The relative effect of upper cervical spine manipulation, placebo and a control, on neck muscle activity, pain and disability in participants with cervicogenic headache

By

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

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DEDICATION

To my Father in Heaven, Who gave me the strength and endurance to complete this dissertation, all the glory and honour goes to you.

But those who hope in the Lord

will renew their strength.

They will soar on wings like eagles;

they will run and not grow weary,

they will walk and not be faint.

Isaiah 40:31

To my parents, who have supported me through the last six years, financially and emotionally, I could not have completed my degree without you. Thank you for instilling in me the values you have and shaping my character. I will always endeavour to make you proud. I love you both endlessly.

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ABSTRACT

Objectives: Cervical spinal manipulation (CSM) has been shown to be effective in treating cervicogenic headache (CGH) by reducing pain and disability. There is a paucity of research investigating whether changes in muscle activity account for the changes in patient reported outcomes in patients with CGH following CSM. The aim of this study was to determine the effect of upper CSM compared to a placebo and a control intervention in terms of subjective (pain and disability rating) and objective measures (muscle activity and force output of the trapezius, posterior cervical and sternocleidomastoid muscles) in the treatment of CGH.

Methods: A randomized, controlled, pre-test, post-test, experimental design allowed for 45 participants, aged 18-50, with CGH to be allocated to a CSM, placebo or control group. Pain and disability were measured before and telephonically 48 hours after the intervention. Muscle activity and force output of the trapezius, posterior cervical and sternocleidomastoid (SCM) muscles were measured before and immediately after the intervention. IBM SPSS was used to analyse the data with significance set at p=0.05. The subjective and objective measures were not normally distributed and this resulted in non-parametric statistical tests being utilised to analyse the data. Wilcoxon Signed Rank Tests were used to determine significance within groups and Independent-samples Kruskal-Wallis Tests and Pairwise multiple comparison tests were used to determine significance between groups.

Results: No significant differences were found between the three groups for age (p=0.460) and gender (p=0.566), with a marginally significant finding for race (p=0.046). Subjective measure assessment found only a significant decrease in pain (p=0.001) and disability (p=0.001) from pre- post-test within the CSM group. Intergroup analysis found that the CSM group experienced the greatest reduction in pain (p=0.001) and disability (p=0.001) when compared to the placebo and control groups. Clinically, only pain in the CSM group decreased significantly. Objective findings showed significant intragroup increases in muscle activity, found bilaterally in the SCM (p=0.017 SCMR) (p=0.012 SCML) and trapezius muscles (p=0.041 TrapR) (p=0.041 TrapL) in the CSM group and only in the trapezius muscle (p=0.031 TrapR) (p=0.027 TrapL) bilaterally in the placebo group. Force output only increased in the trapezius muscle on the left (p=0.027) in the placebo group and bilaterally in the control group (p=0.031 TrapR) (p=0.041 TrapL) There were no significant intergroup differences between the groups for muscle activity and force output in the trapezius, posterior cervicals and SCM.

Conclusion: CSM resulted in increased muscle activity in the SCM and trapezius muscles bilaterally from pre- to post-test and a decrease in reported pain and disability when compared to a control and a placebo group. Indicating that CSM reduces pain and disability in CGH sufferers yet the relationship that muscle activity has with these findings requires further investigation. Trends suggest that increased muscle activity in the SCM and trapezius muscles during a maximum voluntary contraction show greater muscle activation following CSM.

Key indexing terms: Cervical spinal manipulation, cervicogenic headache, force output, muscle activity, spinal manipulative therapy.

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LIST OF ABBREVIATIONS

C:	Control
CGH:	Cervicogenic headache
CI:	Confidence interval
cm:	Centimetres
CNS:	Central nervous system
CSM:	Cervical spinal manipulation
CT:	Combined therapy
DUT:	Durban University of Technology
EMG:	Electromyography
ET:	Exercise therapy
HVLA:	High-velocity low-amplitude
Kg:	Kilograms
L:	Left
LBP:	Low back pain
M:	Median
m:	Meters
mm:	Millimeters
mVs:	Microvolts
MA:	Muscle activity
MVC:	Maximum voluntary contraction
n:	Sample number
NP:	Neck pain
p:	Probability value of statistical significance
PC:	Posterior Cervical muscles
PS:	Paraspinal muscles
PPT:	Pressure pain threshold

PT:	Participants
Q1:	First quartile
Q2:	Median
Q3:	Third quartile
R:	Right
RCT:	Randomized Clinical Trial
RMS:	Root mean square
ROM:	Range of motion
SCM:	Sternocleidomastoid muscles
±SD:	Standard deviation
±SE:	Standard error
sEMG:	Surface electromyography
SMT:	Spinal manipulative therapy
SO:	Suboccipital
Trap:	Trapezius

LIST OF DEFINITIONS

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

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

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

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

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

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

Muscle inhibition: The inability to fully activate a muscle due to on-going reflex inhibition (Venes 2017).

Headache: Pain located above an imaginary line drawn from the outer or lateral part of the orbit of the eye to the centre of the external auditory meatus of the ear (Olesen 2018).

Cervicogenic headache (CGH): Headache of cervical origin due to dysfunction of the cervical spine and its associated disc and soft tissue structures (Page 2011; Society 2013).

Placebo: Positive physiological or psychological changes associated with the use of inert medications, sham procedures, or therapeutic symbols within a healthcare encounter (Miller *et al.* 2005).

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

Motor unit:	The cell body and dendrites of a motor neuron, the multiple branches of its axon, and the muscle fibre that it innervates (Konrad 2005).
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).
Nociceptive:	Stimulus-response process involving the stimulation of pain sensitive nociceptors and the transmission of impulses along the peripheral nerves to the CNS (Venes 2017).

CHAPTER ONE

INTRODUCTION

1.1 INTRODUCTION

A headache (HA) is defined as pain located above an imaginary line drawn from the outer or lateral part of the orbit of the eye to the centre of the external auditory meatus of the ear (International Headache Society 2018; Olesen 2018). More than half of the global population will experience headaches during their lifetime (Haas *et al.* 2018). Headaches are broadly classified as being either "primary" or "secondary". Primary headaches are benign, recurrent headaches not caused by any underlying disease or structural problems, for example migraines (De Luca and Bartleson 2010). Secondary headaches result from a pathological process, which can be traumatic, infective or vascular in nature (Page 2011).

A cervicogenic headache (CGH) is a secondary headache of cervical origin occurring due to a dysfunction of the cervical spine (i.e. tenderness to palpation, restricted intersegmental range of motion, palpable asymmetric intervertebral muscle tension and abnormal joint play) and its associated disc and soft tissue structures (Page 2011; International Headache Society 2018). This type of headache is mainly unilateral in presentation and is often experienced following whiplash trauma (Olivier, Pramod and Maleka 2018). The clinical features associated with this headache are: dysfunctional cervical facet joints, myofascial trigger points located in the sternocleidomastoid, trapezius and posterior cervical muscles and precipitation of the headache by neck movement (Haldeman and Dagenais 2001; Miguel 2017).

The global prevalence of CGH varies between 0.4 and 4.6% (Haas *et al.* 2018). In chronic headache sufferers, the prevalence can be as high as 18%. CGH has been shown to be as debilitating, as common headache types like migraines and tension-type headaches (Hanson *et al.* 2016). Internationally, the mean age of CGH sufferers' is 42.9 years old, whereas in South Africa it has been reported to be 36.45 years old, and there is a 4:1 female predominance both in South Africa and abroad (Haldeman and Dagenais 2001; Olivier, Pramod and Maleka 2018).

The exact aetiology of CGH is not fully understood. CGH is hypothesized to occur from the convergence of the afferent input of the upper cervical spine nerve roots (C1-C3), with the afferent tracts of the trigeminal nerve in the trigemino-cervical nucleus (Fleming,

Forsythe and Cook 2007; Bogduk 2014). The first three cervical spinal nerves and their rami are the primary peripheral nerve structures that can refer pain to the head (Biondi 2005). Therefore, cervical facet joints, intervertebral discs and neck musculature (sternocleidomastoid, trapezius and posterior cervicals), innervated by the first three cervical nerves, may give rise to CGH when placed under excessive stress and strain (Becker 2010). Chronic muscular tension, acute whiplash injury, intervertebral disc disease and progressive facet joint arthritis are common pathologies which may produce a CGH. Tension type and migraine headaches are the two main differential diagnoses for CGH, due to the similarities in symptoms and clinical features found in these two primary headaches (Haldeman and Dagenais 2001; Page 2011).

Cervical spinal manipulation (CSM) is one of the main treatment methods recommended for CGH as stated in several guidelines (Bryans *et al*. 2011; Côté *et al.* 2019). Multiple studies have found that cervical spine manipulation is an effective treatment method for CGH, although the exact mechanism through which manipulation decreases the pain and disability associated with CGH is yet to be determined (Haldeman and Dagenais 2001; Biondi 2005; Becker 2010; Chaibi and Russell 2012). Spinal manipulation is postulated to produce biochemical changes which alter the transmission of sensory stimuli to the central nervous system, thereby effecting physiological mechanisms (Pickar 2002; Gyer 2019). The neurophysiological effects of spinal manipulation are thought to result in a change in motor control and muscle activity and a reduction in pain perception (Evans 2002; Gyer 2019). The above effects are attributed to spinal manipulation activating the serotonin and noradrenaline pathways of the descending pain modulation circuit of the central nervous system (Chaibi *et al.* 2015; Gyer 2019).

Evidence does exist to support the above neurophysiological mechanism; however, more research is needed to extend this body of evidence (Pickar 2002; Gyer 2019). Thus, this study aimed to assess the effect of upper cervical spinal manipulation on the muscle activity of the trapezius, posterior cervical and sternocleidomastoid muscles, to add to the existing framework regarding the neurophysiological effects of spinal manipulation and its effect on CGH.

1.2 STUDY AIMS, OBJECTIVES AND HYPOTHESES

1.2.1 Aim

The aim of this study was to determine the effect of upper CSM compared to a placebo and a control intervention, in terms of subjective measures (pain and disability ratings) and objective measures, such as muscle activity and force output of the trapezius, posterior cervical and sternocleidomastoid muscles in participants with CGH.

1.2.2 Study Objectives

Objective one:

To determine the pre- and post-effect of CSM in terms of subjective and objective measures in participants with CGH.

Objective two:

To determine the pre- and post-effect of a placebo intervention in terms of subjective and objective measures in participants with CGH.

