tr>
<tr>
<td>5. Are you currently taking any pain medication or muscle relaxants?</td>
<td>No, if yes, then the participant required a three day washout period prior to participation in the study.</td>
</tr>
<tr>
<td>6. Have you ever sustained a serious injury, broken any bones or undergone any surgery (e.g. for plantar fasciitis, ligament repair etc.) on the foot and ankle that you have sprained?</td>
<td>No.</td>
</tr>
</tbody>
</table>

If the respondent met the criteria they were invited to attend a consultation at the CDC at DUT. They were asked to bring their identity document to the initial consultation. At the consultation they were given a verbal explanation of the study followed by a letter of information (Appendix F) and informed consent (Appendix G) to read an complete. They were given an opportunity to ask research related questions that they may have had. Once the participants agreed to participate they were told that they were free to withdraw from the study at any time. The participant then underwent a case history (Appendix H), senior physical (Appendix I), foot and ankle regional examination (Appendix J) and a SOAPE note (Appendix K) in order to determine their eligibility for the study against the following inclusion and exclusion criteria:

### 3.5.1 Inclusion criteria

1. Participants were required to be between the ages of 18 and 45 years. This excluded participants that had not yet completed skeletal maturity and that
may possibly have had degenerative changes to bones and joints. This aided in ensuring population homogeneity.

2. A clinical diagnosis of CAIS was required. The diagnosis was made if the participant met the following criteria (Gribble et al., 2013):

1) A history of at least one significant ankle sprain, that would have either been a grade one or grade two ankle sprain (Reid, 1992; Caufiled, 2000; Pellow and Brantingham, 2001; Ajis and Maffulli, 2006). The grading method used takes into account the number of ligaments injured and was based on clinical severity (Lynch, 2002).

   Grade one: Mild sprain with mild damage to the ligaments, no haemorrhage or bruising, minimal oedema, point tenderness and no gross instability.

   Grade two: Moderate sprain with partial tearing of the ligaments, minimal haemorrhage or bruising if at all, localised oedema, minimal instability.

2) A history of the previously injured ankle joint “giving way”.

3) A general self-reported foot and ankle function questionnaire, this was only required if the level of self-reported function is important to the research.

3. Participants were required to give informed consent (Appendix G) prior to participation in the study.

3.5.2 Exclusion Criteria

1. Participants who had sustained an acute injury or an acute re-injury three months prior to the consultation were excluded from the study (Gribble et al., 2013).

2. Participants who presented with a primary or secondary disorder that could mimic instability (e.g. connective tissue disorders) or that could affect normal neurological functioning (e.g. uncontrolled diabetes mellitus and peripheral neuropathies), as determined through clinical examination, were excluded.

3. Participants who presented with diffuse oedema on both sides of the Achilles tendon, early haemorrhage and bruising, possible tenderness medially and laterally and gross instability were excluded from the study, this was indicative of grade three ankle sprains (Reid, 1992; Caufield, 2000; Pellow and Brantingham, 2001; Ajis and Maffulli, 2006).
4. Participants who presented with any absolute or relative contra-indications to manipulation based on the findings of the case history, physical examination and foot and ankle regional examination were excluded from the study (Bergman et al., 1993; Pellow and Brantingham, 2001).

5. Participants who were on any pain medication or muscle relaxants were excluded unless they were willing to undergo a three day washout period prior to participating in the study (Poul et al., 1993; Dreyer et al., 2012).

3.6 Sampling strategy

3.6.1 Sample size

A sample size calculation was done using GPower version 3.1.9.2. The sample size was calculated at 80% power, with a medium effect size of 0.25 and an alpha of 0.05, using repeated measures ANOVA with in-between interactions. This resulted in a sample of 42 participants being required to participate in the study.

3.6.2 Sample allocation

Participants were randomly allocated into one of three groups using a randomisation table obtained from GraphPad Software, QuickCalcs (GraphPad Software Inc, 2015). The researcher was not blinded to the group allocation.

- Group one – talocrural joint manipulation group.
- Group two – sham talocrural joint manipulation group.
- Group three – control group where no active treatment was given.

3.7 Intervention

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

Group one – the participants received a long axis talocrural joint separation adjustment. This Chiropractic technique involved setting up the ankle joint in dorsiflexion and eversion prior to the long axis thrust being applied (Bergman et al., 1993; Lawrence, 2001). This was done to minimise trauma to the lateral ligament complex of the previously injured ankle joint (Bergman et al., 1993; Lawrence, 2001). The adjustment technique utilised in this study is the same technique used by Pellow

Group two – the participants underwent a sham talocrural joint manipulation which consisted of the same setup as per group one who received the long axis talocrural joint separation adjustment however, no thrust was applied. This group aided the researcher in determining if the effects were from the setup of the manipulation, and not the manipulation itself.

Group three – the control group involved the participants remaining motionless for approximately three seconds which was the average time taken for the setup and manipulation in groups one and two respectively. This group allowed comparison to no intervention, and the passing of time as a variable.

3.8 Measurement tools

The surface electromyographic (sEMG) equipment utilised in this study was the Biopac – Bionomadix complete wireless research system (Biopac Systems Inc, 2015). The complete system included the MP150 Data Acquisition System, Acqknowledge software and the Bionomadix Dual-channel Wireless EMG Transmitter and Receiver Pair (Biopac Systems Inc, 2015). The EMG data was transmitted at a rate of 2000Hz; raw data was bandlimited from 5.0Hz to 500Hz and the system incorporated internal highpass and lowpass filters that provided for high quality amplification of the EMG waveform (Biopac Systems Inc, 2015). The highpass and lowpass filters aided in eliminating noise and artefact interference. Care was taken to ensure that there were no other electrical devices in close proximity to the EMG machine and that the room was as quiet at possible by ensuring that all windows and doors were closed and the people in the surrounding area were aware of the study being conducted (Clancy et al., 2002).

