

**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 arthogenic 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 ( $H_{max}/M_{max}$ ). 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 ( $H_{\max}:M_{\max}$ )
<i>N</i> :	Number of participants (total sample)
<i>p</i> :	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 <sup>++</sup> :	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 Maffuli, 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 rationale 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.

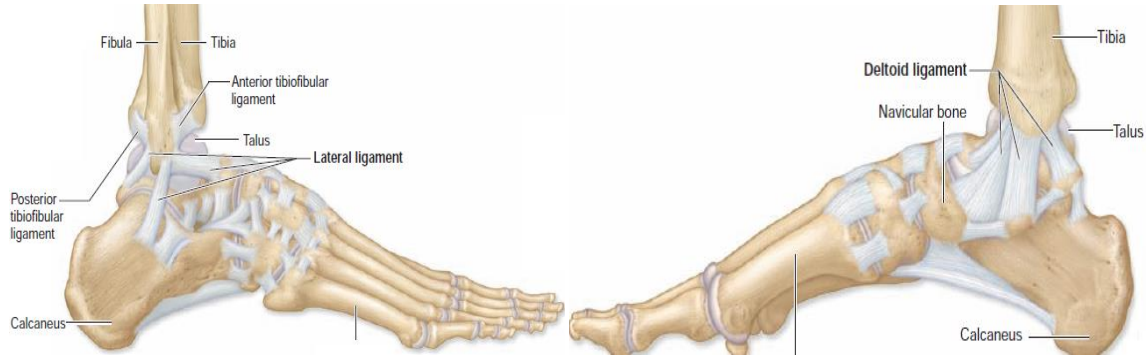
Key terms used included: “chronic ankle instability syndrome”, “ankle instability”, “ankle sprain”, “epidemiology”, “incidence and prevalence”, “mechanisms of manipulation”, “neurophysiology of manipulation”, “ankle joint manipulation”, “extremity joint manipulation”, “arthrogenic muscle inhibition”, “pathophysiology”, “surface electromyography”, “Hoffmann reflex”, “H-reflex and M-wave in the soleus muscle”, “H-reflex and M-wave in the peroneal muscles”, “guidelines to chronic ankle instability syndrome”.

## 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).

Movement	Normal range
Inversion	0°-35°
Eversion	0°-25°
Dorsiflexion	0°-20°
Plantarflexion	0°-50°

### 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).

Muscle	Origin	Insertion	Innervation	Action
Peroneus tertius	Inferior third of anterior surface of fibula and interosseous membrane	Dorsum of the fifth metatarsal base	Deep fibular nerve (L4, L5)	Dorsiflexes ankle and aids in eversion
Peroneus longus	Head and superior two thirds of the lateral surface of the fibula	First metatarsal base and medial cuneiform	Superficial fibular nerve (L5, S1, S2)	Everts foot and weakly plantarflexes ankle
Peroneus brevis	Inferior two thirds of lateral surface of fibula	Tuberosity on dorsal, lateral side of fifth metatarsal base	Superficial fibular nerve (L5, S1, S2)	Everts foot and weakly plantarflexes ankle
Soleus	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	Posterior surface of calcaneus via calcaneal tendon	Tibial nerve (S1, S2)	Plantarflexes ankle independent of position of knee and steadies leg on foot

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 sarcolemma resulting in the release of calcium ( $\text{Ca}^{++}$ ) ions and skeletal muscle contraction (Tortora and Derrickson, 2009; Snell, 2010; McKinley and O'Loughlin, 2012).

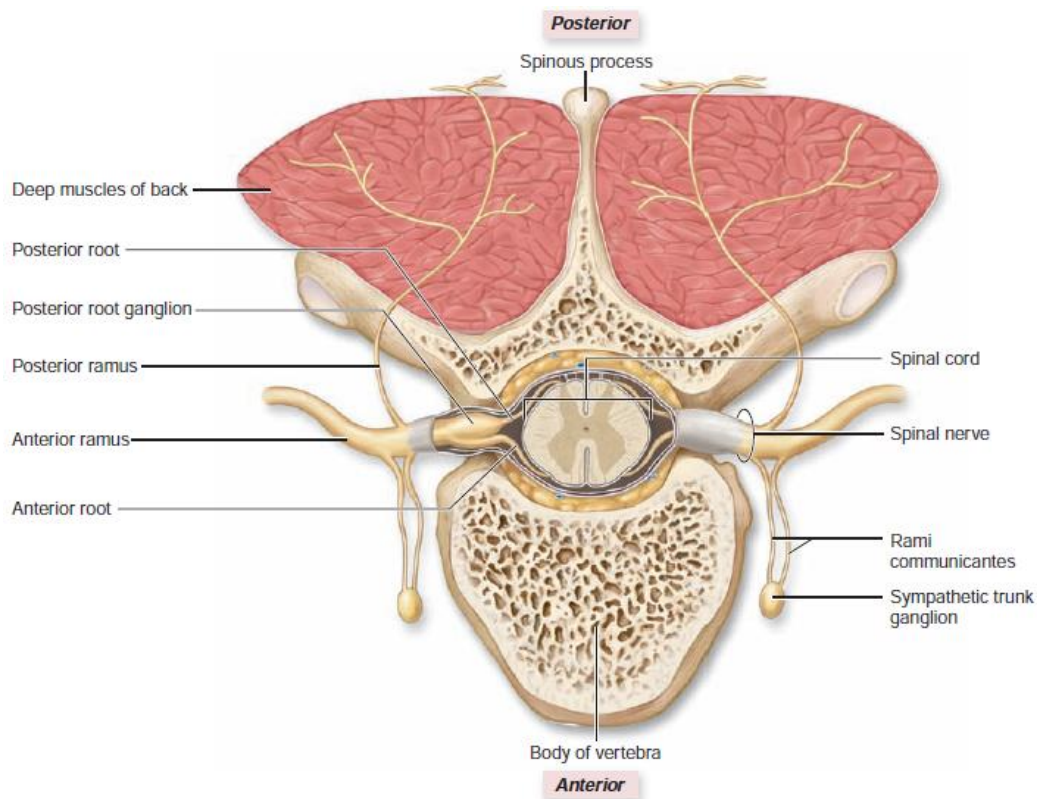
### **2.2.3 Innervation of the ankle joint**

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/effector 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).



**Figure 2.2:** Spinal nerve anatomy (McKinley and O’Loughlin, 2012).

### 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

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-sprain following an acute ankle sprain (Caulfield, 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 (Caulfield, 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 Brantigham, 2001; Lynch, 2002).

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

Grade	Description
I	Microscopic damage without any macroscopic damage.
II	Macroscopic stretching/damage while the ligament remains intact.
III	Complete tear of the ligament.

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).

Grade	Description
I	Stretching of the ATFL.
II	Tearing of the ATFL with/without tearing of the CFL.
III	Tearing of the ATFL and CFL with a capsular tear or tear of the PTFL.

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).

Grade	Description
I	Mild sprain, mild ligament damage, no haemorrhage or bruising, minimal oedema, point tenderness and no gross instability.
II	Moderate sprain, partial tearing of the ligaments, minimal haemorrhage and bruising, localised oedema and minimal instability if at all.
III	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.

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

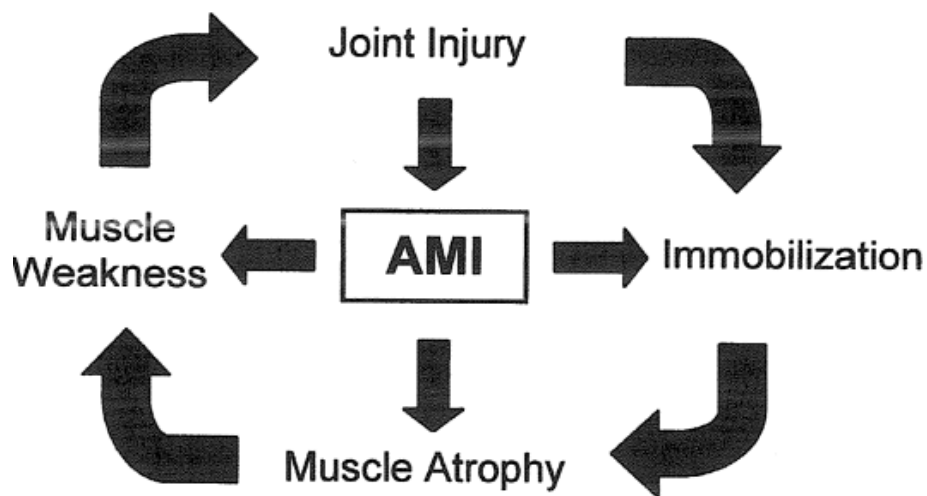
A correlation has been made between strength deficits of the peroneal muscles and an increased incidence of repeated ankle sprains however, this correlation can vary between patients (Hertel, 2002; Kaminski and Hartsell, 2002). The exact cause of the strength deficit is unclear; possibilities include 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).