Objective three:

To determine the pre- and post-effect of a control in terms of subjective and objective measures in participants with CGH.

Objective four:

To compare the effect of CSM, the placebo intervention and a control in terms of subjective and objective measures in participants with CGH.

1.2.3 Hypothesis

1.2.3.1 Null Hypotheses (Ho):

Objective 1:

Ho: There will be no statistically significant ($p < 0.05$) difference between the pre- and post- manipulation effects in terms of subjective and objective measures in participants with CGH.

Objective 2:

Ho: There will be no statistically significant ($p < 0.05$) difference between the pre- and post- placebo effect in terms of subjective and objective measures in participants with CGH.

Objective 3:

Ho: There will be no statistically significant ($p < 0.05$) difference between the pre- and post- control effect in terms of subjective and objective measures in participants with CGH.

Objective 4:

Ho: There will be no statistically significant ($p < 0.05$) difference between the three groups in terms of subjective and objective measurements in participants with CGH.

1.2.3.2 Alternate Hypothesis:

Objective 1:

Ha: There will be a statistically significant ($p < 0.05$) difference between the pre- and post-manipulation effect in terms of subjective and objective measures in participants with CGH.

Objective 2:

Ha: There will be a statistically significant ($p < 0.05$) difference between the pre- and post-placebo effect in terms of subjective and objective measures in participants with CGH.

Objective 3:

Ha: There will be a statistically significant ($p < 0.05$) difference between the pre- and post-control effect in terms of subjective and objective measures in participants with CGH.

Objective 4:

Ha: CSM will result in a statistically significant improvement ($p<0.05$) in CGH, in terms of the subjective and objective measures, when compared to the placebo and control interventions in participants with CGH.

1.3 DELIMITATIONS

This study was limited to people with CGH, between the ages of 18 - 50 years old, and recruitment was not limited by gender. Diagnoses were made by the researcher and the supervising clinician. People with contra-indications to spinal manipulation and surface electromyography (sEMG) were excluded.

The study focused on the immediate effect of CSM compared to a placebo and a control, thus only pre- and post-intervention measurements were taken. The measures to assess the effect were limited to pain rating, disability, force output and muscle activity. Muscle activity and force output readings were limited to the three main muscles associated with CGH - the sternocleidomastoid, trapezius and posterior cervical muscles.

1.4 SIGNIFICANCE OF THE STUDY

Half of the global population will experience headaches in their lifetime (Haas *et al.* 2018). CGHs, although not as common as migraine and tension type headaches, produce a significant amount of disability and can limit many activities of daily life (Becker 2010). Many CGH sufferers overuse or become dependent on analgesics, which in most instances do not provide adequate pain relief (Biondi 2005). Considering this, it is prudent that an alternative evidenced based treatment method, such as CSM, is sought (Haas *et al.* 2018).

The benefits of CSM for CGH sufferers are documented, yet further research is needed to clarify the recommended dosage of CSM for producing maximum clinical effect and the manner in which CSM brings about this effect (Chaibi *et al.* 2015; Hanson *et al.* 2016). This study aimed to add to the body of knowledge by investigating the neurophysiological effects of spinal manipulation which will assist manual therapists in selecting CSM as a treatment for this condition.

1.5 FLOW OF DISSERTATION

Chapter one has presented the introduction for the study, the study's aim and objectives, hypotheses, delimitations and flow of the dissertation.

Chapter two presents the literature review and provides an overview of the anatomy of the cervical spine, the diagnosis and management of CGH and a critical discussion on the literature related to the effect of manipulation on CGH, muscle activity, pain and disability.

Chapter three details the research methodology used in this study in order to accomplish the aims and objectives. The study's design, methods, techniques and instruments, as well as any ethical implications, are outlined and explained.

Chapter four presents the results of the data analysed in this study. The characteristics of the sample, together with the muscle activity, maximum voluntary contraction pain, disability and headache diary data are presented in the form of figures and tables.

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

Chapter six concludes the study and includes the study's limitations and recommendations.

CHAPTER TWO

LITERATURE REVIEW

2.1 INTRODUCTION

This chapter presents an overview of the literature related to this area of research. Initially, the cervical spine is discussed, followed by a discussion on the current literature on CGH, including epidemiology, pathophysiology, aetiology, associated signs and symptoms, diagnosis and treatment. Thereafter, the chapter outlines the literature related to spinal manipulation, in the context of CGH.

Cervical spine dysfunction may lead to altered afferent input to the central nervous system (CNS), resulting in plastic changes in the way the CNS responds to subsequent input (Haavik and Murphy 2012). Joint dysfunction is postulated to cause altered afferent input and can have an inhibitory effect on surrounding muscles. Over prolonged periods, this altered afferent input may lead to maladaptive neural plastic changes to the CNS. There is growing evidence in support of spinal manipulation normalizing aberrant muscle activity (Falla, Bilenkij and Jull 2004; Bicalho *et al*. 2010; Haavik and Murphy 2012). Multiple studies have found cervical spine manipulation (CSM) to be an effective treatment method for CGH (Haldeman and Dagenais 2001; Biondi 2005; Chaibi and Russell 2012; Haas *et al.* 2018). The mechanism through which spinal manipulation decreases the pain and disability associated with CGH, and affects muscle activity, requires further investigation in order to add to the increasing evidence in support of manipulation's neurophysiological effect (Evans 2002; Pickar 2002; Gyer 2019).

Literature for this study was gained from the following sources: Google Scholar, Summon, PubMed, ScienceDirect, eMedicine, ResearchGate and the Durban University of Technology Institutional Repository. Key terms used to investigate this literature included: cervicogenic headache, headache, spinal manipulation, cervical spine manipulation, neurophysiological effect, surface electromyography and headache pain/disability.

2.2 OVERVIEW OF THE CERVICAL SPINE

The cervical spine is defined as the area between the head and the thoracic spine. The cervical region of the vertebral column is made up of seven cervical vertebrae joined by intervertebral discs. The intervertebral discs protect the vertebrae and are efficient shock absorbers. The arrangement of these vertebrae results in a convex curvature or lordosis of the cervical spine. These seven cervical vertebrae are centrally placed in the vertebral column, supporting the weight of the head and neck and allowing for intervertebral articulations (Moore, Dalley and Agur 2014).

Intervertebral articulations are produced by the zygapophyseal, or facet joints, which are oblique, relatively horizontal and directed primarily superiorly and inferiorly. The transverse processes are located laterally on each vertebra end, in two projections, via the anterior tubercle and the posterior tubercle. Each cervical vertebra has a foramina transversarium, or transverse foramen, located on the transverse process. Vertebral arteries and their accompanying veins pass (with the exception of the C7, which only transmits small accessory veins) through this vertically-orientated canal. The triangular-shaped intervertebral canal passes through the center of the cervical vertebrae, which houses and protects the cervical segment of the spinal cord (Moore, Dalley and Agur 2014). The first (C1), second (C2) and seventh (C7) cervical vertebrae are atypical vertebrae, while third (C3), fourth (C4), fifth (C5) and sixth (C6) cervical vertebrae are typical vertebrae (Moore, Dalley and Agur 2014).

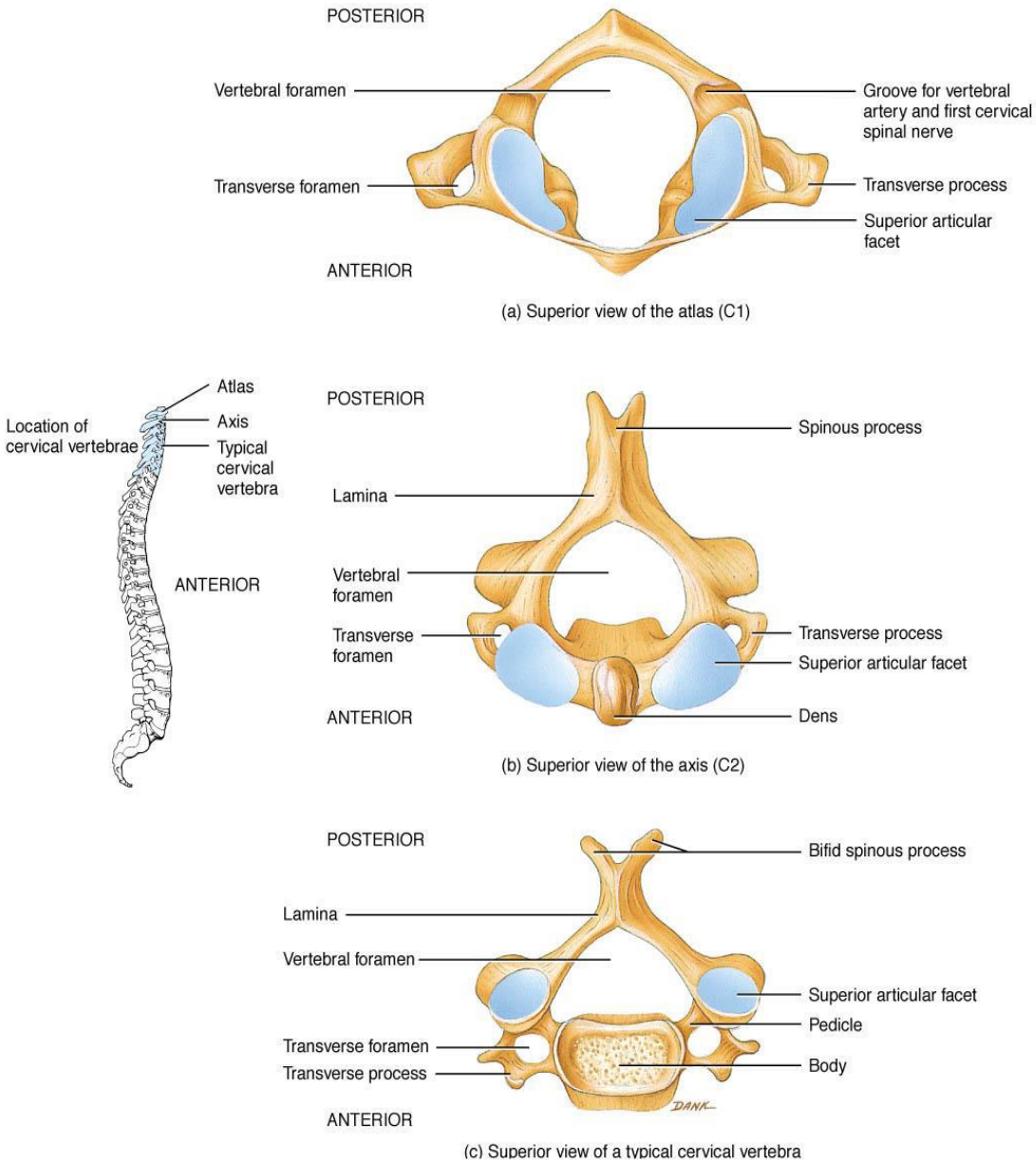


Figure 2.1: Structure of typical and atypical cervical vertebra (Moore, Dalley and Agur 2014)

2.3 JOINTS OF THE CERVICAL SPINE

The atlanto-occipital joint is formed by the articulating surface of the atlas and the occipital bone. The atlanto-axial joints are made up of three synovial joints: one centrally-located atlanto-odontoid joint and two lateral atlanto-axial joints. The atlanto-odontoid joint is a pivot joint comprising of the articulation between the zygapophyseal joint on the anterior surface of the odontoid process, with the reciprocally-shaped zygapophyseal joint on the posterior arch of the atlas. The two gliding lateral atlanto-axial joints are between the inferior zygapophyseal joints of the lateral masses of C1 and the superior zygapophyseal joints of C2 (Moore, Dalley and Agur 2014).