The electrodes that were used were disposable, 11mm diameter, pre-gelled Ag/AgCl conductors (EL503, Biopac Systems Inc, 2015). The placement areas of the electrodes were shaved and cleaned with alcohol swabs (Palmieri et al., 2004; McVey et al., 2005; Tucker et al., 2005; Grindstaff et al., 2011). The sEMG electrodes were placed two centimetres apart on the muscle bellies of the peroneal and soleus muscles. The electrodes on the peroneal muscles were placed two
centimetres distal to the head of the fibula. The electrodes on the soleus muscle were placed three centimetres distal to the medial head of the gastrocnemius muscle. A ground or reference electrode was placed on the ipsilateral medial malleolus (Palmieri et al., 2004; McVey et al., 2005; Tucker et al., 2005; Grindstaff et al, 2011; Klykken et al, 2011). The stimulating electrode cathode was placed in the superior medial portion of the popliteal fossa to stimulate the sciatic nerve prior to its bifurcation into the tibial and common peroneal nerves. The anode was placed two centimetres distal to the cathode in the popliteal fossa in a longitudinal fashion (Chen and Zhou, 2011). The procedure for finding the correct placement of the cathode was as follows: the cathode was placed over the fibula head and 1ms square wave impulse was delivered with an intensity that elicited a motor response, the stimulating electrode was then moved in a superomedial direction until a motor response was observed in both the peroneal and soleus muscles (Palmieri et al., 2004; Tucker et al., 2005; Grindstaff et al., 2011; Klykken et al., 2011).

H-reflex and M-wave measurements were elicited using the STM100C Biopac Stimulator Module (Biopac Systems Inc, 2015) with a 200V maximum stimulus isolation adaptor (STMISOC, Biopac Systems Inc, 2015). Care was taken to ensure that the electrodes were placed on the same side of the body (i.e. only on the left side or only on the right side of the body to ensure that the current did not pass through the heart), and that the electrodes were placed as far away from the heart as possible.

There are multiple ways in which H-reflex studies may be set up, according to Palmieri et al., 2004; Tucker et al., 2005; Grindstaff et al., 2011; Klykken et al., 2011 this set up is appropriate for eliciting H-reflex in the lower limb. The Biopac Systems Inc (2015) set up utilized in this study has shown to be reliable and accurate in H-reflex testing (Hoffman et al., 2008; Querry et al., 2008; Kim et al., 2015).

### 3.9 Study procedure

The participants that fitted the criteria for the telephonic interview presented to the Chiropractic Day Clinic for the initial consultation. Once the initial examination was complete the participant was then required to lie on a plinth for the pre-intervention measurements.
The placement areas of the electrodes (EL503, Biopac Systems Inc, 2015) were shaved and cleaned with alcohol swabs (Palmieri et al., 2004; McVey et al., 2005; Tucker et al., 2005; Grindstaff et al., 2011) after which the electrodes were placed as mentioned above.

Participants were then placed supine on the plinth. The test limb was placed in 15° of knee flexion (measured using a goniometer) and the plantar surface of the foot placed on a secure 45° wedge (self-made) thereby supporting the foot in a slightly plantarflexed position (Tucker and Turker, 2004; Tucker et al., 2005). The slightly plantarflexed position of the foot while the knee was in flexion resulted in relaxation of the gastrocnemius muscle thereby reducing potential depressive influence on the soleus muscle (Tucker and Turker, 2004; Tucker et al., 2005). These H-reflex testing methods have been reported to be reliable and accurate (Palmieri et al., 2004; Tucker et al., 2005; Knikou and Taglianetti, 2006; Chen and Zhou, 2011; Grindstaff et al., 2011; Klykken et al., 2011; Kim et al., 2015). This participant positioning, as seen in Figure 3.1, was used during all measurements.

![Participant positioning](image)

**Figure 3.1:** Participant positioning

Once the participant was in position he/she was informed to remain motionless and relaxed while the measurements were taken. The pre-intervention $H_{\text{max}}$ and $M_{\text{max}}$ measurements were then taken. In order to obtain the $H_{\text{max}}$ and $M_{\text{max}}$ a series of 1ms square wave impulses were delivered at increasing increments of 0.2V with 10 seconds rest in between stimuli, the rest was to ensure that post activation depression did not interfere with the H-reflex amplitude. Once the $H_{\text{max}}$ was achieved three measurements were recorded (Tucker and Turker, 2004; Tucker et al., 2005;
Grindstaff et al., 2011; Klykken et al., 2011). The stimulus intensity was then increased beyond the level of the $H_{\text{max}}$ until the $M_{\text{max}}$ was obtained. Once the $M_{\text{max}}$ was determined three measurements were taken. The average of the three $H_{\text{max}}$ and $M_{\text{max}}$ measurements were then expressed as a ratio $H/M$ (Palmieri et al., 2004; Tucker and Turker, 2004; Tucker et al., 2005; Grindstaff et al., 2011; Klykken et al., 2011). This served as the pre-intervention measurement. The participant then underwent the relevant intervention according to the group he/she fell into. Immediately after the intervention the post-intervention measurements were taken in the same method as the pre-intervention measurements (Palmieri et al., 2004; Tucker and Turker, 2004; Tucker et al., 2005; Grindstaff et al., 2011; Klykken et al., 2011). Figure 3.2 shows an example of the H-reflex and M-wave on the raw sEMG data obtained from the study participants.

![Figure 3.2: Participant raw sEMG recordings. A) H-reflex response of the soleus and peroneal muscles. B) As the stimulation intensity increased the H-reflex amplitude decreased and the M-wave began to appear. C) At the maximum amplitude of the M-wave the H-reflex was completely absent.](image)

### 3.10 Data analysis

The H-reflex amplitude varies considerably between individuals therefore in order to compare the readings between individuals the data needed to be normalised. This study utilised the method of standardising the $H_{\text{max}}$ to the $M_{\text{max}}$ amplitude ($H/M$ ratio). The $H_{\text{max}}$ is an estimate of the number of motor neurons being recruited and the $M_{\text{max}}$...
is the total motor neuron pool, therefore the H/M ratio is interpreted as the portion of the entire motor neuron pool capable of being recruited (Palmieri et al, 2004).

Data was captured using Microsoft Excel and transferred to the latest version of SPSS Statistics 23.0 (2013) and Statgraphics Centurion 15.1 (2006), were it was statistically analysed. Descriptive statistics (multivariate analysis) were used to determine means and standard deviations, data was summarised using a contingency table. Inferential statistics were used to measure the effect of the interventions (i.e. testing the hypothesis).