**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; Ajs 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.

Author	Sample Size	Study Design	Intervention	Outcome Measures	Results
Lubbe <i>et al.</i> (2015)	N=30	RCT	1: T/C JM and Rehabilitation 2: Rehabilitation	VAS, FADI, WB dorsiflexion test, algometer, motion palpation, BBS.	1: significant improvements in VAS, algometer and motion palpation.
Joseph <i>et al.</i> , 2010.	N=40,	RCT	1: T/C HVLA JM. 2: T/C mobilisation	One leg standing test, NRS.	Both groups significant improvement in balance, ROM and function and pain.
Kohne <i>et al.</i> , 2007.	N=30	RCT	1: Single T/C JM. 2: Six T/C joint manipulations	Proprioception, ROM and point tenderness.	Significant increase in proprioception and dorsiflexion ROM in group two. Both groups improved however; group two demonstrated a greater improvement.
Pellow and Brantingham, 2001.	N=30, subacute & chronic grade I & II ankle inversion sprains	Single-blind, controlled pilot study	1: T/C JM 2: Placebo	McGill pain questionnaire, NRS, ankle dorsiflexion ROM, pain threshold and ankle function	1: a significant improvement in pain, ROM and ankle function compared to a placebo.

(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 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.

#### **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**

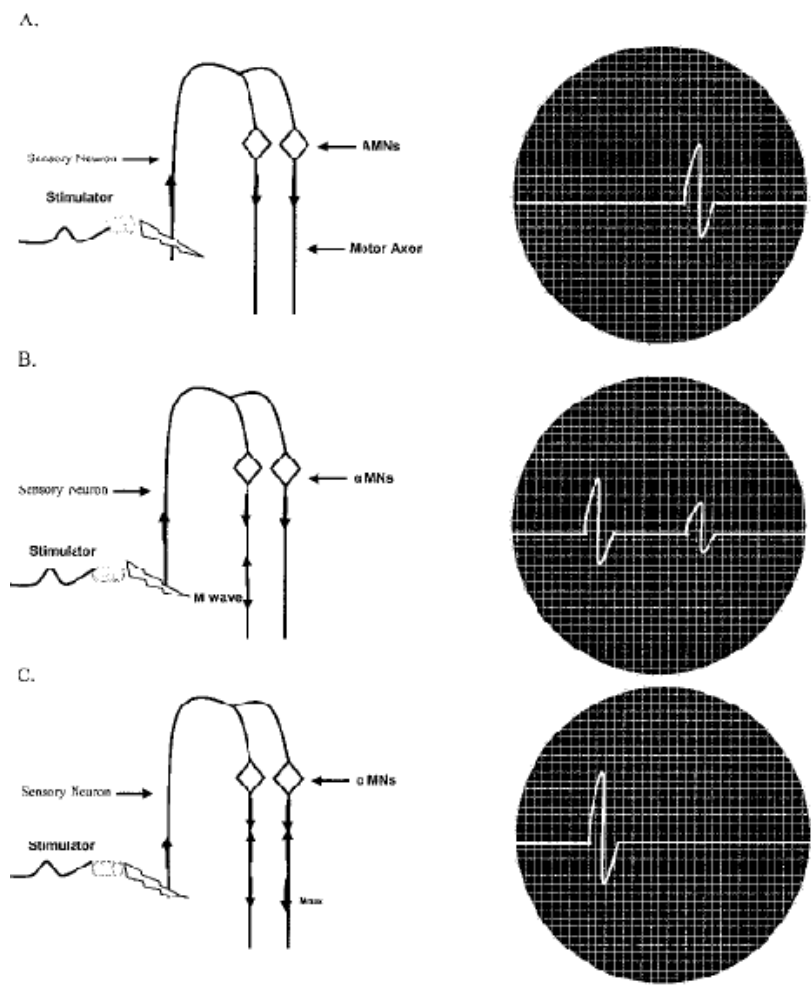
The Hoffmann Reflex (H-reflex) is one of the most widely researched reflexes in neurophysiological literature (Misiaszek, 2003; Knikou, 2008). The H-reflex is an electrically induced reflex equivalent to a mechanically induced spinal stretch reflex, the only difference being that the H-reflex bypasses 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 ( $H_{max}$ ) 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 ( $M_{max}$ ) 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  $M_{max}$  amplitude is a stable value between pre and post-intervention measurements. It has been recommended that the  $M_{max}$  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  $H_{max}$  as opposed to the  $M_{max}$  (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 2.7:** The effects of spinal manipulation on muscle activity.

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



Grindstaff <i>et al.</i> , 2014.	N=75, history of knee joint injury and current quadriceps inhibition.	RCT	1:Lumbopelvic SM 2: SM positioning (no thrust) 3: Grade IV patella mobilization 4: Grade I patella mobilization 5: Control	sEMG H-reflex of quadriceps over time (pre, post 0, 30, 60, 90 min)	No significant differences in H-reflex between groups across time.
Harvey and Descarreaux, 2013.	N=60, participants with low back pain.	RCT	1: L/S SM 2: Control	sEMG activity of PS muscles, Kinematics, Pain intensity.	No significant differences between the groups.
Fryer and Pearce, 2012.	N=14, asymptomatic participants.	RCT crossover study.	1: HVLA manipulation lumbosacral joint. 2: Control.	Motor evoked potentials and H-reflex measured from the gastroc muscle.	Significant decrease in corticospinal and spinal reflex excitability following HVLA manipulation.
Lelanne <i>et al.</i> , 2009.	N=27, participants with chronic low back pain.	RCT	1: L/S SM. 2: Control.	Trunk and pelvic angles and sEMG activity of PS muscles in trunk flexion-extension.	Significant decrease in sEMG of PS muscles at full flexion following SMT.
Colloca and Keller, 2000.	N=40, participants with low back pain.	RCT	1: Manually assisted SM. 2: Control.	sEMG of PS muscles during trunk extension maximum voluntary contraction.	Significant increase in sEMG activity of PS muscles post manually assisted SM.

(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; Grindtsaff *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 AML associated with the CAIS. Denegar *et al.* (2002) found that distal tibiofibular joint restrictions as well as 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; Sefton *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 eThekweni 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.

	Question	Required answer for participation
1.	Are you willing to answer some questions with regard to participating in this research study?	Yes.
2.	How old are you?	Between the ages of 18 and 45 years.
3.	When did you first sprain your ankle?	Longer than three months prior to the consultation.
4.	How many times have you sprained your ankle?	Twice or more.
5.	Are you currently taking any pain medication or muscle relaxants?	No, if yes, then the participant required a three day washout period prior to participation in the study.
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?	No.

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 and 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; Caufield, 2000; Pellow and Brantingham, 2001; Ajs 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; Ajs 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

and Brantingham (2001), Kohne *et al.* (2007), Joseph *et al.* (2010) and Lubbe *et al.* (2015).

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 as 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.