The cervical facet joints can be found bilaterally and are classified as synovial planar joints. Facet joints allow six directions of movement in the cervical spine. The orientation of the facet joints controls the direction and range of movement. Each facet joint is surrounded by an articular capsule postero-laterally. The capsules are longer in the cervical spine when compared to other spinal segments and this increased length allows for a greater degree of movement in the cervical spine. These joints are of interest clinically as the aberrant motion of these joints may irritate the nerves innervating the facet joint capsules, producing altered neck muscle activity and a cervicogenic headache (Becker 2010). Facet joint capsules have a rich supply of sensory innervation. Innervation occurs from the level superior to the joint and at the level of the joint.

Three different types of sensory receptors have been identified in the facet joint capsule (McLain and Raiszadeh 1995; Gatterman 2005; Candido and England 2015). These three types of mechanoreceptors ensure the cervical vertebrae are properly aligned:

- Type I - Extremely sensitive static and dynamic mechanoreceptors that fire continually, even when the joint is still.
- Type II - Less sensitive dynamic mechanoreceptors which do not fire when the joint is motionless.
- Type IV - Slow conducting nociceptive mechanoreceptors.

The uncovertebral joints are synovial joints lined by fibrocartilage and made up of the uncinate processes growing superiorly from the supero-lateral borders of C3 to C6 vertebral bodies, projecting into the outer aspect of the annulus fibrosis and corresponding with the reciprocally shaped cavities of the vertebra above.

Table 2. 1: Cervical joint motion (Moore, Dalley and Agur 2014)

Joint	Motion
Atlanto-occipital	Flexion, extension, slight lateral flexion and rotation of the head
Atlanto-axial	Rotation of the head
Facet	Flexion, extension, lateral flexion and rotation of the neck
Uncovertebral	Flexion and extension of the neck

2.4 CERVICAL SPINAL NERVES

Spinal nerves can be classified as peripheral nerves. They consist of parallel packages of axons with layers of connective tissue covering the neuroglial cells. Spinal nerve roots function primarily to join the central nervous system to afferent receptors, muscles and glands of the body (Tortora 2012). The first pair of cervical nerves exits the spinal cord between the occipital bone and the atlas. The residual cervical spinal nerves exit the spinal cord through the intervertebral foramina between contiguous vertebrae. Spinal nerves C1-C7 exit the vertebral canal above their corresponding vertebrae. Spinal nerve C8 exits the spinal canal between C7 and T1 (Tortora 2012). Structures innervated by the first three cervical nerves form the anatomical basis for the proposed pathophysiology of CGH (Fleming, Forsythe and Cook 2007; Becker 2010).

2.5 CERVICOGENIC HEADACHE

The term cervicogenic headache was first coined by Sjaastad *et al.* (1983) to describe a headache related to a dysfunction of the cervical spine and the associated soft tissue structures. According to the International Headache Society (2018), a cervicogenic headache is defined as a headache caused by a disorder of the cervical spine and its component bony, disc and/or soft tissue elements, usually but not invariably accompanied by neck pain. Pain is referred from the cervical spine and its component bony, disc and/or soft tissue elements to the face and head in a unilateral ram's horn distribution (Haldeman and Dagenais 2001; Chaibi and Russell 2012).

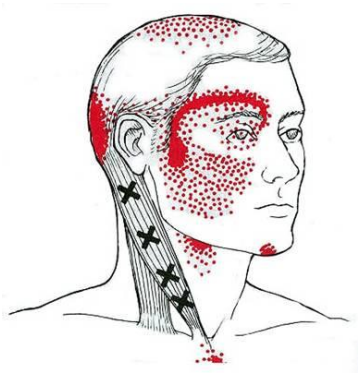


Figure 2.2: Ram's horn pain distribution of CGH with myofascial dysfunction in the SCM (Simmons *et al*. 1999)

2.5.1 Incidence and Prevalence of CGH

Global prevalence of CGH ranges between 0.4% and 4.6%. (Hanson *et al.* 2016; Haas *et al.* 2018). Half of the global population will experience headaches during their lifetime and 15-20% of these will be CGH (Page 2011; Hanson *et al.* 2016). These headaches are associated with a high burden of suffering, considerable socio-economic cost and decreased physical functioning (Biondi 2005; Fernández-de-las-Peñas, Arendt-Nielsen and Gerwin 2010).

2.5.2 Risk Factors of CGH

Common risk factors for CGH include whiplash, concussion, weightlifting, poor ergonomics, cervical osteoarthritis, female gender and changes in muscle length, such as Janda's Upper Crossed Syndrome (Page 2011). Continued neck contraction, as result of increased computer and visual display usage, has dramatically raised reports of neck discomfort and headache precipitation in the global population (Alison Middleditch and Jean Oliver 2005). A study conducted by Rifat and Moeller (2003) found weightlifting athletes have a high incidence of CGH. Other specific occupations which may predispose an individual to CGH include hair-dressing, truck driving and carpentry (Page 2011). Internationally, the mean age of CGH sufferers is 42.9 years old, whereas in South Africa it has been reported to be 36.45 years old, and there is a 4:1 female predominance both in South Africa and abroad (Haldeman and Dagenais 2001; Olivier, Pramod and Maleka 2018).

2.5.3 Associated Signs and Symptoms of CGH

Major signs and symptoms of a CGH according to Biondi (2005) include:

- Precipitation of head pain, similar to that usually occurring through neck movement and/or sustained awkward head positioning.
- Precipitation of head pain, similar to that usually occurring through external pressure over the upper cervical or occipital region on the symptomatic side.
- Restriction of the cervical range of motion.
- Ipsilateral neck, shoulder, or arm pain of a rather vague non-radicular nature or, occasionally, arm pain of a radicular nature.
- Moderate to severe, non-throbbing, and non-lancinating headache pain, normally starting in the neck.
- Headache duration varies with intermittent or continuous pain.

Minor attack-related phenomena, which are only occasionally present, include nausea, phonophobia, photophobia, dizziness, ipsilateral blurred vision, dysphagia and ipsilateral periocular oedema (Biondi 2005).

2.5.4 Aetiology and Pathophysiology of CGH

The exact aetiology of CGH is only currently hypothesized and it is, therefore, an area of controversy and debate. According to Anthony (2000), the trigeminal pathway theory is somewhat supported by the fact that injection of the greater and lesser occipital nerves with steroids decrease headaches by blocking the trigeminal relay. More recently, Chua *et al.* (2011) reported impairments of sensory testing of the head in CGH patients, compared to patients with neck dysfunction without headache. They proposed that the pathophysiology of CGH includes a central sensitization of pain, likely from the trigeminal spinal nucleus. Some controversy stems from the multitude of possible underlying causes for CGH and the fact that the hemi-cranial pain does not originate from the head but rather from any anatomical structure in the neck, innervated by the first three cervical nerves (Becker 2010)

CGH pathophysiology has an anatomical and neurophysiological basis. The neurophysiological basis for CGH is the convergence of the afferent input of the upper cervical spine nerve roots (C1-C3), with the afferent tracts of the trigeminal nerve in the trigeminocervical nucleus (Fleming, Forsythe and Cook 2007; Bogduk 2014). This convergence results in cervical spine nociceptive input being expressed in the sensory distribution of the trigeminal nerve, most commonly the ophthalmic branch of the trigeminal nerve, which innervates the forehead, temple, and orbit and has its greatest topographic representation near the dorsal horns of spinal nerves C1-C3 (Fleming, Forsythe and Cook 2007; Becker 2010; Bogduk 2014).

The first three cervical facet joints, atlanto-occipital and atlanto-axial joints, C2-3 intervertebral disc and sternocleidomastoid, trapezius and posterior cervical muscles, have a close proximity to one another and are all innervated by the first three cervical nerves. This forms the anatomical basis for CGH's proposed pathophysiology. The atlanto-occipital joint is innervated by the C1 spinal nerve. The atlanto-axial joint, the first three cervical facet joints, the sternocleidomastoid and the trapezius muscles are innervated by C2 and C3 spinal nerves. The posterior cervical muscles are innervated by C3 and the lower spinal nerves. Due to the above joints, muscles and nerves having a close proximity to one another, any trauma or pathological condition around these joints can be a source of referred head pain (Biondi 2005).

2.5.5 Muscle Activity and Force Output Changes in CGH

Hypertonicity of the SCM, posterior cervical and upper trapezius may occur as a result of arthrogenic muscle inhibition (AMI). An arthrogenic muscle response is a constant reflex reaction of the muscles adjacent to a joint after structural damage to the joint has occurred. Muscle inhibition or an increased potential for muscle activation (facilitation) then occurs (McVey *et al.* 2005). AMI can be caused by cervical facet dysfunction and may lead to altered neurophysiologic input into the facet joint mechanoreceptors and an impairment of cervical muscle-resting tone. Dysfunctional mechanoreceptors then send inhibitory input via afferent neurons to the spinal cord, which then synapses with inhibitory interneurons. Decreased activation in the motor neuron pool of the involved muscles adjacent to the damaged joint follows. A reduction in motor unit recruitment and the force of contraction of involved muscles then occurs (Rice and McNair 2010; Luedtke *et al.* 2016).