3.11 Ethical considerations

This study used a sham intervention and a control group. In order to not disadvantage the participants in this group they were offered one free treatment that was given by the investigator following the study. At the start of the study all participants were informed that they had a one in three chance of being allocated to one of the groups.

Each participant was treated fairly and equally with no discrimination occurring in participant selection in terms of race, gender, nationality and religion, in alignment with the ethical principle of justice.

All participants were required to sign the letter of information (Appendix F) and informed consent (Appendix G) prior to participating in the study. No coercion was used to recruit participants. In addition participant confidentiality was ensured by using the allocation of codes to the participants, ensuring that no participant names appeared in the dissertation or publication stemming from the project, allowing for participant autonomy.

Non-maleficence and beneficence: The welfare of the participants was protected as the interventions and equipment being utilised in this study are safe and registered. A free treatment was offered to the participants as compensation for participating in the study. All participant data was kept in the participant’s clinical file in the CDC, with the signed letter of information and informed consent being kept in the Chiropractic program. All research data was coded to ensure participant confidentiality. After a period of five years the research data will be shredded.
results of this study will assist manual therapists using joint manipulation to further their understanding on how manipulation results in its effects.

Permission to conduct the study in campus at the CDC was obtained (Appendix B). Permission was obtained prior to placing advertisements in the above mentioned locations (Appendix E). The DUT Institutional Research Ethics Committee granted full ethical approval prior to the commencement of the study (IREC 116/15: Appendix C).
Chapter Four

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

4.2 Sampling outcome
In total 49 individuals between the ages of 18 and 45 years suffering from CAIS were recruited for the study. From the 49 participants four were excluded due to difficulty eliciting the H-reflex, with a further three being excluded due to unavailability, this brought the total sample to 42 participants.

4.3 Demographic and anthropometric characteristics of the participants

4.3.1 Age
Table 4.1 shows the mean and standard deviation (±SD) of the age of the participants per group. Overall mean age of the participants was 26.55 years (± 6.18 years) with the range from 18 – 43 years of age. There was no statistically significant difference (p = 0.802) between the groups in terms of age.

4.3.2 Gender
The gender distribution between groups was similar. Table 4.1 shows the distribution of the males and females in each group, there was no statistically significant difference (p = 0.501) between the groups in terms of gender.

4.3.3 Body mass index
The overall mean body mass index (BMI) was 24.76 (range = 17.63 – 31.02). Equal numbers of participants were within the normal range (45.24%) and the overweight range (45.24%). The remaining participants were underweight (2.38%) and obese (7.14%). Table 4.1 shows the mean BMI for each of the groups. There was no statistically significant difference (p = 0.865) between the groups.
Table 4.1: Demographic and anthropometric characteristics of the participants

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Age Mean ±SD</th>
<th>Gender</th>
<th>Gender Mean ±SD</th>
<th>BMI Mean ±SD</th>
<th>Min</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>25.93 ± 5.57</td>
<td>4</td>
<td>24.98 ± 4.03</td>
<td>19.20 - 31.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Manipulation</td>
<td>14</td>
<td>27.36 ± 6.32</td>
<td>7</td>
<td>24.20 ± 3.49</td>
<td>17.63 - 28.69</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sham</td>
<td>14</td>
<td>26.36 ± 6.96</td>
<td>4</td>
<td>25.09 ± 2.69</td>
<td>20.82 - 30.23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>14</td>
<td>26.55 ± 6.18</td>
<td>17</td>
<td>24.76 ± 3.38</td>
<td>17.63 - 31.02</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

4.4 Electromyography

4.4.1 Soleus muscle electromyography

Baseline H-reflex and M-wave measurements of the soleus muscle revealed no statistically significant ($p > 0.05$) differences between the groups indicating that groups were homogenous at the commencement of the study. Table 4.2 shows the mean pre and post $M_{\text{max}}$ findings for the soleus muscle per group. Using Wilcoxon signed rank test no statistically significant differences were observed between the pre- and post-readings for the $M_{\text{max}}$ in each of the three groups. This indicates that the $M_{\text{max}}$ was stable in each group allowing for the H/M ratio to be assessed as a dependent measure.

Table 4.2: The pre and post $M_{\text{max}}$ readings for the soleus muscle

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre- $M_{\text{max}}$</th>
<th>Post- $M_{\text{max}}$</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manipulation</td>
<td>8.66</td>
<td>8.37</td>
<td>0.730</td>
</tr>
<tr>
<td>Sham</td>
<td>8.55</td>
<td>9.00</td>
<td>0.109</td>
</tr>
<tr>
<td>Control</td>
<td>7.97</td>
<td>8.22</td>
<td>0.198</td>
</tr>
</tbody>
</table>

Figure 4.1 shows the mean pre-intervention and post-intervention soleus muscle H:M ratios, the error bars represent one SD. Using Wilcoxon test for the intra-group analysis, no statistically significant difference was found between the pre and post measurements in the manipulation ($p = 0.975$) or the sham groups ($p = 0.056$), in contrast the control group showed a statistically significant change ($p = 0.019$) from pre-intervention to post-intervention measurements.
Using a one-way multivariate analysis of variance (MANOVA) no statistically significant differences were found between the groups. Tukey’s honest significant difference (HSD) post-hoc test resulted in a statistically significant difference ($p = 0.028$) being found between the sham and control group for the post-intervention soleus H:M ratio measures.