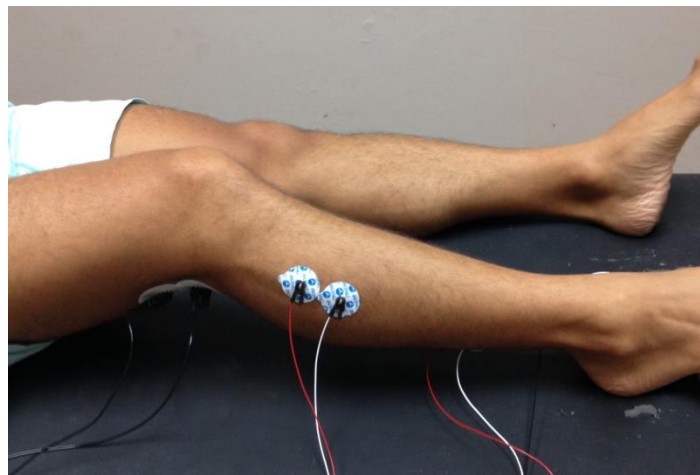
There are multiple ways in which H-reflex studies may be set up, according to Palmieri *et al.*, 2004; Tucker *et al.*, 2005; Grindstaff *et al.*, 2011; Klykken *et al.*, 2011 this set up is appropriate for eliciting H-reflex in the lower limb. The Biopac Systems Inc (2015) set up utilized in this study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

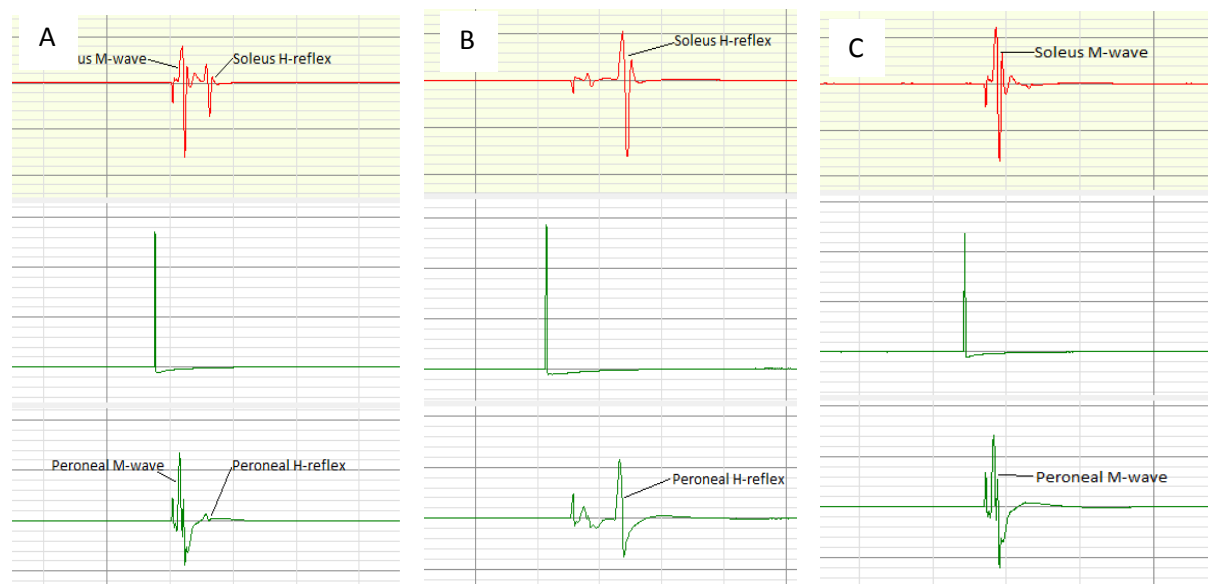
Participants were then placed supine on the plinth. The test limb was placed in 15° of knee flexion (measured using a goniometer) and the plantar surface of the foot placed on a secure 45° wedge (self-made) thereby supporting the foot in a slightly plantarflexed position (Tucker and Turker, 2004; Tucker *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.



**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  $H_{max}$  and  $M_{max}$  measurements were then taken. In order to obtain the  $H_{max}$  and  $M_{max}$  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  $H_{max}$  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  $H_{max}$  until the  $M_{max}$  was obtained. Once the  $M_{max}$  was determined three measurements were taken. The average of the three  $H_{max}$  and  $M_{max}$  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.

### 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  $H_{max}$  to the  $M_{max}$  amplitude (H/M ratio). The  $H_{max}$  is an estimate of the number of motor neurons being recruited and the  $M_{max}$

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), where 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. The

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 ( $\pm$ SD) of the age of the participants per group. Overall mean age of the participants was 26.55 years ( $\pm$  6.18years) 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

Group	N	Age		Gender		BMI			
		Mean	±SD	Female	Male	Mean	±SD	Min	Max
Manipulation	14	25.93	5.57	4	10	24.9803	4.02725	19.20	31.02
Sham	14	27.36	6.32	7	7	24.2085	3.48847	17.63	28.69
Control	14	26.36	6.96	6	8	25.0976	2.69023	20.82	30.23
Total	42	26.55	6.18	17	25	24.7621	3.38456	17.63	31.02

## 4.4 Electromyography

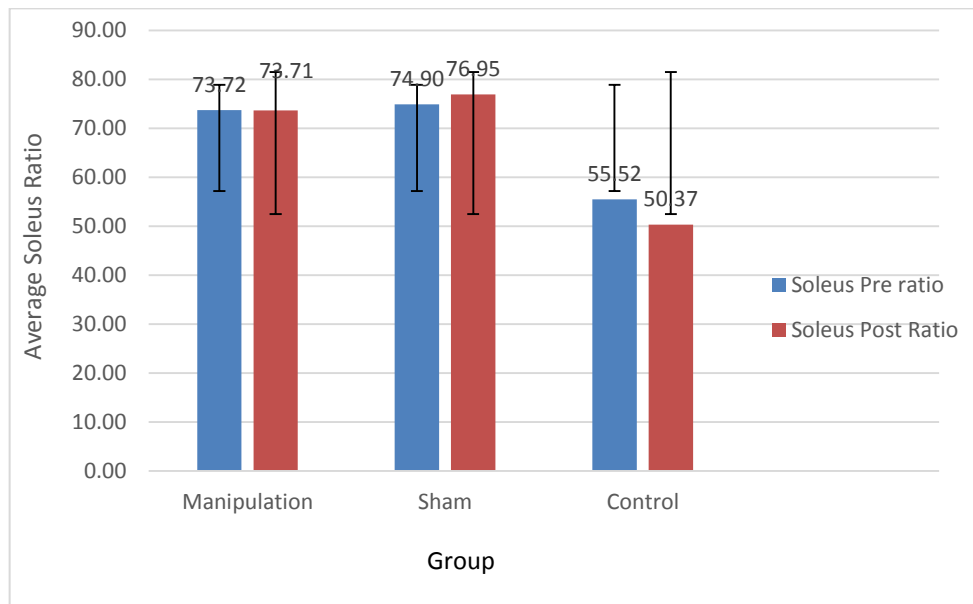
### 4.4.1 Soleus muscle electromyography

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  $M_{max}$  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  $M_{max}$  in each of the three groups. This indicates that the  $M_{max}$  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  $M_{max}$  readings for the soleus muscle

Group	Pre- $M_{max}$	Post- $M_{max}$	$p$ -value
Manipulation	8.66	8.37	0.730
Sham	8.55	9.00	0.109
Control	7.97	8.22	0.198

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.



**Figure 4.1:** The pre-intervention and post-intervention mean and standard deviation for the soleus muscle H/M ratio.

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.

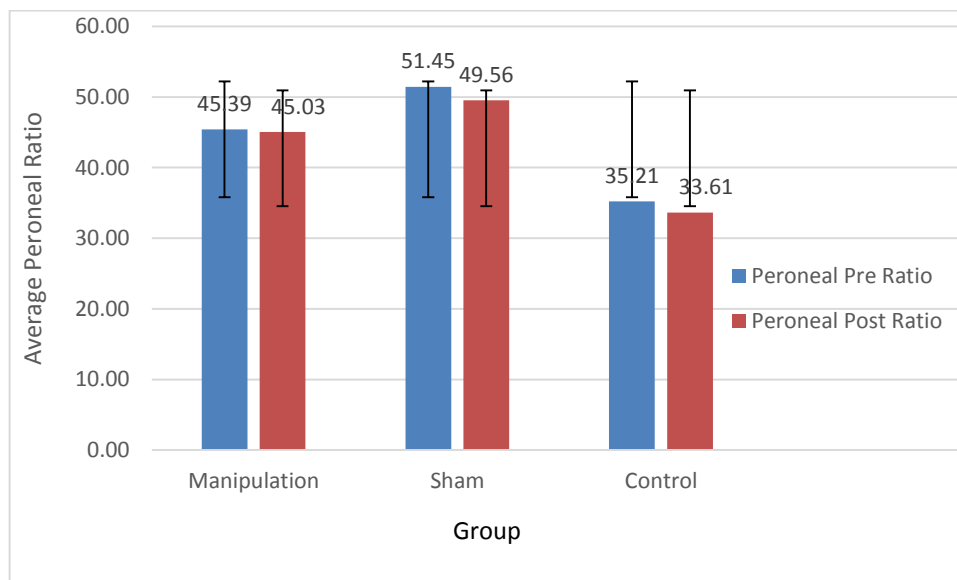
#### 4.4.2 Peroneal muscle electromyography