Weakness of the deep cervical flexors and compensatory hypertonicity of the SCM has been observed in individuals with neck pain and CGH (Falla, Bilenkij and Jull 2004; Jull and Falla 2016). Therefore, the AMI mechanism may be indicated in hypertonicity of the SCM, trapezius and posterior cervicals, as these muscles may be more frequently used to stabilize the cervical spine and counteract any weakness of inhibited muscles in individuals with CGH. Russo *et al.* (2018) observed that individuals with chronic low-back pain had arthrogenic inhibition of the multifidus muscle and a resultant compromise in lumbar spine stability as illustrated in **Figure 2.4.**

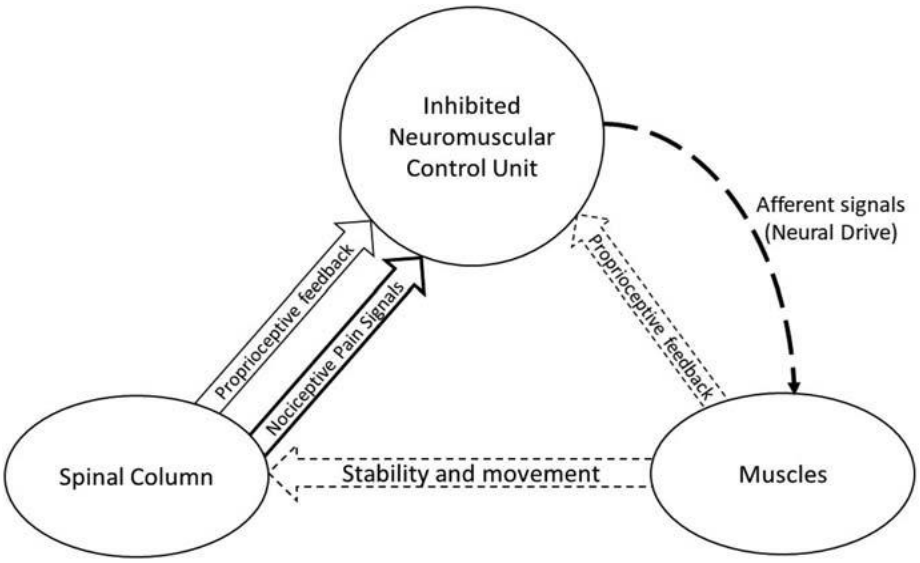


Figure 2.3: AMI mechanism affecting spinal stability (Russo *et al*. 2018)

2.5.6 Diagnosis

CGHs may be challenging to diagnose clinically due to the overlap of clinical features with other headaches, especially migraine and tension-type (Becker 2010). Currently, two major sets of diagnostic criteria exist for CGH. The two sets of criteria represent different approaches to diagnosing CGH. The first criterion originated in Europe and revolves around CGH's distinct clinical features (Sjaastad, Fredriksen and Pfaffenrath 1990). The second criterion originated from North America and Australia and involves using CGH clinical features and controlled diagnostic nerve blocks to establish headache pain of cervical origin (International Headache Society 2018).

2.5.6.1 Clinical Diagnosis

The main clinical features used to make a diagnosis of CGH include a restricted range of motion in the cervical spine; precipitation of headache by neck movement; sustained awkward head positioning or by external pressure on the upper cervical or occipital area on the symptomatic side; ipsilateral non-radicular neck, shoulder and arm pain and unilaterality of the headache pain, although a bilateral form of CGH does exist (Sjaastad, Fredriksen and Pfaffenrath 1990; Becker 2010). Other clinical features that can be used to differentiate CGH from primary headaches include restricted facet joints or a "facet lock" which may refer pain from the back of the neck, evidence of cervical dysfunction during manual examination and pain from myofascial tripper points in the neck (Page 2011).

2.5.6.2 Intervention Diagnosis

Practitioners use fluoroscopically-guided, controlled diagnostic nerve blocks to test whether particular structures are the source of pain in patients with suspected CGH (Bogduk and Govind 2009; International Headache Society 2018). The main structures that are tested are the atlanto-axial joint, C2-C3 facet joint and C3-C4 facet joint. While these blocks may provide complete relief of headache pain, and as a result produce objective evidence for a headache of cervical origin (Bogduk and Govind, 2009), they are invasive and impractical for most clinical settings (Fleming, Forsythe and Cook 2007).

2.5.7 Treatment

A CGH can be treated using pharmacology and, in severe cases, with surgery (Martelletti and van Suijlekom 2004). Cervical spine manipulation, exercise therapy and the use of modalities all fall under the general term of 'conservative' therapy. Conservative therapy has been shown to be more effective than pharmacological and surgical interventions due to many individuals not responding to medication and the high risks associated with cervical surgery (Page 2011; Chaibi and Russell 2012). A combination of cervical spine

manipulation and exercise therapy has been shown to yield superior clinical results (Racicki *et al.* 2013; Côté *et al.* 2019).

2.5.7.1 Exercise Therapy

A randomized controlled trial reported that a six-week regimen of low-load endurance exercises, such as postural correction and the cranio-cervical flexion exercise, to train muscle control of the cervico-scapular region, was effective in reducing headache intensity and frequency. Furthermore, when these exercises were combined with cervical spine manipulation, a longer therapeutic effect was maintained (Jull *et al.* 2002).

2.5.7.2 Modalities

There is a paucity of literature regarding the use of modalities in the treatment of CGH (Page 2011). Modalities like transcutaneous electrical nerve stimulation and cryotherapy are used to reduce pain and speed up the natural healing process. There are a few studies that show support for these modalities, in combination with other therapies, in the treatment of CGH (Haldeman and Dagenais 2001). Recently, there has been a rise in the popularity of low-level lasers in the treatment of musculo-skeletal conditions but no known study has examined lasers in the treatment of CGH (Page 2011).

2.6 CERVICAL SPINE MANIPULATION

Cervical spine manipulation involves a brief, high velocity, low amplitude impulse directed at a dysfunctional synovial facet joint. This mechanical event causes slight momentary deformations of the spine and surrounding soft tissues and carries the vertebrae beyond their normal physiological range of motion (ROM) without exceeding the boundaries of anatomic integrity. Vertebral rotations and facet joint capsule strain magnitudes during CSM have been shown to be within the ranges that occur during physiological movements (Bergmann and Peterson 2010; Millan *et al.* 2012).

2.6.1 Theoretical Models of Spinal Manipulation

It has been theorized that spinal manipulation initiates separation of articular surfaces, causing a stretch of the muscles adjacent to the joint being manipulated, resulting in an elongation of the concomitant muscle spindles. Stretching of the joint capsule itself also occurs, leading to mechanoreceptor activation and altered afferent stimuli. This, in turn, facilitates a change in motor neuron excitability. Motor neuron stimuli then travel in type I and type II afferent nerve fibres to the posterior horn of the spinal cord. A synapse between the interneuron and afferent neuron occurs, resulting in motor neuron stimulation

or inhibition. The motor neuron pool then increases or decreases in excitability when motor neuron stimuli reach the muscles adjacent to the joint being manipulated. Modulation of the inter-neuronal pools occurs through the dorsal root ganglia and substanstia gelatinosa, thereby eliminating pain transmission (Dhami and DeBoer 1992; Evans 2002; Pickar 2002; Haavik-Taylor and Murphy 2007; Pickar and Bolton 2012; Gyer 2019). There is an increasing body of evidence in support of the above neurophysiological theory (Haavik and Murphy 2012).

2.6.2 Clinical Research Investigating the Effects of Spinal Manipulation

The neurophysiological effects of spinal manipulation are only hypothesized at present and require further investigation (Nilsson 1995; Evans 2002; Pickar 2002; Leach 2004; Haavik-Taylor and Murphy 2007; Bergmann and Peterson 2010; Cardinale *et al.* 2015; Gyer 2019). Some of the reported neurophysiological effects of SMT are; modification of spinal reflex excitability and alteration of paraspinal cutaneous temperature, motor neuron excitability, muscle activity of paraspinal muscles and increase in muscle strength. Furthermore SMT may affect sensorimotor integration and pain regulation in the dorsal horn of the spinal cord and the periaqueductal grey area (Millan *et al.* 2012). It is still unclear on how these neurophysiological effects may influence biomechanical effects such as range of motion. While many studies have investigated the neurophysiological effects of spinal manipulation, there is a lack of consensus among researchers on whether manipulation increases or decreases muscle activity (Haavik and Murphy 2012), as shown in **Table 2.2**. However, there is advancing evidence in support of spinal manipulation normalizing aberrant muscle activity (Sterling, Jull and Wright 2001; Bicalho *et al.* 2010; Haavik and Murphy 2012; Gyer 2019).

Table 2. 2: Clinical studies investigating the neurophysiological effects of SMT

Author	Sample size and population	Study design	Intervention	Outcome measures	Results
Keller and Colloca (2000)	n=40 LBP participants	RCT	1. Activator SMT 2. Control	MA of PS during trunk MVC extensions	Significant increase in PS MA during MVC extension following manually assisted SMT
Lehman and McGill (2001a)	n=14 LBP participants	Analytic al cohort	1. L/S SMT 2.Control	PS and abdominal MA, and lumbo-pelvic kinematics	No significant changes following SMT. MA decreased in participants with higher levels of pain or more severe dysfunction

Suter and McMorland (2002b)	n =16 Chronic NP	RCT	1. CSM 2. Control	Active CROM, elbow flexor force, sEMG of elbow flexor and PPT	Significant decrease in muscle inhibition and increase in force out, CROM and PPT following SMT of C/S.
DeVocht, Pickar and Wilder (2005)	n =16 LBP participants	RCT	1. SMT 2. Activator SMT	MA of PS (resting	Significant decrease in MA. Initial increase in MA followed by decrease compared to pre-intervention levels.
Dunning and Rushton (2009)	n =54 Asymptomatic participants	RCT	1. CSM 2. Sham 3. Control	Resting EMG of biceps brachii muscle bilaterally	Increase in MA immediately after C/S SMT.
Lalanne, Lafond and Descarreaux (2009)	n= 27 adults with chronic LBP	EXP	1. L/S SMT 2. Control	MA of erector spinae	Significant reduction of MA at full trunk flexion at the L2 erector spinae level was observed in the L/S SMT group
Harvey and Descarreaux (2013)	n=60 LBP participants	RCT	1. L/S SMT 2. Control	MA, pain intensity, and lumbopelvic kinematics	Control group had increase in PS MA during task compared to SMT group.
Pires *et al.* (2015)	n =32 Women chronic NP	RCT	1.Upper T/S SMT 2.Control	MA of SCMs at rest and during isometric contractions for cervical flexion and elevation of the shoulder girdle. NP using visual analog scale.	No significant differences were found in the immediate intragroup or intergroup analyses of groups