### 4.4.2 Peroneal muscle electromyography

Baseline H-reflex and M-wave measurements of the peroneal muscle revealed no statistically significant ($p > 0.05$) differences between the groups indicating that groups were homogenous at the commencement of the study. Table 4.3 shows the mean pre- and post- $M_{\text{max}}$ findings for the peroneal muscle per group. Using Wilcoxon signed ranked test no statistically significant differences were observed between the pre- and post-readings for the $M_{\text{max}}$ in each of the three groups. This indicates that the $M_{\text{max}}$ was stable in each group allowing for the H/M ratio to be assessed as a dependent measure.
Table 4.3: The pre and post $M_{\text{max}}$ readings for the peroneal muscle

<table>
<thead>
<tr>
<th>Group</th>
<th>Pre- $M_{\text{max}}$</th>
<th>Post- $M_{\text{max}}$</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manipulation</td>
<td>6.42</td>
<td>6.50</td>
<td>0.875</td>
</tr>
<tr>
<td>Sham</td>
<td>6.11</td>
<td>6.53</td>
<td>0.140</td>
</tr>
<tr>
<td>Control</td>
<td>5.43</td>
<td>5.71</td>
<td>0.109</td>
</tr>
</tbody>
</table>

Using Wilcoxon test was for the intra group analysis, no statistical significance ($p > 0.05$) was found between pre-intervention and post-intervention measurements within the groups. Figure 4.2 demonstrates the mean pre-intervention and post-intervention peroneal muscle H/M ratio for each of the groups, the standard error bars represent one standard deviation.

Figure 4.2: The pre-intervention and post-intervention mean and standard deviation for the peroneal muscle H/M ratio.

No statistically significant differences ($p = 0.470; F = 1.010$) were found in the inter-group analysis which was performed using a one-way MANOVA. Tukey’s HSD post-hoc test revealed no statistically significant ($p > 0.05$) differences between the groups with regards to pre-intervention and post-intervention peroneal muscle H/M ratio.
Chapter Five

5.1 Introduction

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

5.2 Discussion of the demographic and anthropometric data

5.2.1 Age

Age is important when using sEMG because an increase in age has been associated with a decrease in muscle mass, strength and physical function (Gallagher et al., 1997; Billot et al., 2010; Hairi et al., 2010). Billot et al. (2010) showed that elder individuals (±80 years of age) required a greater level of muscle activity to produce the same amount of torque as younger individuals. Increase in age has also been associated with an increased risk of developing osteoarthritis, with the incidence rate sharply increasing after the age of 50 years (Neogi and Zhang, 2013). These factors could influence the sEMG H/M ratio readings therefore the age of the study population was controlled by limiting it to 18 - 45 years of age. No statistically significant \( p = 0.802 \) difference was noted between the groups with regards to participant age. The participants of this study had a mean age of 26.55 years, this is a young sample but is similar to the participant age of previous studies (Grindstaff et al., 2011; Lubbe et al., 2015).

5.2.2 Gender

Gender has not been associated with differences in muscle activity. EMG recordings may not be affected by gender, however gender was controlled for in this study and no statistically significant \( p = 0.501 \) differences were noted between the groups adding to the homogeneity of the study. This is consistent with the gender distribution of previous studies (Kohne et al., 2007; Joseph et al., 2010; Lubbe et al., 2015).

5.2.3 Body mass index

During the study it was found to difficult to detect the H-reflex in participants with a thicker layer of subcutaneous fat. Since the subcutaneous fat layer acts as an
insulator between the muscle and the electrodes, the thicker the fat layer the smaller the signal that is picked up by the electrodes. Therefore it is possible to have higher resting sEMG amplitude in a thin individual compared to an individual with a thicker layer of subcutaneous fat.

It has been reported that there is a negative correlation between skinfold thickness and sEMG amplitude (Criswell, 2010). This is consistent with Bartuzi et al. (2010) who found that a sEMG signal is dependent on the amount of subcutaneous fat. They also found that this is dependent on the muscle being tested as certain muscles will have a thicker layer of overlying subcutaneous fat (Criswell, 2010). Nordander et al. (2003) suggested that the data be normalized to reduce the variability in data due to skin fold thickness.

The BMI ranges in this study are consistent with the BMI ranges in previous similar studies (Grindstaff et al., 2011; Grindstaff et al., 2014; Lubbe et al., 2015). Based on the observations made in this study, it is recommended that future studies control for a specific skin fold thickness over the muscle being assessed.

5.3 Electromyography

5.3.1 Soleus muscle

The results of this study demonstrate that ankle joint manipulation may not have a statistically significant effect on the muscle activity of the soleus muscle. This is in contrast to Grindstaff et al. (2011) who demonstrated a statistically significant ($p = 0.04$) increase in soleus muscle readings following inferior tibiofibular joint manipulation. Grindstaff et al. (2011) differed from the current study in that the intervention consisted of manipulation of the inferior tibiofibular joint as opposed to the talocrural joint, if no cavitation was heard the manipulation was repeated whereas the current study only performed the manipulation a single time regardless of joint cavitation. The manipulation and sham groups performed similarly with neither demonstrating a change in H/M ratio measurements; however the control group demonstrated a decrease in muscle activity.

Niazi et al (2015) found that lumbar spinal manipulation on participants with spinal dysfunction increases the net excitability of the motor neurons of the soleus muscle.
potentially increasing the muscle’s activity. This study also demonstrated a decrease in the muscle activity of the soleus muscle in the control group.

A decrease in soleus muscle activity in the control group may be attributed to muscle fatigue. Participant positioning in the current study required the participant to remain motionless in the supine position while the test limb lay in 15° of knee flexion with the foot resting on a 45° wedge (Tucker et al., 2005). This positioning was chosen to minimise participant movement during the intervention (talocrural long axis manipulation) thereby controlling for any changes that may occur as a result of participant repositioning.

Subjects were continuously reminded to remain motionless and relaxed. However it was noted that participants had difficulty keeping the test limb in a relaxed position and complained of limb fatigue. According to Chen and Zhou (2011) experimental procedures such as high intensity or prolonged contractions, long duration of studies and repeated application of electrical stimulation may lead to fatigue. Local muscle fatigue results in decreased sEMG amplitude i.e. a lower H/M ratio (Garland and McComas, 1990; Cifrek et al., 2009; Chen and Zhou, 2011; Paillard, 2012). This can be seen in the readings of the control group that demonstrated a decrease in soleus muscle H/M ratio from pre to post readings.