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-  $M_{max}$  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  $M_{max}$  in each of the three groups. This indicates that the  $M_{max}$  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  $M_{max}$  readings for the peroneal muscle

Group	Pre- $M_{max}$	Post- $M_{max}$	$p$ -value
Manipulation	6.42	6.50	0.875
Sham	6.11	6.53	0.140
Control	5.43	5.71	0.109

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 ( $\pm 80$  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<sub>max</sub> 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

- Aagaard, P., Simonsen, E.B., Andersen, J.L., Magnusson, P. and Dyhre-Poulsen, P. 2002. Neural adaptation to resistance training: changes in evoked V-wave and H-reflex responses. *Journal of Applied Physiology*, 92: 2309-2318.
- Ajis, A. and Maffulli, N. 2006. Conservative management of chronic ankle instability. *Foot and Ankle Clinic North America*, 11: 531-537.
- Al-Mulla, M.R., Sepulveda, F. and Colley, M. 2012. *EMG Methods for Evaluating Muscle and Nerve Function: sEMG Techniques to Detect and Predict Localised Muscle Fatigue*. Europe: Intech.
- Anandacoomarasamy, A. and Barnsley, L. 2005. Long term outcomes of inversion ankle injuries. *British Journal of Sports Medicine*, 39: 1-4.
- Andersen, S., Fryer G.A. and McLaughlin, P. 2003. The effect of talocrural joint manipulation on range of motion at the ankle joint in subjects with a history of ankle injury. *Australasian Chiropractic and Osteopathy*, 11(2): 57-62.
- Bartuzi, P., Tokarski, T. and Roman-Liu, D. 2010. The effect of the fatty tissue on EMG signal in young women. *Acta of Bioengineering and Biomechanics*, 12(2): 87-92.
- Beckman, S.M. and Buchanan, T.S. 1995. Ankle inversion injury and hypermobility: Effect on hip and ankle muscle electromyography onset latency. *Archives of Physical Medicine and Rehabilitation*, 76: 1138-1143.
- Bergman, T., Petersen, D. and Lawrence, D. 1993. *Chiropractic Technique Principles and Procedure*. USA: Churchill Livingstone.
- Bialosky, J.E., Bishop, M.D., Price, D.D., Robinson, M.E. and George, S.Z. 2009. The mechanisms of manual therapy in the treatment of musculoskeletal pain: A comprehensive model. *Manual Therapy*, 14(5): 531-538.
- Billot, M., Simoneau, E.M., Van Hoecke, J. and Martin, A. 2010. Age-related relative increases in electromyography activity and torque according to the maximal capacity during upright standing. *European Journal of Applied Physiology*, 109: 669-680.

- Bonnel, F., Toullec, E., Mabit, C. and Tourne, Y. 2010. Chronic ankle instability: Biomechanics and pathomechanics of ligaments injury and associated lesions. *Orthopaedics and Traumatology: Surgery and Research*, 96: 424-432.
- Brantingham, J.W., Globe, G. Pollard, H., Hicks, M., Korporaal, C. and Hoskins, W. 2009. Manipulative therapy for lower extremity conditions: expansion of literature review. *Journal of Manipulative and Physiological Therapeutics*, 32(1): 53-71.
- Bruno, A., Tacconelli, S. and Patrignani, P. 2014. Variability in the response to non-steroidal anti-inflammatory drug: mechanisms and perspectives. *Basic and Clinical Pharmacology and Toxicology*, 114: 56-63.
- Cardinale, M., Boccia, G., Greenway, T., Evans, O. and Rainoldi, A. 2015. The acute effects of spinal manipulation on neuromuscular function in asymptomatic individuals: a preliminary study. *Physical Therapy in Sport*, 16: 121-126.
- Cattagni, T., Martin, A. and Scaglioni, G. 2014. Is spinal excitability of the triceps surae affected by muscle activity or body position? *Journal of Neurophysiology*, 111: 2525-2532.
- Caulfield, B. 2000. Functional instability of the ankle joint, features and underlying causes. *Physiotherapy*, 86: 8.
- Chaibi, A., Benth, J.S. and Russell, M.B. 2015. Validation of placebo in manual therapy randomized controlled trial. *Scientific Reports*, 5:11774.
- Chan, K.W., Ding, B.C. and Mroczek, K.J. 2011. Acute and chronic lateral ankle instability in the athlete. *Bulletin of the NYU Hospital for Joint Diseases*, 69(1): 17-26.
- Chen, Y.S. and Zhou, S. 2011. Soleus H-reflex and its relation to static postural control. *Gait and Posture*, 33(2011): 169-178.
- Chinn, L. and Hertel, J. 2010. Rehabilitation of ankle and foot injuries in athletes. *Clinical Sports Medicine*, 29: 157-167.
- Cifrek, M., Medved, V., Tonkovic, S. and Ostojic, S. 2009. Surface EMG based muscle fatigue evaluation in biomechanics. *Clinical Biomechanics*, 24: 327-340.

- Clancy, E.A., Morin, E.L. and Merletti, R. 2002. Sampling noise-reduction and amplitude estimation issues in surface electromyography. *Journal of Electromyography and Kinesiology*, 12: 1-16.
- Colloca, C.T. and Keller, T.S. 2000. Mechanical force spinal manipulation increases trunk muscle strength assessed by electromyography: a comparative clinical trial. *Journal of Manipulative and Physiological Therapeutics*, 23(9): 585-595.
- Cordova, M.L. and Ingersoll, C.D. 2003. Peroneus longus stretch reflex amplitude increases after ankle brace application. *British Journal of Sports Medicine*, 37: 258-262.
- Courtney, C.A., Witte, P.O., Chmell, S.J. and Hornby, T.G. 2010. Heightened flexor withdrawal response in individuals with knee osteoarthritis is modulated by joint compression and joint mobilization. *The Journal of Pain*, 11(2): 179-185.
- Criswell, E. 2<sup>nd</sup> ed. 2010. *Cram's Introduction to Surface Electromyography*. Sudbury, Massachusetts: Jones and Bartlett Publishers.
- de Bie, R.A., Heemskerk, M.A.M.B., Lenssen, A.F., van Moorsel, S.R., Rondhuis, G., Stomp, D.J., Swinkels, R.A.H.M. and Hendriks, H.J.M. 2003. Clinical practice guidelines for physical therapy in patients with chronic ankle sprain (unpublished document).
- de Vries, J.S., Krips, R., Sierevelt, I.N., Blankevoort, L. and van Dijk, C.N. 2011. Interventions for treating chronic ankle instability. *Cochrane Database of Systematic reviews*, 8.
- Delahunt, E., McGrath, A., Doran, N. and Coughan, G.F. 2010. Effect of taping on actual and perceived dynamic postural stability in persons with chronic ankle instability. *Archives of Physical Medicine and Rehabilitation*, 91: 1383-1390.
- Delahunt, E., Monaghan, K. and Caulfield, B. 2006. Altered neuromuscular control and ankle joint kinematics during walking in subjects with functional instability of the ankle joint. *The American Journal of Sports Medicine*, 34(12): 1970-1976.

Denegar, C.R., Hertel, J. and Fonseca, J. 2002. The effect of lateral ankle sprain on dorsiflexion range of motion, posterior talar glide and joint laxity. *Journal of Orthopaedic and Sports Physical Therapy*, 32(4): 166-173.

Denegar, C.R. and Miller, S.J. 2002. Can chronic ankle instability be prevented? Rethinking management of lateral ankle sprains. *Journal of Athletic Training*, 37(4): 430-435.