(PPT=pressure pain threshold, PS=paraspinal muscles, MA=muscle activity, NP=neck pain, LBP=Low back pain, SMT=spinal manipulative therapy, CSM= cervical spinal manipulation, RCT=randomized clinical trial, sEMG=surface electromyography, EXP=experimental)

2.6.3 Cervical Spinal Manipulation in the Treatment of CGH

Upper cervical facet dysfunction (C1-C3) is the most likely cause of CGH and therefore many studies have focused on treating CGH using cervical manipulation/mobilization (Biondi 2005; Becker 2010; Chaibi and Russell 2012). According to Haavik-Taylor and Murphy (2007), cervical facet joint dysfunction refers to tenderness to palpation of the relevant joints, restricted intersegmental range of motion, palpable asymmetric intervertebral muscle tension and abnormal joint play. This correlates with Dewitte *et al.* (2014), who report that cervical facet joint dysfunction refers to spinal movement patterns that, when examined actively and passively, suggests a movement restriction that is local to one or two functional spinal units. Cervical manipulation may alter range of motion and neurophysiological function of the dysfunctional cervical facet joint, but the mechanism through which these clinical effects are attained requires further investigation (Nilsson, Christensen and Hartvigsen 1997; Haavik-Taylor and Murphy 2007; Haas *et al.* 2018). Several studies investigate the clinical effects of cervical manipulation/ mobilization on CGH; these are summarized in **Table 2.3**:

Table 2. 3: Clinical studies investigating the effect of cervical manipulation on CGH

Author	Sample size and population	Study design	Intervention	Outcome measures	Results
Haas *et al.* 2018	N=256	RCT	Participants were randomized to four dose levels of CSM: 0, 6, 12, or 18 sessions. They were treated three times per week for 6 weeks and received a focused light massage control in sessions when CSM was not assigned.	Headache frequency per month	Headache frequency reduced from 16 to 8 days for the highest and most effective dose of 18 CSM visits.
Yang *et al.* 2017	N=20 with CGH N=20 without CGH	EXP	Gr 1: craniocervical flexion Gr 2: suboccipital relaxation	Tone, stiffness and elasticity of the L and R SO and upper TRAP muscles in the experimental and control. Headache intensity in both groups.	Tone, stiffness and headache intensity decreased in the CGH group (p<0.01). No statistical significant difference was reported for elasticity between groups.
Dunning *et al.* 2016	N=110 with CGH	RCT	Gr 1: received both cervical and thoracic spinal manipulation-58 PT Gr 2: received mobilization of the cervical and thoracic spine and exercise therapy-52 PT	Headache intensity as measured by the Numeric Pain Rating Scale Headache disability as measured by the Neck	Participants who received both cervical and thoracic manipulation experienced significantly greater reductions in headache intensity ($p < 0.001$) and disability ($p < 0.001$)

				Disability Index	than those who received mobilization and exercise at a 3-month follow-up.
Jull *et al.* 2002	N=200	RCT	Gr 1: CSM Gr 2: Exercise Gr 3: Combined Gr 4: Control	Headache frequency	CSM and ET had reduced headache frequency after 6 weeks *(p*<0.05*)*
Nilsson, Christensen and Hartvigsen 1997	N=53 with CGH	RCT	Gr 1: CSM-28 PT Gr2: Deep massage and low-level laser-25 PT	Analgesic use per day. Headache hours per day. Headache intensity per episode.	Analgesic use per day reduced by 36% in Gr 1 compared to no change in Gr 2. Headache hours per day reduced by 69% in Gr 1 compared to 37% in Gr 2. Headache intensity per episode reduced by 36% in Gr 1 compared to 16% in Gr 2.

(CGH=Cervicogenic Headache, RCT=Randomized Clinical Trial, CSM=Cervical Spinal Manipulation, GR 1=Group 1, Gr 2=Group 2, PT=Participants, ET=Exercise Therapy, CT=Combined Therapy, C=Control, L=Left, R=Right, SO=Suboccipital, TRAP=Trapezius, EXP=Experimental)

2.6.4 Effect of Manipulation on Pain and Disability in Individuals with CGH

A systematic review by Chaibi and Russel (2012) on manual therapies in the treatment of CGH examines seven RCT s in which CSM was administered in order to decrease the pain and disability associated with CGH. All six RCTs were considered to be of good methodological quality. The authors conclude that CSM was an effective method for restoring normal cervical range of motion and alleviating pain and disability associated with CGH. Recently, Haas *et al.* (2018), in the first full-scale dose-response study of CSM finds that CGH pain and disability have a statistically significant linear dose-response gradient at 12 and 24 week time points. Thus, CSM is an effective evidence-based treatment for CGH.

2.6.5 Effect of Manipulation on Muscle Activity of the Neck in Individuals with CGH

The effect of CSM on muscle activity of the deep and superficial cervical flexors in individuals with CGH and mechanical neck pain has been extensively investigated (Falla *et al.* 2002; Jull, O'Leary and Falla 2008; Jull and Falla 2016). Spinal manipulation appears to decrease the muscle activity of the superficial flexors and improve the strength of the deep flexors, thereby normalizing neck flexor neurophysiological functioning (Sterling, Jull and Wright 2001; Metcalfe, Reese and Sydenham 2006). There is a paucity of literature at present with regards to the effect of CSM on muscle activity and strength of the trapezius and posterior cervicals. More research involving the effect of CSM on neck muscle activity is required to form a holistic strategy for addressing muscle imbalances in CGH sufferers.

2.7 CONCLUSION

From the literature and according to recent guidelines, it is evident that CSM is one of the recommended interventions for the treatment of CGH (Bryans *et al*. 2011; Côté *et al.* 2019). It has been observed that CSM produces neurophysiological effects such as alteration of muscle activity, muscle strength and pain regulation The mechanism through which spinal manipulation produces these neurophysiological effects has not been empirically proven and therefore requires further investigation. Thus, this study has aimed to assess the effect of upper cervical spinal manipulation on muscle activity of the neck and to note the changes in pain and disability to add to the existing framework regarding the neurophysiological effects of spinal manipulation and its effect on CGH.

CHAPTER THREE
METHODOLOGY

3.1 INTRODUCTION

This chapter describes the methodology utilised to achieve the aims and objectives of this study, along with the ethical considerations that were taken into account to ensure participant safety and well-being.

3.2 STUDY DESIGN

This study made use of a post-positivistic paradigm and a quantitative approach using a randomized, controlled, pre-test, post-test, experimental design. This study's design has been utilised in similar research studies (Bicalho *et al.* 2010; de Camargo *et al.* 2011; Chowdhury *et al.* 2013) investigating the effect of spinal manipulation. It allows for comparing both a control and an intervention group and measuring the change resulting from the experimental treatment (Chowdhury *et al.* 2013).

3.3 STUDY LOCATION AND PERMISSION TO CONDUCT THE STUDY

The study took place at the DUT Chiropractic Day Clinic in Durban, South Africa following the approval from the Clinic Director (Appendix A) and the Institutional Research Ethics Committee (IREC) (Appendix B).

3.4 STUDY POPULATION

The population investigated in this study were people residing in the eThekwini municipality, who suffered from CGH and were between 18 and 50 years of age.

3.5 SAMPLING

3.5.1 Participant Recruitment

Participants were recruited through advertisements (Appendix C) placed, with permission from the appropriate authorities, around sports clubs, gyms, university campuses and

local shopping malls around Durban. Potential participants were asked to contact the researcher telephonically for more information.

3.5.2 Sample Selection

Participants who contacted the researcher were screened for eligibility using a telephonic interview as detailed in **Table 3.1**:

Table 3.1: Telephonic questions to determine preliminary inclusion for the study

Questions:	**Answers to participate:**
Are you willing to answer a few questions?	Yes
Have you had a headache ≥ 5 days per month for at least 3 months?	Yes
Was the headache located on one side of the head near the forehead region?	Yes
Did the headache shift to the other side of the head at all?	No
Did the headache start or get worse when moving your head?	Yes
Do you often find your neck in an awkward position while the headache is occurring?	Yes
Is it painful when someone touches the back of your neck near your skull?	Yes
Do you experience a dull aching pain in your neck, shoulder or arm on the same side of your body that the headache is occurring?	Yes
Do you have a history of head/neck trauma?	No
Are you currently receiving any physically manipulative treatment for your headache?	No

If the participant met the telephonic interview criteria, they were then invited to a consultation at the Chiropractic Day Clinic where a case history (Appendix D), physical examination (Appendix E) and cervical regional exam (Appendix F) were performed to determine their eligibility according to the following criteria:

3.5.2.1 Inclusion Criteria

1. All participants were required to be between 18 and 50 years of age. The criteria excluded those under 18 thereby avoiding the need for parental consent and included older individuals as the mean age of CGH sufferers has been reported to be 42.9 years old (Biondi 2005).

2. Participants were required to have a minimum of three of the following diagnostic criteria described by Sjaastad (1990) for CGH for inclusion in the study:

   - Unilateral headache without side shift.
   - Precipitation of headache by neck movement, sustained awkward head positioning or by external pressure on the upper cervical or occipital area on the symptomatic side.
   - Restricted range of motion in the upper cervical spine.
   - Ipsilateral non-radicular neck, shoulder and arm pain.
   - Upper cervical spine facet fixation.

The International Headache Society diagnostic criteria for CGH could not be used in this study as it requires the use of diagnostic nerve blocks which was impractical for this study setting.

3.5.2.2 Exclusion Criteria

The following five criteria excluded a participant from the study:

1. Contraindications to CSM determined during the case history, physical examination and cervical regional examination. Examples include:

   - Vertebral-basilar artery insufficiency.
   - Atherosclerosis of major vessels.
   - Metabolic disorders, such as osteoporosis, osteomalacia and clotting disorders.
   - Tumours (lung, thyroid, breast and bone).
   - Bone infections (Osteomyelitis, tuberculosis).
   - Trauma (Instability, fractures, severe sprains and strains).
   - Arthritis.
   - Neurological complications.
2. Those with primary headaches, e.g. migraines.
3. Participants who were not willing to have the back of their neck shaved.
4. Participants currently receiving any physical manipulative treatment for their headache.
5. Participants with contraindications to surface electromyography, such as allergies to the adhesive gel used in the electrodes.