This effect was not observed in the intervention or sham groups. Joint manipulation has been shown to decrease or prevent muscle fatigue in symptomatic individuals (Maduro de Camargo et al., 2011; Niazi et al., 2015). Niazi et al. (2015) assessed the effects of spinal manipulation on the H-reflex, M-wave, volitional (V) wave and maximum voluntary contraction (MVC) of the plantar flexor muscles in participants with spinal dysfunction. The control group demonstrated a decreased V/M\textsubscript{max} ratio and MVC in the post measurements indicating fatigue while the manipulation group demonstrated an improvement in MVC. The authors believe that spinal manipulation may have induced significant changes in the net excitability of the muscle by way of an increased stimulation of the descending efferent neurons and/or the stimulation of the afferent supply. Based on these observations they also concluded that spinal manipulation prevents muscle fatigue.

This study differs from the current study in that it utilised spinal joint manipulation and not extremity joint manipulation. The study also incorporated MVC and V-wave
measurements. It has been suggested that the H-reflex measurements recorded at MVC provide a more functional measurement than those recorded at rest (Racinais et al., 2013). Incorporating the V-wave (which is elicited during MVC) with the H-reflex allows one to differentiate between spinal and supraspinal contribution to the muscle being tested (Aagaard et al., 2002; Racinais et al., 2013; Cattagni et al., 2014). A limitation of Niazi et al. (2015) was that they compared the spinal manipulation to a control group only, therefore it is possible that the observed effects may be a result of movement of the patient as opposed to the actual manipulation.

Maduro de Camargo et al. (2011) found that manipulation of the C5/C6 in participants with mechanical neck pain increased sEMG amplitude and resistance to fatigue of the deltoid muscle at rest and during isotonic and isometric contractions when compared to a control. This study analysed the root mean square (RMS) to assess the amplitude of the contractions and the median frequency (MF) of the EMG power spectrum (Hz) to assess muscle fatigue. This method of assessing muscle fatigue (that is the MF of the EMG power spectrum recorded during an isometric contraction) is more accurate and more widely accepted than the H/M ratio recorded at rest used in the current study (Al-Mulla et al., 2012). The H/M ratio measurement was utilised in the current study as the objective was to assess muscle activity/excitation not muscle fatigue.

The foot and ankle joint complex of the participants in the sham group was set up similar to that for a talocrural joint manipulation. This required the test limb to be passively extended, the ankle placed in a dorsiflexed and everted position and then passively held in this position for three seconds before being repositioned into the test position. This set up and movement of the limb may have allowed the supporting muscles time to relax and recover prior to the post intervention measurements. This was not the case with the control group who had to maintain the test position for the entire duration of the study.

The passive movement of the foot and ankle joint complex into this position and back to the test position may have resulted in stimulation of the mechanoreceptors within the joint and surrounding structures. It is possible that this manual technique affected the surrounding musculature in the same way as the ankle joint manipulation (Kaur et al., 2014). This may explain why no significant difference was noted between the
pre and post intervention H/M ratio measurements within the manipulation and sham groups but was evident within the control group.

The inter-group analysis revealed a statistically significant difference between the sham and control groups with regards to post soleus muscle H/M ratio measurements. It was observed from the figures that there was a difference between the manipulation and control groups' post soleus muscle H/M ratio, however this difference was not adequate enough to be of statistical significance. On the other hand the post soleus muscle H/M ratio measurements between the manipulation and sham groups' appeared to be very similar. It is possible for this to be interpreted as the control group demonstrating a decrease in post soleus muscle H/M ratio measurements when compared to the post soleus muscle H/M ratio measurements of the manipulation and sham groups indicating the presence of muscle fatigue in the control group only.

It is prudent to remember that the objective of the current study was not to assess the effects of extremity joint manipulation on muscle fatigue but to assess the effects of ankle joint manipulation on muscle activity in terms of sEMG H/M ratio measurements. With this in mind the current study demonstrated that ankle joint manipulation may not affect the muscle activity of the soleus muscle in terms of H/M ratio measurements when compared to a sham intervention and control.

5.3.2 Peroneal muscle

The peroneal muscle response in the manipulation group demonstrated the same result as the soleus muscle with no significant difference being noted between the pre intervention and post intervention measurements. This is congruent with Grindstaff et al., (2011) who demonstrated no statistically significant difference in peroneal muscle activity post distal tibiofibular joint manipulation in participants with CAIS. The sham and control groups demonstrated the same results as the manipulation group.

CAIS has been shown to affect the muscle activity of both the soleus and peroneal muscles (McVety et al, 2005; Sefton et al, 2008; Palmieri-Smith et al, 2009; Klykken et al, 2011). It is possible that even though Palmieri-Smith et al. (2009) demonstrated that AMI of the peroneal muscle is present in CAIS, the degree of inhibition of the
muscle may not be adequate enough to alter activation levels in the controlled environment of the current study. Palmieri-Smith et al. (2009) found that the affected limb only demonstrated a 10% level of inhibition when compared to the unaffected limb, it is likely that the number of motor neurons which were activated in the affected muscle were adequate enough to be unaffected by the test conditions in the current study (i.e. maintaining a static limb position while recordings being taken, receiving a single ankle joint manipulation or the set up).

Another plausible explanation for the results obtained in the current study is that sEMG recordings were taken with the muscle at rest. Recent studies have demonstrated H-reflex measurements taken while a muscle is actively contracting provide a greater occurrence, amplitude and reliability of readings (Knikou, 2008; Racinais et al., 2013; Doguet and Jubeau, 2014). The current study was performed at rest in an attempt to limit the amount of movement and stimulation of mechanoreceptors which would have been stimulated by an isometric and isotonic muscle contraction. This stimulation as a result of muscle contraction may have interfered with the objective of the study which was to determine the effect of ankle joint manipulation on the soleus and peroneal muscles in CAIS.

It must be noted that although a power analysis was conducted the study may have been under powered to obtain a significant result.
Chapter Six

6.1 Conclusion

The purpose of the current study was to determine the effects of ankle joint manipulation on soleus and peroneal muscle activity in CAIS. Analysis of the results revealed that ankle joint manipulation had no statistically significant effect on the muscle activity of the soleus and peroneal muscles in terms of sEMG H/M ratio measurements in CAIS. The results of the study were unable to reject the null hypothesis. It is plausible that ankle joint manipulation and mobilisation decreases the amount of muscle fatigue, however this was not the aim of this study. Further research is needed to determine the effects of extremity joint manipulation on muscle activity.