Doguet, V. and Jubeau, M. 2014. Reliability of H-reflex in vastus lateralis and vastus medialis muscles during passive and active isometric conditions. *European Journal of Applied Physiology*, 114: 2509-2519.

Doherty, C., Delahunt, E., Caulfield, B., Hertel, J., Ryan, J. and Bleakley, C. 2013. The incidence and prevalence of ankle sprain injury: A systematic review and meta-analysis of prospective epidemiological studies. *Sports Medicine*, 44(1): 123-140.

Dougherty, P. 2014. Somatosensory systems (online). Available: <http://neuroscience.uth.tmc.edu/s2/chapter02.html> (Accessed 02 December 2015).

Dreyer, A.C., Dreyer, M.S., Hattingh, E. and Thandar, Y. 2012. *Pharmacology for Nurses and Other Health Workers*. 3<sup>rd</sup> edition. Pearson Education South Africa (Pty) Ltd.

Dunning, J. and Rushton, A. 2009. The effects of cervical high-velocity low-amplitude thrust manipulation on resting electromyographic activity of the biceps brachii muscle. *Manual Therapy*, 14: 508-513.

Eils, E., Demming, C., Killmeier, G., Thorwesten, L., Volker, K. and Rosenbaum, D. 2002. Comprehensive testing of 10 different ankle braces, Evaluation of passive and rapidly induced stability in subjects with chronic ankle instability. *Clinical Biomechanics*, 17: 526-535.

Evans, D.W. 2002. Mechanisms of spinal high-velocity, low-amplitude thrust manipulation: Previous theories. *Journal of Manipulative and Physiological Therapeutics*, 25: 251-262.

Ferran, N.A. and Maffulli, N. 2006. Epidemiology of sprains of the lateral mortise complex. *Foot and Ankle Clinic North America*, 11: 659-662.



Fryer, A. and Pearce, A.J. 2012. The effect of lumbosacral manipulation on corticospinal and spinal reflex excitability on asymptomatic participants. *Journal of Manipulative and Physiological Therapeutics*, 35: 86-93.

Fryer, G.A., Mudge, J.M. and McLaughlin, P.A. 2002. The effect of talocrural joint manipulation on range of motion at the ankle. *Journal of Manipulative and Physiological Therapeutics*, 25: 384-390.

Gallagher, D., Visser, M., de Meersman, R.E., Sepulveda, D., Baumgartner, R.N., Pierson, R.N., Harris, T. and Heymsfield, S.B. 1997. Appendicular skeletal muscle mass: effects of age, gender and ethnicity. *Journal of Applied Physiology*, 83(1): 229-239.

Garland, S.J. and McComas, A.J. 1990. Reflex inhibition of human soleus muscle during fatigue. *Journal of Physiology*, 429: 17-27.

Gribble, P.A., Delahunt, E., Bleakley, C., Caulfield, B., Docherty, C., Fong, D., Hertel, J., Hiller, C., Kaminski, T., McKeon, P., Refshauge, K., Van der Wees, P., Vicenzino, B. and Wikstrom, E. 2013. Selection criteria for patients with chronic ankle instability in controlled research: a position statement of the International Ankle Consortium. *Journal of Orthopaedic and Sports Physical Therapy*, 43(8): 585-591.

Grindstaff, T.L., Beazell, J.R., Sauer, L.D., Magrum, E.M., Ingersoll, C.D. and Hertel, J. 2011. Immediate effects of a tibiofibular joint manipulation on lower extremity H-reflex measurements in individuals with chronic ankle instability. *Journal of Electromyography and Kinesiology*, 21: 652-658.

Grindstaff, T.L., Pietrosimone, B.G., Sauer, L.D., Kerrigan, D.C., Patrie, J.T., Hertel, J. And Ingersoll, C.D. 2014. Manual therapy directed at the knee or lumbopelvic region does not influence quadriceps spinal reflex excitability. *Manual Therapy*, 19: 299-305.

Haavik, H. and Murphy, B. 2012. The role of spinal manipulation in addressing disordered sensorimotor integration and altered motor control. *Journal of Electromyography and Kinesiology*, 22: 768-776.

Hairi, N.N., Cumming, R.G., Naganathan, V., Handelsman, D.J., Le Couteur, D.G., Creasey, H., Waite, L.M., Seibel, M.J. and Sambrook, P.N. 2010. Loss of muscle

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.

Harvey, M.P. and Descarreaux, M. 2013. Short term modulation of trunk neuromuscular responses following spinal manipulation: A control group study. *BMC Musculoskeletal Disorders*, 14: 92-99.

Hebert-Blouin, M.N., Tubbs, R.S., Carmichael, S.W. and Spinner, R.J. Hilton's law revisited. *Clinical Anatomy*, 27(4):548-555.

Hertel, J. 2002. Functional anatomy, pathomechanics and pathophysiology of lateral ankle instability. *Journal of Athletic Training*, 37(4): 364-375.

Hiller, C.E., Kilbreath, S.L. and Refshauge, K.M. 2011. Chronic ankle instability: Evolution of the model. *Journal of Athletic Training*, 46(2): 133-141.

Hoffman, M., Harter, R.A., Hayes, B.T., Wojtys, E.M. and Murtaugh, P. 2008. The Interrelationships among sex hormone concentrations, motorneuron excitability and anterior tibial displacement in women and men. *Journal of Athletic Training*, 43(4): 364-372.

Holmes, A. and Delahunt, E. 2009. Treatment of common deficits associated with chronic ankle instability. *Sports Medicine*, 39: 207-224.

Hopkins, J.T. and Ingersoll, C.D. 2000. Athrogenic muscle inhibition: A limiting factor in joint rehabilitation. *Journal of Sports Rehabilitation*, 9: 135-159.

Hopkins, J.T., Brown, T.N., Christensen, L. and Palmieri-Smith, R.M. 2009. Deficits in peroneal latency and electromechanical delay in patients with functional ankle instability. *Journal of Orthopaedic Research*, 27: 1541-1546.

Joseph, L.C., de Busser, N. and Brantingham, J.W. 2010. The comparative effect of muscle energy technique vs. manipulation for the treatment of chronic recurrent ankle sprains. *Journal of American Chiropractic Association*, 47(7): 8-22.

Kaminski, T.W. and Hartsell, H.D. 2002. Factors contributing to chronic ankle instability: A strength perspective. *Journal of Athletic Training*, 37(4): 394-405.

- Kaur, A.N., Sharma, A., Singh, A. and Singh, J. 2014. Manipulation versus mobilization: a systematic review. *Indian Journal of Physiotherapy and Occupational Therapy*, 8(1): 1-4.
- Kieran, J.A. 2014. *Barr's The Human Nervous System: An anatomical view point*. 10<sup>th</sup> ed. USA: Lippincott Williams and Wilkins.
- Kim, K.M., Ingersoll, C.D. and Hertel, J. 2015. Facilitation of Hoffmann reflexes of ankle muscles in prone but not standing positions by focal ankle-joint cooling. *Journal of Sport Rehabilitation*, 24: 130-139.
- Kirk, R.E. 2003. Experimental Design. In: *Handbook of Psychology*. Wiley Online Library, 23-45.
- Klykken, L.W., Pietrosimone, B.G., Kim, K.M., Ingersoll, C.D. and Hertel, J. 2011. Motor-neuron pool excitability of the lower leg muscles after acute lateral ankle sprain. *Journal of Athletic Training*, 46(3): 263-269.
- Knikou, M. 2008. The H-reflex as a probe: Pathways and pitfalls. *Journal of Neuroscience Methods*, 171: 1-12.
- Knikou, M. and Taglianetti, C. 2006. On the methods employed to record and measure the human soleus H-reflex. *Somatosensory and Motor Research*, 23(1/2): 55-62.
- Kohne, E., Jones, A., Korporaal, C., Price, J., Brantingham, J. and Globe, G. 2007. A prospective, single-blinded, randomised, controlled clinical trial of the effects of manipulation on proprioception and ankle dorsiflexion in chronic recurrent ankle sprains. *Journal of the American Chiropractic Association*, 44: 7-17.
- Konradsen, L. 2002. Factors contributing to chronic ankle instability: Kinesthesia and joint position sense. *Journal of Athletic Training*, 37(4): 381-385.
- Lalanne, K., Lafond, D. and Descarreaux, M. 2009. Modulation of the flexion-relaxation response by spinal manipulative therapy: A control group study. *Journal of Manipulative and Physiological Therapeutics*, 32: 203-209.
- Lawrence, D.J. 2001. Chiropractic manipulation for the foot: diversified chiropractic techniques. *Manual Therapy*, 6(2): 66-71.