3.5.3 Sample Size

An *a priori* sample size calculation was determined for this study using GPower version 3.1.9.2 (Faul *et al.* 2007). A 5% level of significance, power of 0.80 and an effect size of 0.40 indicated that a sample of 45 participants would be required.

3.5.4 Sample Allocation

The eligible participants were randomly allocated into one of three groups using the hat method. The participant, with their eyes turned away, reached into the hat and randomly picked a small piece of folded paper. The participant then gave the piece of paper to the researcher. The piece of paper, with A, B or C printed on it, was then unfolded by the researcher without the participant seeing what letter was printed (Schulz and Grimes 2002). No blinding was applied in this study. Sample allocation, assessment and application of the interventions were performed by the researcher alone.

The following groups were formed:

- Group A = Intervention (n=15).
- Group B = Placebo (n=15).
- Group C = Control (n=15).

3.6 MEASUREMENT TOOLS

This study made use of independent and dependent variables. Independent variables are stable and unaffected by any other variables that are also measured (Hill 1998); therefore, the independent variable in this study was upper cervical spine manipulation. Dependent variables are expected to change as a result of an experimental manipulation of the independent variable or variables, and thus the dependent variables in this study were force output, muscle activity, pain and disability.

3.6.1 Subjective Measures

3.6.1.1 Pain Rating

Pain was measured by the Numerical Pain Rating Scale (Appendix G). The Numerical Pain Rating Scale is a valid and reliable method for measuring pain. The threshold for the minimal clinically important difference has been found to be 1.3 (Cleland, Childs and Whitman 2008). Therefore, a decrease of 1.3 in pain perception was deemed favourable and to indicate a clinically significant change.

3.6.1.2 Headache Disability

Disability was measured by the Headache Disability Index (HDI) (Appendix H) (Jacobson *et al.* 1994). The HDI is a self-administered, 25 question questionnaire in which participants were asked to answer both emotional and functional questions with a choice of three responses ('Yes', 'Sometimes' or 'No') that most closely described their condition. Scores for each answer were 'Yes' = 4 points, 'Sometimes' = 2 points and 'No' = 0 points, giving a total scale of 100%. The questionnaire was completed at the first consultation prior to an intervention taking place and 48 hours later, telephonically. Each participant's disability was therefore recorded and compared against the intervention they received.

The HDI is a valid subjective tool and has been used successfully to measure disability associated with headaches (Blizzard, Grimmer and Dwyer 2000). A 29-point change (95% confidence interval) or greater in the total score from test-retest must occur before the changes can be attributed to treatment effects (Jacobson *et al.* 1994). Consent to use the index was granted (Appendix H).

3.6.1.3 Headache Diary

Participants were required to keep a headache diary (Appendix J) seven days prior to the consultation and were asked to record headache intensity, frequency and duration, as was done by De Busser (2001) and Prithipal (2003). A headache diary is a self-administered tool in which participants are required to mark off the appropriate answer which best describes the characteristics of their headache. A headache diary is a simple and reliable tool in assessing headache intensity, frequency and duration (Tassorelli *et al.* 2008; Niere *et al.* 2004). In the case of this study, a headache diary was not used to measure the effect of the interventions but rather to see the character of the participant's headache prior to the study.

Headache frequency pertains to the number of CGHs experienced over a one week period. The frequency of CGHs was calculated by dividing the number of discreet headache periods by seven, representing the number of days in the week. Headache duration pertains to the length of time in minutes between headache onset and offset. The duration was calculated by dividing the total headache duration in minutes by the number of headache episodes experienced. Headache intensity pertains to the severity of the headache episode (De Busser 2001). The intensity was calculated by assigning numerical values to intensity categories, for example, 'mild' was assigned the value of one, 'moderate' was assigned the value of two, 'fairly severe' was assigned the value of three and 'severe' was assigned the value of four. The mean of these numerical values was then calculated.

3.6.2 Objective Measures

3.6.2.1 Muscle Activity

A surface electromyography (sEMG) was used to measure muscle activity in volts. A sEMG is a repeatable and reliable tool for measuring muscle activity (Mathur, Eng and MacIntyre 2005; de Carmago 2011). Electromyographic activity was obtained using the Biopac Bionomadix MP150 Data Acquisition System (Biopac systems® Inc.) which has a data sampling rate of 2000 Hz. It has an internal high-pass and low-pass filter from 5.0 Hz to 500 Hz to ensure quality amplification of the sEMG waveform and elimination of any artefact or noise interference.

sEMG signal can be distorted due to cross-talk between muscles, signal to noise ratio and ambient noise from devices, such as cellular telephones (Staudenmann *et al.* 2010). Cross-talk refers to a signal contribution originating from other muscles (Staudenmann *et al.* 2010). Cross-talk was minimized by using small disposable, conductor electrodes placed using the standardized inter-detection surface spacing of 1-2 centimetres (Criswell 2010). To minimize electrical noise, which may be caused by the movement of electrodes, cables and connectors, the subject and ambient noise from electromagnetic radiation (Roy *et al.* 2007; Staudenmann *et al.* 2010), cellular telephones and other electrical devices were turned off, dead skin was removed by abrading the skin and the participant was asked to remain as still as possible (Roy *et al.* 2007).

The correct electrode placement is important in order to gain a reliable sEMG reading (Roy *et al.* 2007). Electrodes were placed between the innervation zone and the fibre tendon intersection and aligned to the muscle fibre direction (Staudenmann *et al.* 2010), in the muscle belly (Sommerich *et al.* 2000). The electrode placement as described by Criswell (2010) was used in this study to place electrodes on the trapezius, sternocleidomastoid and posterior cervical muscles, as detailed in **Table 3.2.** The electrodes were disposed of after measurements had been taken and a clean set was used for each new participant.

Table 3.2: Placement of the sEMG electrodes on the muscles of the cervical spine (Criswell 2010)

Muscle	Electrode placement	Example
Trapezius	Two electrodes were placed so that they ran parallel to the muscle fibres of the upper trapezius, along the ridge of the shoulder, slightly lateral to and one-half distance between the cervical spine at C7 and the acromion.	
Sternocleidomastoid (SCM)	The electrodes were placed half the distance between the mastoid process and the sternal notch, slightly posterior to the centre of the muscle belly so that they run parallel to the muscle fibres and are 2cm apart.	
Posterior cervicals	The spinous processes of the cervical spine and the two muscle bellies that lie just lateral to it were palpated. Two electrodes (2cms apart) were placed so that they ran parallel to the spine, approximately 2 cm from the midline, over the muscle belly at the level of C4.	

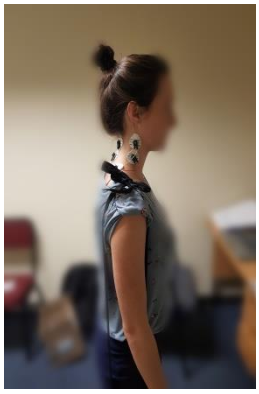
The three values of muscle activity (mean and peak amplitudes) were recorded bilaterally for each muscle tested. Normalization was used to ensure the standardization of the sEMG data. The data obtained from the surface EMG were kept electronically and were directly transferred to an excel spreadsheet for analysis.

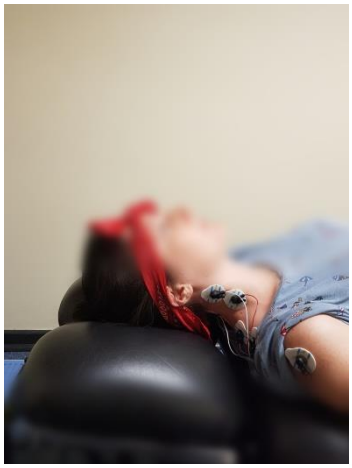
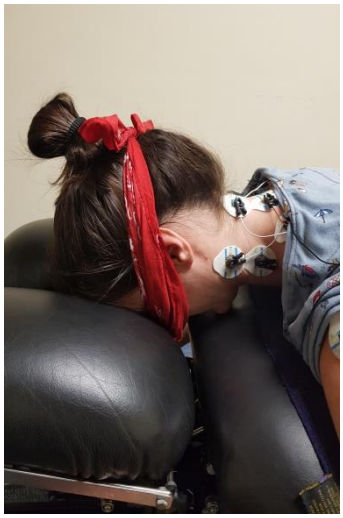
3.6.2.2 Force Output

The Biopac-TSD121C dynamometer was used to measure force output. Force output was recorded using a maximum isometric voluntary muscle contraction (MVC) in kilograms for each muscle included in the study. For the SCM and posterior cervicals, no distinction was made for left and right, as force output was recorded during flexion for the SCM and extension for the posterior cervicals. A distinction between left and right force output of the trapezius was implemented as force output was measured during shoulder elevation. The mean and peak MVC was recorded pre-test and post-test.

Rope slings were attached to the dynamometer to allow assessment of various muscles in the supine, prone and standing position. According to Maayah *et al.* (2012), the dynamometer has high intra-and-inter-reliability for testing muscle strength. The researcher taught the participant to perform MVC s of the three muscles to maximum effort, in line with recommendations made by Halaki and Ginn (2012). The participant then practiced the above movements. Each muscle MVC was only practiced once to guard against the effects of fatigue. Participants exerted maximum effort against resistance for five seconds, followed by a 30 second rest interval before the subsequent muscle MVC was practiced. Once deemed proficient by the researcher, objective measurements were recorded. The SCM was measured first, followed by the posterior cervicals and then bilaterally, the trapezius muscles. In order to standardize the manner in which the MVC was obtained, the following method was used, as seen in **Table 3.3.**

Table 3.3: Force output positions (Falla *et al*. 2002; Ekstrom, Soderberg and Donatelli 2005; Rezasoltani *et al*. 2005)

Muscle	Position	Movement against resistance
Trapezius (Bilaterally)	Standing 	Resistance was applied in the form of a rope sling with adjustable straps attached around the participant's shoulder (Just distal to electrode placement), connected to the dynamometer which was secured to the bed. The participant produced a shoulder shrug exerting maximum effort against the dynamometer. The resistance was against shoulder elevation.