6.2 Limitations

There were a number of limitations in the current study therefore there is potential for future studies based on the following limitations.

Although BMI was controlled it was noted that the subcutaneous fat layer thickness differs between participants as well as between different muscles within the same participant. This may affect sEMG readings as this acts as an insulator between the muscle and electrodes.

Participant positioning was in the supine position in this study as per Palmieri et al., (2004), Tucker et al., (2005) and Grindstaff et al., (2011). This position was utilised due to talocrural joint manipulation being conducted in the supine position. However this position resulted in some difficulties with electrode placement. Palmieri et al., (2004) and Tucker et al., (2005) stated the recordings taken in a prone position are as reliable as those taken in the supine position.

Ankle joint mobility was assessed in the foot and ankle regional examination prior to the pre-intervention measurements being taken, however the mobility of the joint was not included as a variable in this study. In a clinical setting manipulation and higher grade mobilizations are reserved for participants who demonstrate hypomobility of the ankle joint. Whitman et al., (2009) developed a clinical prediction rule to
determine who will demonstrate the greatest improvements following manual therapy applied to a sprained ankle joint. It was found that one of the criteria that predict a successful outcome is ankle joint hypomobility. It can be assumed that individuals who present with CAIS associated with hypomobility of the ankle joint may demonstrate a more significant change in soleus and peroneal muscle activity following manual therapy.

Assessing the effect of ankle joint manipulation on the soleus and peroneal muscles in terms of sEMG H/M ratio only assesses the effect of the manipulation on the spinal reflex loop. It is plausible that supraspinal factors may influence the effects of the AMI on the involved muscles.

It is possible that the sample size for the study was not large enough to detect a statistically significant difference between groups. A post-hoc power calculation found that in order to have a medium to large effect, a total sample size of 67 (i.e. 22 participants per group) would be needed to observe an 80% power.

### 6.3 Recommendations

The following recommendations can be made:

1. Skinfold thickness measured over the muscle being tested should be used as participant inclusion criteria.
2. In terms of participant placement the sEMG recordings should be done with the participant in the prone position
3. It is recommended that future studies include ankle joint hypomobility as an inclusion criterion.
4. This study only evaluated the immediate effect of ankle joint manipulation. Previous studies have evaluated the effects of manipulation at various time intervals post intervention. Future studies should assess the short and long term effects of manipulation on muscle activity. This information may be helpful in providing a window of optimal muscle functioning in which rehabilitation will provide the greatest amount of benefit.
5. It is suggested that future studies include assessment of the supraspinal contribution towards the involved muscle activity and the effects of manipulation on such activity.
6. H/M ratio recordings were taken at rest in order to decrease the amount of stimulation on the mechanoreceptors from sources other than the manipulation and sham intervention. According to Racinais et al. (2013) H-reflex measurements recorded during active muscle contraction provide a greater indication of the functional performance of the muscle. Therefore it is recommended that future studies include H/M ratio measurements recorded during MVC, it may also be beneficial to include assessment of the volitional (V) wave recorded at MVC, this will allow the author to make comparisons between the spinal and supraspinal contributions to the muscle.

7. The current study assessed only the neurophysiological effects of ankle joint manipulation, it is recommended that future studies assess the neurophysiological as well as the clinical effects of ankle joint manipulation thus allowing a comparison to be made.
References


strength, mass (sarcopenia), and quality (specific force) and its relationship with functional limitation and physical disability: The concord health and ageing in men project. *Journal of American Geriatrics Society*, 58:2055-2062.


## Appendices

### Appendix A: Muscles of the leg

The muscles of the leg excluding those mentioned in the Table 2.2 (Moore *et al.*, 2010; Vizniak, 2010; McKinley and O'Loughlin, 2012).

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Origin</th>
<th>Insertion</th>
<th>Innervation</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tibialis anterior</td>
<td>Lateral condyle and superior half of lateral surface of tibia and interosseous membrane</td>
<td>Medial and inferior surfaces of medial cuneiform and base of first metatarsal</td>
<td>Deep fibular nerve (L4, L5)</td>
<td>Dorsiflexion and inversion of foot and ankle</td>
</tr>
<tr>
<td>Extensor digitorum longus</td>
<td>Lateral condyle of tibia and superior three quarters of medial surface of fibula and interosseous membrane</td>
<td>Middle and distal phalanges of lateral four digits</td>
<td>Deep fibular nerve (L4, L5)</td>
<td>Extends lateral four digits and dorsiflexes ankle</td>
</tr>
<tr>
<td>Extensor hallucis longus</td>
<td>Middle part of anterior surface of fibula and interosseous membrane</td>
<td>Dorsal aspect of base of distal phalanx of great toe</td>
<td>Deep fibular nerve (L4, L5)</td>
<td>Extends great toe and dorsiflexes ankle</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>Lateral head: lateral aspect of lateral condyle of femur. Medial head: popliteal surface of femur; superior to medial condyle.</td>
<td>Posterior surface of calcaneus via calcaneal tendon</td>
<td>Tibial nerve (S1, S2)</td>
<td>Plantarflexes ankle with knee extended, raises heel during walking and flexes leg at knee</td>
</tr>
<tr>
<td>Tibialis posterior</td>
<td>Interosseous membrane; posterior surface of tibia inferior to soleal line and posterior surface of fibula</td>
<td>Tuberosity of navicular, cuneiform, cuboid and sustentaculum tali of calcaneus and; bases of second,</td>
<td>Tibial nerve (L4, L5)</td>
<td>Plantarflexes ankle and inverts foot</td>
</tr>
<tr>
<td>Muscle</td>
<td>Origin</td>
<td>Insertion</td>
<td>Nerve</td>
<td>Function</td>
</tr>
<tr>
<td>--------------------------</td>
<td>--------------------------------------------------------------------------------------------</td>
<td>----------------------------------------------------</td>
<td>--------------------------------</td>
<td>--------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Flexor hallucis longus</td>
<td>Inferior two thirds of posterior surface of fibula and inferior part of interosseous membrane</td>
<td>Base of distal phalanx of great toe</td>
<td>Tibial nerve (S2, S3)</td>
<td>Flexes great toe, weakly plantarflexes ankle and supports medial longitudinal arch of foot</td>
</tr>
<tr>
<td>Flexor digitorum longus</td>
<td>Medial posterior surface of tibia inferior to soleal line and by a broad tendon to fibula</td>
<td>Bases of distal phalanges of lateral four digits</td>
<td>Tibial nerve (S2, S3)</td>
<td>Flexes lateral four digits, plantarflexes ankle and supports longitudinal arches of foot</td>
</tr>
<tr>
<td>Plantaris</td>
<td>Inferior end of lateral supracondylar line of femur and oblique popliteal ligament</td>
<td>Posterior surface of calcaneus via calcaneal tendon</td>
<td>Tibial nerve (S1, S2)</td>
<td>Weakly assists in plantarflexing the ankle</td>
</tr>
</tbody>
</table>
Appendix B: Permission to use Chiropractic Day Clinic