Lee, A. and Lin, W.H. 2008. Twelve week biomechanical mortise platform system training on postural stability and mortise proprioception in participants with unilateral functional mortise instability. *Clinical Biomechanics*, 23: 1065-1072.

Lentell, G., Baas, B., Lopez, D., McGuire, L., Sarrels, M. and Snyder, P. 1995. The contributions of proprioceptive deficits, muscle function and anatomic laxity to functional instability of the ankle. *Journal of Orthopaedic and Sports Physical Therapy*, 21: 206-215.

Lindsey-Renton, C. 2005. The immediate effect of manipulation in chronic ankle instability in terms of objective clinical findings. M.Tech thesis: Chiropractic. Durban University of Technology.

Lopez-Rodriguez, S., de-las-Penas, C.F., Albuquerque-Sendin, F., Rodriguez-Blanco, C. and Palomeque-del-Cerro, L. 2007. Immediate effects of manipulation of the talocrural joint on stabilometry and baropodometry in patients with ankle sprains. *Journal of Manipulative and Physiological Therapeutics*, 30(3): 186-192.

Loudon, J.K., Reiman, M.P. and Sylvain, J. 2013. The efficacy of manual joint mobilisation/manipulation in treatment of lateral ankle sprains: a systematic review. *British Journal of Sports Medicine*, 10: 1-6.

Lubbe, D., Lakhani, E., Brantingham, J.W., Parkin-Smith, G.F., Cassa, T.K., Globe, G.A. and Korporaal, C. 2015. Manipulative therapy and rehabilitation for recurrent ankle sprain with functional instability: A short-term, assessor-blind, parallel-group randomized trial. *Journal of Manipulative and Physiological Therapeutics*, 38: 22-34.

Lynch, S.A. 2002. Assessment of the injured ankle in the athlete. *Journal of Athletic Training*, 34(4): 406-412.

Maduro de Camargo, V., Albuquerque-Sendin, F., Berzin, F., Cobos Stefanelli, V., Rodrigues de Souza, D.P. and Fernandez-de-las-Penas, C. 2010. Immediate effects on electromyographic activity and pressure pain thresholds after a cervical manipulation in mechanical neck pain: a randomized controlled trial. *Journal of Manipulative and Physiological Therapeutics*, 34: 211-220.

Maigne, J.Y. and Vautravers, P. 2003. Mechanisms of action of spinal manipulative therapy. *Joint Bone Spine*, 70(30): 336-341.

McBride, D. and Ramamurthy, C. 2006. Chronic ankle instability: management of chronic lateral ligamentous dysfunction and the varus tibiotalar joint. *Foot and Ankle Clinic North America*, 11: 607.

McKinley, M. and O'Loughlin, V.D. 2012. *Human Anatomy: Third Edition*. 3<sup>rd</sup> ed. New York: McGraw-Hill.

McVey, E.D., Palmieri, R.M., Docherty, C.L., Zinder, S.M. and Ingersoll, C.D. 2005. Arthrogenic muscle inhibition in the leg muscles of subjects exhibiting functional ankle instability. *Foot and Ankle International*, 26: 1055-1061.

Misiaszek, J.E. 2003. The H-reflex as a tool in neurophysiology: its limitations and uses in understanding nervous system function. *Muscle and Nerve*, 28: 144-160.

Moore, K.L., Dalley, A.F. and Agur, A.M.R. 2010. *Clinically Orientated Anatomy*. 6<sup>th</sup> ed. USA: Lippincott Williams and Wilkins.

Murray, S.M. 2009. The immediate effects of thoraco-lumbar spinal manipulation compared to lower lumbar spinal manipulation on core muscle endurance and activity in patients with mechanical low back pain. M.Tech thesis: Chiropractic. Durban University of Technology.

Neogi, T.N. and Zhang, Y. 2013. Epidemiology of OA. *Rheumatic Disease Clinics of North America*, 39(1): 1-19.

Niazi, K.I., Turker, K.S., Flavel, S., Kinget, M., Duehr, J. and Haavik, H. 2015. Changes in H-reflex and V-waves following spinal manipulation. *Exp Brain Res*, 233: 1165-1173.

Nordander, C., Willner, J., Hansson, G.A., Larsson, B., Unge, J., Granquist, L. and Skerfving, S. 2003. Influence of the subcutaneous fat layer, as measured by ultrasound, skinfold calipers and BMI, on the EMG amplitude. *European Journal of Applied Physiology*, 89: 514-519.

Noska, K. 2006. The immediate and short term effect of spinal manipulative therapy on the lower leg musculature in lateral ankle sprain measured by surface electromyography during maximum voluntary contraction. M.Tech Chiropractic. University of Johannesburg.

Ong, C.K.S., Lirk, P., Tan, C.H. and Seymour, R.A. 2007. An evidence-based update on nonsteroidal anti-inflammatory drugs. *Clinical Medicine and Research*, 5(1): 19-34.

Osborne, M.D. and Rizzo Jr, T.D. 2003. Prevention and treatment of ankle sprains in athletes. *Sports Medicine*, 33: 1145-1150.

Paillard, T. 2012. Effects of general and local muscle fatigue on postural control: A review. *Neuroscience and Biobehavioral Reviews*, 36: 162-176.

Palmieri, R.M., Ingersoll, C.D. and Hoffman, M.A. 2004. The Hoffmann Reflex: Methodologic considerations and applications for the use in sports medicine and athletic training research. *Journal of Athletic Training*, 39(3): 268-277.

Palmieri-Smith, R.M., Hopkins, J.T. and Brown, T.N. 2009. Peroneal activation deficits in persons with functional ankle instability. *American Journal of Sports Medicine*, 37: 982-988.

Papadopoulos, E.S., Nicolopoulos, C., Anderson, E.G., Curran, M. and Athanasopoulos, S. 2005. The role of ankle bracing in injury prevention, athletic performance and neuromuscular control: a review of the literature. *The Foot*, 15: 1-6.

Pellow, J.E. and Brantingham, J.W. 2001. The efficacy of adjusting the mortise in the treatment of subacute and chronic Grade I and Grade II ankle inversion sprains. *Journal of Manipulation and Physiological Therapeutics*, 24: 17-24.

Pickar, J.G. 2002. Neurophysiological effects of spinal manipulation. *The Spine Journal*, 2: 357-371.

Pickar, J.G. and Bolton, P.S. 2012. Spinal manipulative therapy and somatosensory activation. *Journal of Electromyography and Kinesiology*, 22(5): 785-794.

Poul, J., Buchannan, N. and Graham, R. 1993. Local action transcutaneous flubiprofen in the treatment of soft tissue rheumatism. *British Journal of Rheumatology*, 32: 1000-1003.

Pourkazemi, F., Hiller, C.E, Raymond, J., Nightingale, E.J. and Refshauge, M.K. 2014. Predictors of chronic ankle instability after an index lateral ankle sprain: A systematic review. *Journal of Science and Medicine in Sport*, 17: 568-573.

Querry, R.G., Pacheco, F., Annaswamy, T., Goetz, L., Winchester, P.K. and Tansey, K.E. 2008. Synchronous stimulation and monitoring of soleus H reflex during robotic body weight-supported ambulation in subjects with spinal cord injury. *Journal of Rehabilitation Research and Development*, 45(1): 175-186.

Quinn, E. 2014. *What is normal range of motion in a joint?* (online). Available: <http://sportsmedicine.about.com/od/glossary/g/Normal-ROM.htm> (Accessed 02 December 2015).

Racinais, S., Maffiuletti, N.A. and Girard, O. 2013. M-wave, H- and V-reflex recruitment curves during maximal voluntary contraction. *Journal of Clinical Neurophysiology*, 30(4): 415-421.

Reid, C.D. 1992. *Sports Injury Assessment and Rehabilitation*. USA: Churchill Livingstone Inc.