SCM	Supine 	Resistance was applied in the form of an adjustable bandanna attached around the participant's forehead, connected to a rope sling and attached to the dynamometer, which was secured to the bed. The participant was asked to exert maximum effort against the dynamometer. The resistance was against cervical flexion.
Posterior cervicals	Prone 	Resistance was applied in the form of an adjustable bandanna attached around the dorsal aspect of the participant's head (at the level of the External Occipital Protuberance), connected to a rope sling and attached to the dynamometer which was secured to the bed. The participant was asked to exert maximum effort against the dynamometer. The resistance was against cervical extension.

3.7 INTERVENTIONS

3.7.1 CSM – Group A

The participants received a CSM applied to the facet joint in the upper cervical spine which was identified by the researcher as having hypo-mobility (i.e. joint fixation), using motion palpation. This involved the evaluation of cervical facet joint movement by palpating facet joints, while using a series of left and right cervical rotations (Bergmann and Peterson 2010). The participants were in a supine position and received low amplitude, high-velocity (HVLA), postero-anterior (PA) directed thrust, at the restricted upper cervical spine segment**.** The researcher's indifferent hand supported the head,

while the thumb contacted the fixated facet segment and delivered a PA rotational thrust (Bergmann and Peterson 2010).

3.7.2 Placebo - Group B

The participants in the placebo group were placed in the supine position. Their upper cervical spine was taken to end range of PA rotation of a fixated cervical facet joint. No HVLA thrust was applied (Haavik and Murphy 2012). The researcher's indifferent hand supported the head, while the researcher's thumb contacted the upper cervical spine segment and took the joint to the end range of PA rotation; this position was held for 5 seconds, similar to the time taken to deliver a HVLA thrust (Bergmann and Peterson 2010). This allowed the participant to perceive that they were experiencing treatment.

3.7.3 Control - Group C

Participants in this group lay supine for five minutes and did not receive any form of intervention or neck motion palpation but were subject to all the measurements as per group A and B.

3.8 RESEARCH PROCEDURE

All components of the research procedure were performed by the researcher alone. After the participant was deemed eligible to participate, the participant was contacted telephonically in order for the research study to be explained and for the participant to agree to use a headache diary for seven days. Once the headache diary was completed, a consultation was booked for the participant at the DUT CDC. During the consultation, the research procedure was explained verbally to the participant. Thereafter, the participant was required to read and sign a letter of information (Appendix M) and informed consent (Appendix N). The participant was then allowed to query any aspects of the study and was informed that he/she could exit the study at any point if he or she so wished. The assessment of the participant then took place in order to confirm his or her eligibility. A case history (Appendix D), physical examination (Appendix E) and cervical regional exam (Appendix F) were performed and signed off by the clinician on duty. Thereafter, the participant was randomly allocated into one of the three groups using the hat method.

Subjective measurements were then taken first. Thereafter, the sEMG and force output readings were taken. The electrodes were placed according to **Table 3.2** and then the participant performed three repetitions of each MVC as the pre-test, with rest periods of 30 seconds between each repetition to reduce the effects of fatigue (Criswell 2010). The

three values of muscle activity obtained by the sEMG and the three values of force output obtained by the dynamometer were then recorded. The electrodes remained on the participant throughout the study to avoid any alterations to the results due to a difference in electrode placements between readings. The participant was allowed five minutes to rest before an intervention took place (Criswell 2010).

The interventions were then applied as per the group designation of the participant. Post-intervention sEMG and force output measurements were then taken within 5 minutes of the interventions. Post-intervention pain and disability measurements were recorded 48 hours later. The electrodes were removed and the participant was thanked for his or her involvement in the study. On completion of the study, all participants were given a voucher (Appendix O) for one free treatment. The voucher was marked with the date it was issued and for whom it was valid. The voucher was valid for three months. Any participants requiring further treatment (from either group A, B or C) were referred to an appropriate practitioner or to the clinic as an outpatient for treatment.

3.9 DATA REDUCTION AND ANALYSIS

3.9.1 Data Reduction

The subjective and objective measures were recorded in the following manner:

- **Muscle activity**: At the consultation, prior to the interventions, three trials of muscle activity were recorded during MVCs of each muscle. The raw data were then processed using root mean squared (RMS) to convert the raw data to a waveform suitable for analysis. The peak amplitude was then obtained by using the maximum peak muscle activity from the three readings, and this was recorded per muscle. The mean muscle activity was obtained by using the muscle activity for the three trials, per muscle, divided by three, to obtain the average mean muscle activity. This was then repeated after the intervention, placebo or control. In order to compare among the groups, the peak and the mean muscle activity was normalized. This was done by normalizing them to a reference value which, in this study, was the maximum peak amplitude observed from the three trials prior to the interventions, i.e. at the baseline. This converted the muscle activity to a percentage (Konrad 2005; Halaki and Ginn 2012).

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

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

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

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

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

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

The difference in normalized muscle activity between pre- and post-test for all of the muscles was then calculated (i.e. *pre – post = difference*) for all three groups.

- **Force output:** At the consultation, prior to the interventions, three trials of force output were recorded during MVC s of each muscle**.** The mean force output was obtained by using the force output for the three trials per muscle, divided by three, to obtain the average mean force output obtained during the MVC s, per muscle. This was reported as mean force output. This was then repeated after the intervention, placebo or control. In order to compare among the three groups, the mean force output was normalized. This was done by normalizing to a reference value which, in this study, was the maximum force output observed from the three trials, prior to the interventions, i.e. at baseline. This converted the force output to a percentage (Konrad 2005; Halaki and Ginn 2012).

  The pre- and post-mean force output was calculated using the following equation:

  (pre/post mean force output ÷ pre-peak force output) x 100 = %

  The difference in normalized force output between pre and post-test for all of the muscles was then calculated (i.e. *pre – post = difference*) for all three groups.

- **Pain rating:** This was recorded as a raw score out of 10 before the intervention and telephonically 48 hours after the intervention. The difference in pain was then calculated (i.e. *pre – post = difference*) for all three groups.
- **Disability:** This was recorded as a percentage before the intervention and telephonically 48 hours after the intervention. The difference in disability was then calculated (i.e. *pre – post = difference*) for all three groups.
- **Headache diary:** The diary recorded the participants' mean headache intensity (mild, moderate, fairly severe and severe), frequency (number of headaches) and duration (amount of time headache occurred) over a one week period.

3.9.2 Data Analysis

IBM SPSS version 26 was used to analyse the data. A *p*-value <0.05 was considered as statistically significant. Age was normally distributed and, thus, one-way ANOVA was used to compare the ages between the three treatment groups. Race and gender were compared between groups using Fisher's exact tests. All outcome measures were treated as non-normally distributed, due to extreme skewing in some cases, small sample sizes and presence of outliers. Thus, non-parametric tests were used to compare groups and summary statistics reported medians and inter-quartile ranges.

Differences between pre- and post-measurements were calculated as pre- minus post-values, therefore negative changes were increases over time while positive changes were decreases over time. Kruskal-Wallis tests were used to compare three independent groups changes (inter-group differences), while Wilcoxon signed ranks tests were used to compare paired measurements within groups (intra-group changes).

3.10 ETHICAL ISSUES

Ethical clearance was obtained from the DUT Institutional Research Ethics Committee (reference number IREC 032/19) and this study was registered with the Pan African Clinical Trial Registry (reference number PACTR201906717506525). Permission to conduct the study was obtained from the clinic director of the Chiropractic Day Clinic and permission to access student and staff of the university was obtained from the research director.

In this study, the ethical pillar of autonomy was adhered to by ensuring that the participants signed a letter of information and informed consent at the consultation, as well as confirmed verbally that they understood the procedure. No coercion was used to recruit participants. Participant data will be stored at DUT for five years. In addition, participant confidentiality was ensured by allocating codes to the participants, whereby no participant names appeared in the dissertation or any possible publication resulting from the research. The participant's demographic data were kept in the participant's file. All other participant data were stored and coded in an excel spreadsheet on a password protected laptop. Participant one had all his or her data in one column in the excel spreadsheet. The group allocation of participant one (A, B or C) was in a column next to the data. This system was used for all 45 participants.

All participants received a voucher for one free treatment and if further treatment was needed, they were referred to an appropriate practitioner. All participants were informed

they would have a one in three chance of being allocated to the manipulation/intervention group. Beneficence was accounted for as the results of this study could affect how this condition is treated and therefore benefit the profession and patients. Justice was accounted for; therefore, no participants were excluded from the study based on race, gender or occupation. Non-maleficence was enforced, ensuring no harm was done to the participants. Only safe and established interventions and measurement tools were applied to the participants.

CHAPTER FOUR
RESULTS

4.1 INTRODUCTION

This chapter presents the results obtained in the study. The data will be presented in the form of graphs and cross tabulations and is line with the current CONSORT guidelines for the reporting of RCT data.