MEMORANDUM

To: Prof Puckree
   Chair: RHDC

   Prof Adam
   Chair: IREC

From: Dr Charmaine Korporaal
      Clinic Director: FoHS

Date: 21.05.2015

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

Permission is hereby granted to:

Mr Jason Travis Dicks (Student Number: 21011530)

Research title: The effect of ankle joint manipulation on peroneal and soleus muscle activity in patients with chronic ankle instability syndrome.

It is noted that Mr Dicks is currently a M.Tech: Chiropractic student, therefore it is requested that Mr Dicks submit a copy of his RHDC / IREC approved proposal a to the Clinic Administrators before he starts with his research in order that any special procedures with regards to his research can be implemented prior to the commencement of him seeing patients.

Thank you for your time.

Kind regards

Dr Charmaine Korporaal
Clinic Director: FoHS Clinic

Cc: Mrs Pat van den Berg: Chiropractic Day Clinic
   Dr L O’Connor: Research co-ordinator / Research supervisor
   Dr T Puckree: Research co-supervisor
Appendix C: IREC approval

29 September 2015

IREC Reference Number: REC 125/15

Mr J T Dicks
P o Box 15725
Beacon Bay
East London
5201

Dear Mr Dicks

The effect of ankle joint manipulation on peroneal and soleus muscle activity in chronic ankle instability syndrome

I am pleased to inform you that Full Approval has been granted to your proposal REC 125/15.

The Proposal has been allocated the following Ethical Clearance number IREC 116/15. Please use this number in all communication with this office.

Approval has been granted for a period of two years, before the expiry of which you are required to apply for safety monitoring and annual recertification. Please use the Safety Monitoring and Annual Recertification Report form which can be found in the Standard Operating Procedures [SOPs] of the IREC. This form must be submitted to the IREC at least 3 months before the ethics approval for the study expires.

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 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 M Ntshiba
Deputy Chairperson: IREC
Appendix D: Advertisement

Do you suffer from recurrent ankle sprains and are between the ages of 18 and 45?

Research is currently being carried out at the Durban University of Technology.

Free treatment!

To those who qualify to participate in the study.

For more information contact Jason on 031 3732205
Appendix E: 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 effects of ankle joint manipulation on peroneal and soleus muscle activity in chronic ankle instability syndrome (CAIS).

Brief description: This study will assess the effect of ankle joint manipulation on the peroneal and soleus muscles in patients with CAIS. The study requires 42 participants between the ages of 18 and 45 who experience recurrent ankle sprains. All participants will be randomly allocated into one of three groups, group one receiving manipulation, group two a sham group and group three a control group. Each group will undergo the same pre- and post-intervention testing. The results of this study will be used to add to the knowledge of the effect of manipulation in the treatment of CAIS.

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.....................
Dear participant

Thank you for your interest in this research study.

**Title of study:** The effects of ankle joint manipulation on peroneal and soleus muscle activity in chronic ankle instability syndrome.

**Principle investigator:** Jason Dicks

**Co-investigators:** Dr. L. O'Connor (M.Tech Chiropractic) 
Prof. L. Puckree (PhD Exercise physiology)

**Brief introduction and purpose of this study:** You have been selected to participate in a study to investigate the effects of ankle joint manipulation on peroneal and soleus muscle activity in patients with chronic ankle instability syndrome. 42 participants, including you, will take part in this study. The results of this study will be used to add to the knowledge of the effects of manipulation in the treatment of patients with chronic ankle instability syndrome.

**Procedure:** All participants will be randomly allocated into three groups, one group receiving manipulation, one group a sham intervention and the other group acting as a control. Each group will undergo the same pre- and post-intervention testing. This study will take place in a single consultation.

**Risks and costs:** The intervention is safe and is unlikely to cause any side effects, slight tenderness may be experienced, however, this is common post manipulation. The testing procedures are safe and will not give any discomfort. There will be no cost involved for the participant.

**Benefits:** You will receive no remuneration for taking part in this study. Your participation will aid in adding to the knowledge of the chiropractic profession and thus increasing the efficacy of treatment provided for chronic ankle instability syndrome. On completion of your participation you will be eligible for a free follow up treatment at the chiropractic day clinic (CDC) at the Durban University of Technology.

**Withdrawal from the study:** You are free to withdraw from the study at any stage.

**Confidentiality:** All patient information will be kept strictly confidential and stored in the CDC for a period of 5 years after which the files will be shredded. The results of the study will be made available in the Durban University of Technology’s library in the form of a dissertation; no confidential patient documentation will be available.

**Persons to contact with any problems and questions:** Should you have any queries regarding the study, please feel free to contact my supervisor Dr. O’Connor on lauraw@dut.ac.za or co-supervisor Prof. Puckree on puckreet@dut.ac.za. Alternatively you
can contact me on jason.t.dicks@gmail.com. Please feel free to forward any concerns to the Durban University of Technology Research Office, you may contact Prof. Moyo at moyos@dut.ac.za or on 0313732576.
Appendix G: Informed consent

Statement of agreement to participate in this study:

I..................................................................... (Participant’s full name), ID number ................................................, have read the above written information (Letter of Information) in its entirety and understand its contents. Any questions have been answered and explained to me sufficiently by............................................ I am aware that the results of the study, including my personal details regarding my sex, age, date of birth, initials and diagnosis will be anonymously processed into a study report. I agree that the data collected during this study can be processed in a computerised system by the researcher. Furthermore, I understand that I may withdraw from this study at any stage without any consequences to me and my future health care. I therefore give my consent to fully participate in this research study.