Rice, D.A. and McNair, P.J. 2010. Quadriceps arthrogenic muscle inhibition: neural mechanisms and treatment perspectives. *Seminars in Arthritis and Rheumatism*, 40(3): 250-266.

Rice, D.A., McNair, P.J., Lewis, G.N. and Dalbeth, N. 2014. Quadriceps arthrogenic muscle inhibition: the effects of experimental knee joint effusion on motor cortex excitability. *Arthritis Research and Therapy*, 16: 502.

Riemann, B.L. 2002. Is there a link between chronic ankle instability and postural instability? *Journal of Athletic Training*, 37(4): 386-393.

Ritter, N.W. 2014. The effects of lumbar spine manipulation on flexion-relaxation response in chronic low-back pain participants. Master of Osteopathy, Unitech Institute of Technology.

Sefton, J.M., Hicks-Little, C.A. and Hubbard, T.J. 2008. Segmental spinal reflex adaptations associated with chronic ankle instability. *Archives of Physical Medicine and Rehabilitation*, 89: 1991-1995.

Shanks, P., Curran, M., Fletcher, P. and Thompson, R. 2010. The effectiveness of therapeutic ultrasound for musculoskeletal conditions of the lower limb: a literature review. *The Foot*, 20(4): 133-139.

Snell, R.S. 2010. *Clinical Neuroanatomy*. 7<sup>th</sup> ed. USA: Lippincott Williams and Wilkins.

Sousa, A.S. and Tavares, J.M.R. 2012. Surface electromyographic amplitude normalization methods: a review. In: Takada, H. ed. *Electromyography: New Developments, Procedures and Applications*. New York: Nova Science Publishers, Inc, 85-101.

Squires, N.A. Jeng, C.L. 2006. Posterior tibial tendon dysfunction. *Operative Techniques in Orthopaedics*, 16: 44-52.

Suter, E. and McMorland, G. 2002. Decrease in elbow flexor inhibition after cervical spine manipulation in patients with chronic neck pain. *Clinical Biomechanics*, 17: 541-544.

Tortora, G.J. and Derrickson, B.H. 2009. *Principles of Anatomy and Physiology*. 12<sup>th</sup> ed. Asia: John Wiley and Sons.

Tucker, K.J., Tuncer, M. and Turker, K.S. 2005. A review of the H-reflex and M-wave in the human triceps surae. *Human Movement Science*, 24: 667-688.

Tucker, K.J. and Turker, K.S. 2004. Muscle spindle feedback differs between the soleus and gastrocnemius in humans. *Somatosensory and Motor Research*, 21: 189-197.

Turner, W. and Merriman, L.M. 2005. *Clinical Skills in Treating the Foot*. 2<sup>nd</sup> ed. Edinburgh: Elsevier, Churchill and Livingstone.

Vaes, P., Duquet, W. and Van Gheluwe, B. 2002. Peroneal reaction times and eversion motor response in healthy and unstable ankles. *Journal of Athletic Training*, 37(4): 475-480.

Verhagen, E.A.L.M. and Bay, K. 2010. Optimising ankle sprain prevention: a critical review and practical appraisal of the literature. *British Journal of Sports Medicine*, 44: 1082-1088.

Vernon, H., MacAdam, K., Marshall, V., Pion, M. and Sadowska, M. 2005. Validation of a sham manipulative procedure for the cervical spine for use in clinical trials. *Journal of Manipulative and Physiological Therapeutics*, 28: 662-666.



Vizniak, N.A. 2010. *Quick Reference Evidence Based Muscle Manual*. Canada: Professional Health Systems Inc.

Vizniak, N.A. and Carnes, M.A. 2004. *Quick Reference Clinical Chiropractic Manual*. Canada: DC Publishing International.

Waterman, B.R., Owens, B.D., Davey, S., Zacchilli, M.A. and Belmont, P.J. 2010. The epidemiology of ankle sprains in the United States. *The Journal of Bone and Joint Surgery*, 92(13): 2279-2284.

Webster, K.A. and Gribble, P.A. 2010. Functional rehabilitation interventions for chronic ankle instability: a systematic review. *Journal of Sport Rehabilitation*, 19: 98-114.

Whitman, J.M., Cleland, J.A., Mintken, P., Keirns, M., Albin, S.R., Magel, J. and McPoil, T.G. Predicting short-term response to thrust and nonthrust manipulation and exercise in patients post inversion ankle sprain. *Journal of Orthopaedic and Sports Physical Therapy*, 39(3): 188-200.

Zammit, E. and Herrington, L. 2005. Ultrasound therapy in the management of acute lateral ligament sprains of the ankle joint. *Physical Therapy in Sport*, 6:116-121.

Ziltener, J.L, Leal, S. And Fournier, P.E. 2010. Non-steroidal anti-inflammatory drugs for athletes: an update. *Annals and Physical and Rehabilitation Medicine*, 53: 278-288.

# 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).

Muscle	Origin	Insertion	Innervation	Action
Tibialis anterior	Lateral condyle and superior half of lateral surface of tibia and interosseous membrane	Medial and inferior surfaces of medial cuneiform and base of first metatarsal	Deep fibular nerve (L4, L5)	Dorsiflexion and inversion of foot and ankle
Extensor digitorum longus	Lateral condyle of tibia and superior three quarters of medial surface of fibula and interosseous membrane	Middle and distal phalanges of lateral four digits	Deep fibular nerve (L4, L5)	Extends lateral four digits and dorsiflexes ankle
Extensor hallucis longus	Middle part of anterior surface of fibula and interosseous membrane	Dorsal aspect of base of distal phalanx of great toe	Deep fibular nerve (L4, L5)	Extends great toe and dorsiflexes ankle
Gastrocnemius	Lateral head: lateral aspect of lateral condyle of femur. Medial head: popliteal surface of femur; superior to medial condyle.	Posterior surface of calcaneus via calcaneal tendon	Tibial nerve (S1, S2)	Plantarflexes ankle with knee extended, raises heel during walking and flexes leg at knee
Tibialis posterior	Interosseous membrane; posterior surface of tibia inferior to soleal line and posterior surface of fibula	Tuberosity of navicular, cuneiform, cuboid and sustentaculum tali of calcaneus and; bases of second,	Tibial nerve (L4, L5)	Plantarflexes ankle and inverts foot

		third and fourth metatarsals		
Flexor hallucis longus	Inferior two thirds of posterior surface of fibula and inferior part of interosseous membrane	Base of distal phalanx of great toe	Tibial nerve (S2, S3)	Flexes great toe, weakly plantarflexes ankle and supports medial longitudinal arch of foot
Flexor digitorum longus	Medial posterior surface of tibia inferior to soleal line and by a broad tendon to fibula	Bases of distal phalanges of lateral four digits	Tibial nerve (S2, S3)	Flexes lateral four digits, plantarflexes ankle and supports longitudinal arches of foot
Plantaris	Inferior end of lateral supracondylar line of femur and oblique popliteal ligament	Posterior surface of calcaneus via calcaneal tendon	Tibial nerve (S1, S2)	Weakly assists in plantarflexing the ankle

## 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 :

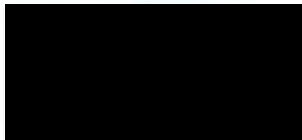
Mr Jason Travis Dicks (Student Number: 21011530)

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

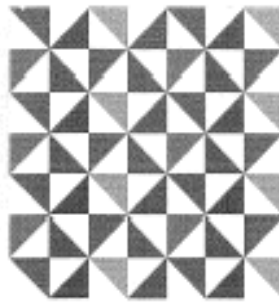


Dr Charmaine Korporaal

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



Institutional Research Ethics Committee  
Faculty of Health Sciences  
Room MS 49, Marfield School Site  
Gate 8, Ritson Campus  
Durban University of Technology

P O Box 1334, Durban, South Africa, 4001

Tel: 031 373 1900

Fax: 031 373 2407

Email: [lvishad@dut.ac.za](mailto:lvishad@dut.ac.za)

[http://www.dut.ac.za/research/institutional\\_research\\_ethics](http://www.dut.ac.za/research/institutional_research_ethics)

[www.dut.ac.za](http://www.dut.ac.za)

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 [SOP's] 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 N Sibiyi  
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.....