Participant’s name..............................
Participant’s signature............................ Date....................

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

Researcher’s name..............................
Researcher’s signature............................ Date....................

Witness’ name.................................
Witness’ signature.............................. Date....................
Appendix H: Case history form
**Student's Case History:**

1. **Source of History:**

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

3. **Present Illness:**

<table>
<thead>
<tr>
<th>Location</th>
<th>Complaint 1 (principle complaint)</th>
<th>Complaint 2 (additional or secondary complaint)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Initial:</td>
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</tr>
<tr>
<td></td>
<td>Recent:</td>
<td></td>
</tr>
<tr>
<td>Cause:</td>
<td></td>
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<td>Duration</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frequency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain (Character)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Progression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aggravating Factors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Relieving Factors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Associated S &amp; S</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous Occurrences</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Past Treatment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outcome:</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th></th>
<th>Noted</th>
<th>Family member</th>
<th>Noted</th>
<th>Family member</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcoholism</td>
<td></td>
<td>Headaches</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anaemia</td>
<td></td>
<td>Heart Disease</td>
<td></td>
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<tr>
<td>Arthritis</td>
<td></td>
<td>Kidney Disease</td>
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<td>CA</td>
<td></td>
<td>Mental Illness</td>
<td></td>
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</tr>
<tr>
<td>DM</td>
<td></td>
<td>Stroke</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drug Addiction</td>
<td></td>
<td>Thyroid Disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epilepsy</td>
<td></td>
<td>TB</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other (list)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
9. Review of Systems (please highlight with an asterisk those areas that are a problem for the patient and require further investigation)

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

![Physical examination form](image)

<table>
<thead>
<tr>
<th>Patient Name:</th>
<th>File no:</th>
<th>Date:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Student:</td>
<td>Signature:</td>
<td></td>
</tr>
</tbody>
</table>

#### 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: |
Appendix J: Foot and ankle regional examination form

<table>
<thead>
<tr>
<th>Active movements</th>
<th>R</th>
<th>L</th>
<th>Non weight bearing</th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plantar flexion</td>
<td></td>
<td></td>
<td>50°</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dorsiflexion</td>
<td></td>
<td></td>
<td>20°</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supination</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pronation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toe dorsiflexion</td>
<td></td>
<td></td>
<td>40° (mtp)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toe plantar flexion</td>
<td></td>
<td></td>
<td>40° (mtp)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Passive movement motion palpation (Passive ROM quality, ROM overpressure, joint play)</th>
<th>R</th>
<th>L</th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle joint: Plantarflexion</td>
<td></td>
<td></td>
<td>Subtalar joint: Varus</td>
<td></td>
</tr>
<tr>
<td>Dorsiflexion</td>
<td></td>
<td></td>
<td>Valgus</td>
<td></td>
</tr>
<tr>
<td>Talocrural: Long axis distraction</td>
<td></td>
<td></td>
<td>Midtarsal: A-P glide</td>
<td></td>
</tr>
<tr>
<td>First ray: Dorsiflexion</td>
<td></td>
<td></td>
<td>P-A glide</td>
<td></td>
</tr>
<tr>
<td>Plantarflexion</td>
<td></td>
<td></td>
<td>rotation</td>
<td></td>
</tr>
<tr>
<td>Circumduction of forefoot on fixed rearfoot</td>
<td></td>
<td></td>
<td>Intemetatarsal glide</td>
<td></td>
</tr>
<tr>
<td>Interphalangeal joints: L-A dist</td>
<td></td>
<td></td>
<td>Tarso metatarsal joints: A-P</td>
<td></td>
</tr>
<tr>
<td>A-P glide</td>
<td></td>
<td></td>
<td>Metatarsophalangeal dorsi flexion (with associated plantar flexion of each toe)</td>
<td></td>
</tr>
<tr>
<td>lat and med glide</td>
<td></td>
<td></td>
<td>rotation</td>
<td></td>
</tr>
<tr>
<td>Resisted Isometric movements</td>
<td>R</td>
<td>L</td>
<td>R</td>
<td>L</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Knee flexion</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plantar flexion</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dorsiflexion</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supination (inversion)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Neurological</th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dermatomes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myotomes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reflexes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Balance/proprioception</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Special tests</th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior drawer test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Talar tilt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thompson test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Homan sign</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tinel's sign</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test for rigid/flexible flatfoot</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kleiger test (med. deltoid)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Alignment</th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heel to ground</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fiss line</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tibial torsion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heel to leg (subtalar neutral)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subtalar neutral position</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Forefoot to heel (subtalar &amp; Midtarsal neutral)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>First ray alignment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digital deformities</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digital deformity flexible</td>
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<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Palpation</th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anteriorly</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medial malleoli</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Med tarsal bones, tibial (post) artery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lat. malleolus, calcaneus, sinus tarsi, and cuboid bones</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior tib/fib joint, tibia, mm of leg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior tibia, neck of talus, dorsalis pedis artery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posteriorly</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcaneus, Achilles tendon, Musculotendinous junction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plantarily</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plantar muscles and fascia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sesamoids</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix K: SOAPE note form

<table>
<thead>
<tr>
<th>Date:</th>
<th>Visit:</th>
<th>Student:</th>
<th>Signature:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attending Clinician:</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>S: Numerical Pain Rating Scale (Patient)</th>
<th>A: Student Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>Least 0 1 2 3 4 5 6 7 8 9 10 Worst</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>O:</th>
<th>P:</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Special attention to:</th>
<th>Next appointment:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Date:</td>
<td>Visit:</td>
</tr>
<tr>
<td>Attending Clinician:</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>S: Numerical Pain Rating Scale (Patient)</th>
<th>A: Student Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>Least 0 1 2 3 4 5 6 7 8 9 10 Worst</td>
<td></td>
</tr>
</tbody>
</table>