## Appendix F: Letter of information



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](mailto:lauraw@dut.ac.za) or co-supervisor Prof. Puckree on [puckreet@dut.ac.za](mailto:puckreet@dut.ac.za). Alternatively you



can contact me on [jason.t.dicks@gmail.com](mailto: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](mailto: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.....



**Student's Case History:**

1. **Source of History:**
2. **Chief Complaint: (patient's own words):**
3. **Present Illness:**

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

4. **Other Complaints:**
5. **Past Medical History:**
  - General Health Status
  - Childhood Illnesses
  - Adult Illnesses
  - Psychiatric Illnesses
  - Accidents/Injuries
  - Surgery
  - Hospitalizations

**6. Current health status and life-style:**

Allergies

Immunizations

Screening Tests incl. x-rays

Environmental Hazards (Home, School, Work)

Exercise and Leisure

Sleep Patterns

Diet

Current Medication

Analgesics/week:

Other (please list):

Tobacco

Alcohol

Social Drugs

**7. Immediate Family Medical History:**

Age of all family members

Health of all family members

Cause of Death of any family members

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

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



**CHIROPRACTIC PROGRAMME**

**PHYSICAL EXAMINATION:  
SENIOR**

<b>Patient Name:</b> _____		<b>File no:</b> _____		<b>Date:</b> _____	
<b>Student:</b> _____			<b>Signature:</b> _____		
<b>VITALS:</b>					
<b>Pulse rate:</b>				<b>Respiratory rate:</b>	
<b>Blood pressure:</b>		R	L	<b>Medication if hypertensive:</b>	
<b>Temperature:</b>				<b>Height:</b>	
<b>Weight:</b>	<i>Any recent change?</i>	Y / N	<i>If Yes: How much gain/loss</i>		<i>Over what period</i>
<b>GENERAL EXAMINATION:</b>					
<b>General Impression</b>					
<b>Skin</b>					
<b>Jaundice</b>					
<b>Pallor</b>					
<b>Clubbing</b>					
<b>Cyanosis (Central/Peripheral)</b>					
<b>Oedema</b>					
<b>Lymph nodes</b>	<b>Head and neck</b>				
	<b>Axillary</b>				
	<b>Epitrochlear</b>				
	<b>Inguinal</b>				
<b>Pulses</b>					
<b>Urinalysis</b>					
<b>SYSTEM SPECIFIC EXAMINATION:</b>					
<b>CARDIOVASCULAR EXAMINATION</b>					
<b>RESPIRATORY EXAMINATION</b>					
<b>ABDOMINAL EXAMINATION</b>					
<b>NEUROLOGICAL EXAMINATION</b>					
<b>COMMENTS</b>					
<b>Clinician:</b> _____			<b>Signature:</b> _____		

# Appendix J: Foot and ankle regional examination form



CHIROPRACTIC PROGRAMME

FOOT AND ANKLE  
REGIONAL EXAMINATION

Patient: \_\_\_\_\_ File no: \_\_\_\_\_ Date: \_\_\_\_\_

Student: \_\_\_\_\_ Signature: \_\_\_\_\_

Clinician: \_\_\_\_\_ Signature: \_\_\_\_\_

**Observation**

Gait analysis (antalgic limp, toe off, arch, foot alignment, tibial alignment).

\_\_\_\_\_

Swelling \_\_\_\_\_

Heloma dura / molle \_\_\_\_\_

Skin \_\_\_\_\_

Nails \_\_\_\_\_

Shoes \_\_\_\_\_

Contours (Achilles tendon, bony prominences) \_\_\_\_\_

**Active movements**

<b>Weight bearing:</b>	<b>R</b>	<b>L</b>	<b>Non weight bearing:</b>	<b>R</b>	<b>L</b>
Plantar flexion			50°		
Dorsiflexion			20°		
Supination					
Pronation					
Toe dorsiflexion			40°(mtp)		
Toe plantar flexion			40° (mtp)		
			Big toe dorsiflexion (mtp) (65-70°)		
			Big toe plantar flexion (mtp) 45°		
			Toe abduction + adduction		
			5° first ray dorsiflexion		
			5° first ray plantar flexion		

<b>Passive movement motion palpation</b> (Passive ROM quality, ROM overpressure, joint play)	<b>R</b>	<b>L</b>		<b>R</b>	<b>L</b>
Ankle joint: <i>Plantarflexion</i>			Subtalar joint: <i>Varus</i>		
<i>Dorsiflexion</i>			<i>Valgus</i>		
Talocrural: <i>Long axis distraction</i>			Midtarsal: <i>A-P glide</i>		
First ray: <i>Dorsiflexion</i>			<i>P-A glide</i>		
<i>Plantarflexion</i>			<i>rotation</i>		
Circumduction of forefoot on fixed rearfoot			Intermetatarsal glide		
			Tarso metatarsal joints: <i>A-P</i>		
Interphalangeal joints: <i>L-A dist</i>			Metatarsophalangeal		
<i>A-P glide</i>			dorsiflexion (with associated		
<i>lat and med glide</i>			plantar flexion of each toe		
<i>rotation</i>					



**Resisted Isometric movements**

	R	L		R	L
Knee flexion			Pronation (eversion)		
Plantar flexion			Toe extension (dorsiflexion)		
Dorsiflexion			Toe flexion (plantar flexion)		
Supination (inversion)					

**Neurological**

	R	L
Dermatomes		
Myotomes		
Reflexes		
Balance/proprioception		

**Special tests**

	R	L
Anterior drawer test		
Talar tilt		
Thompson test		
Homan sign		
Tinel's sign		
Test for rigid/flexible flatfoot		
Kleiger test (med. deltoid)		

**Alignment**

	R	L
Heel to ground		
Feiss line		
Tibial torsion		
Heel to leg (subtalar neutral)		
Subtalar neutral position:		
Forefoot to heel (subtalar & Midtarsal neutral)		
First ray alignment		
Digital deformities		
Digital deformity flexible		

**Palpation**

	R	L
<i>Anteriorly</i>		
Medial malleoli		
Med tarsal bones, tibial (post) artery		
Lat. malleolus, calcaneus, sinus tarsi, and cuboid bones		
Inferior tib/fib joint, tibia, mm of leg		
Anterior tibia, neck of talus, dorsalis pedis artery		
<i>Posteriorly</i>		
Calcaneus, Achilles tendon, Musculotendinous junction		
<i>Plantarily</i>		
Plantar muscles and fascia		
Sesamoids		

## Appendix K: SOAPE note form



**DEPARTMENT OF  
CHIROPRACTIC  
AND SOMATOLOGY**

**CHIROPRACTIC PROGRAMME**

Appendix F - SOAPE

<b>Patient Name:</b>		<b>File number:</b>		<b>Page:</b>
<b>Date:</b>	<b>Visit:</b>	<b>Student:</b>	<b>Signature:</b>	
<b>Attending Clinician:</b>				
<b>S:</b>	<b>Numerical Pain Rating Scale (Patient)</b> Least <b>0 1 2 3 4 5 6 7 8 9 10</b> Worst	<b>Student Rating</b> <input type="checkbox"/>	<b>A:</b>	
<b>O:</b>			<b>P:</b>	
			<b>E:</b>	
<b>Special attention to:</b>		<b>Next appointment:</b>		
<b>Date:</b>	<b>Visit:</b>	<b>Student:</b>	<b>Signature:</b>	
<b>Attending Clinician:</b>				
<b>S:</b>	<b>Numerical Pain Rating Scale (Patient)</b> Least <b>0 1 2 3 4 5 6 7 8 9 10</b> Worst	<b>Student Rating</b> <input type="checkbox"/>	<b>A:</b>	
<b>O:</b>			<b>P:</b>	
			<b>E:</b>	
<b>Special attention to:</b>		<b>Next appointment:</b>		
<b>Date:</b>	<b>Visit:</b>	<b>Student:</b>	<b>Signature:</b>	
<b>Attending Clinician:</b>				
<b>S:</b>	<b>Numerical Pain Rating Scale (Patient)</b> Least <b>0 1 2 3 4 5 6 7 8 9 10</b> Worst	<b>Student Rating</b> <input type="checkbox"/>	<b>A:</b>	
<b>O:</b>			<b>P:</b>	
			<b>E:</b>	
<b>Special attention to:</b>		<b>Next appointment:</b>		