4.2 CONSORT FLOW DIAGRAM

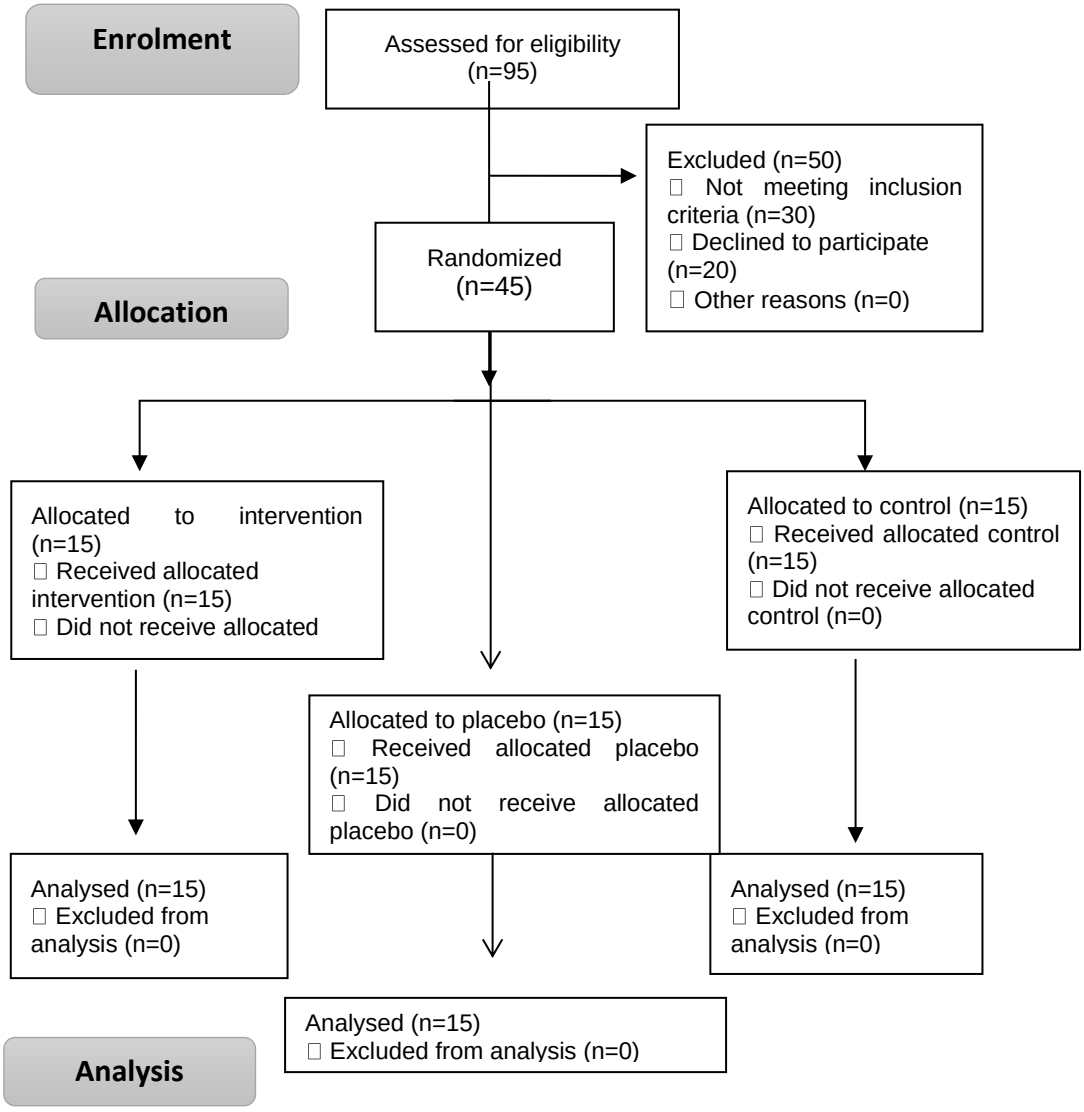


Figure 4.1: Consort flow diagram

4.3 DEMOGRAPHIC CHARACTERISTICS

4.3.1 Age

Table 4.1 shows the descriptive outcomes for the ages of the participants, for the total sample and per group. The participants in the placebo group had the highest mean age, yet there were no significant differences found between the three groups for age (*p*=0.460; one way ANOVA).

Table 4. 1: Age of the participants per group

	n	M	±SD	±SE	CI	Range
Intervention	15	29.7	8.7	2.2	24.8-34.5	20-46
Placebo	15	34.7	11.9	3.1	28.3-41.3	21-50
Control	15	33.3	12.7	3.2	26.3-40.4	19-50
Total	45	32.6	11.2	1.7	29.2-35.9	19-50

4.3.2 Race and Gender

Table 4.2 shows the gender and race distribution of participants per group. More women enrolled in the study than men, but there was no difference between the groups in terms of gender (*p*=0.566; Fisher's exact test). The majority of the participants were from the white race group with a marginally significant finding for race between the groups (*p*=0.046; Fisher's exact).

Table 4. 2: Gender and race distribution of participants per group

		Intervention		**Placebo**		**Control**		**Total**	***p*-value**
		n (%)		n (%)		n (%)			
Gender	Female	13	86.7	10	66.7	11	73.3	34	0.566
	Male	2	13.3	5	33.3	4	26.7	11	
Race	Black	0	0.0	4	26.7	3	20.0	7	0.046
	White	11	73.3	8	53.3	10	66.7	29	
	Indian	4	26.7	3	20.0	0	0.0	7	
	Coloured	0	0.0	0	0.0	2	13.3	2	

4.4 SUBJECTIVE MEASURES: PAIN, DISABILITY AND HEADACHE FREQUENCY, INTENSITY AND DURATION

The subjective measures were not normally distributed and this resulted in non-parametric statistical tests being utilised to analyse the data. Clinical significance was determined for pain rating as a decrease of 1.3 in pain perception (Cleland, Childs and Whitman 2008) and for a disability rating as a 29-point change or greater in the disability total score from test-retest (Jacobson *et al.* 1994).

4.4.1 Pain Rating

At baseline, there were no significant differences found between the three groups when using the Kruskal-Wallis Test for pain (p=0.819), indicating that the groups were similar in their baseline rating of pain.

4.4.1.1 Intragroup Analysis for Pain Rating

Figure 4.2 shows there were significant decreases in pain (using Wilcoxon Signed Rank Test) over time for the intragroup analysis from pre- to post-test within the intervention (p=0.001) and placebo groups (p=0.011), but not in the control group (p=0.157). In the intervention group, a clinically significant decrease in pain from pre- to post-test occurred.

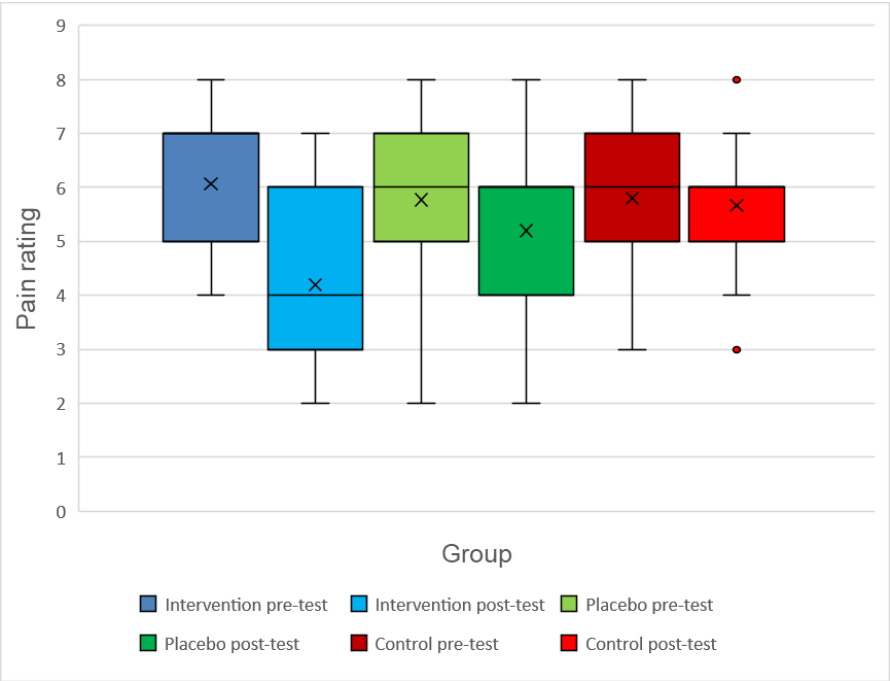


Figure 4.2: Intragroup analysis of the pre and post-test pain rating measures in the three groups

4.4.1.2 Intergroup Analysis for Pain Rating

The difference (i.e. *pre – post = difference*) in pain rating between the three groups was highly significant ($p<0.001$). A higher level of change equated to a greater decrease in pain rating post-test. The intervention group showed the greatest change, indicating that this group experienced a greater decrease in pain post-test. The placebo and control groups also experienced a decrease in pain post-test; however, this decrease was not statistically or clinically significant. Using, Pairwise multiple comparison tests, the intervention group was shown to be different ($p<0.001$) from the placebo and control groups, with the control and placebo groups not being different ($p=0.379$) from each other, as seen in **Figure 4.3**. Clinically, when compared with the placebo and control group, only the intervention group experienced a significant decrease in pain rating.

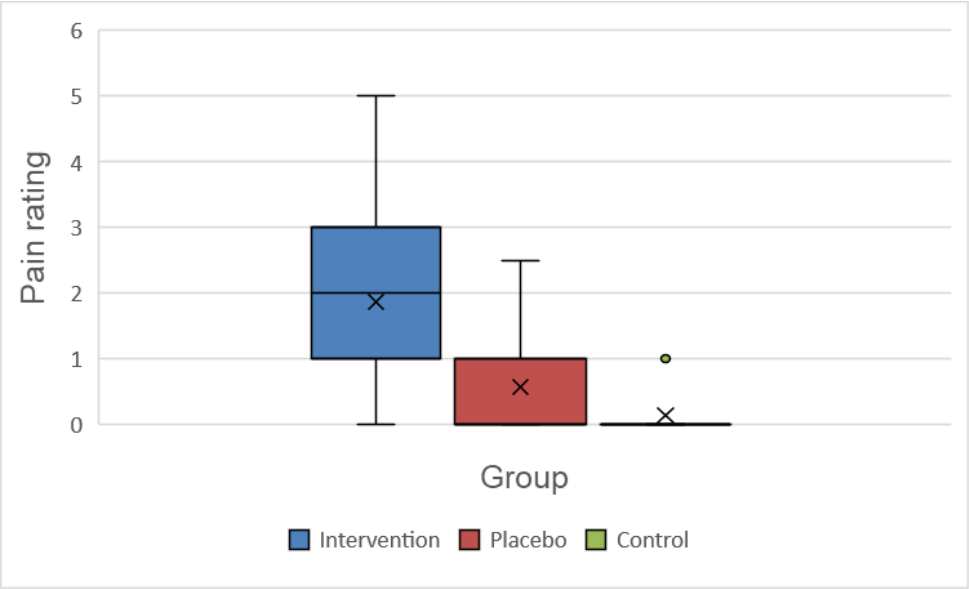


Figure 4. 3: Intergroup analysis of the difference in pain rating measures between the three groups

4.4.2 Disability Rating

There were no significant differences found at baseline (pre-test) between the three groups, for disability rating ($p=0.737$; Kruskal-Wallis Test), indicating that the groups were similar at baseline.

4.4.2.1 Intragroup Analysis for Disability Rating

Figure 4.4 shows there were significant decreases in disability over time for the intragroup analysis, performed using Wilcoxon Signed Rank Test, from pre- to post-test within the intervention group ($p=0.001$), but not within the placebo ($p=0.058$) and control groups

(p=0.201). None of the groups experienced a clinically significant decrease in disability rating from pre- to post-test.

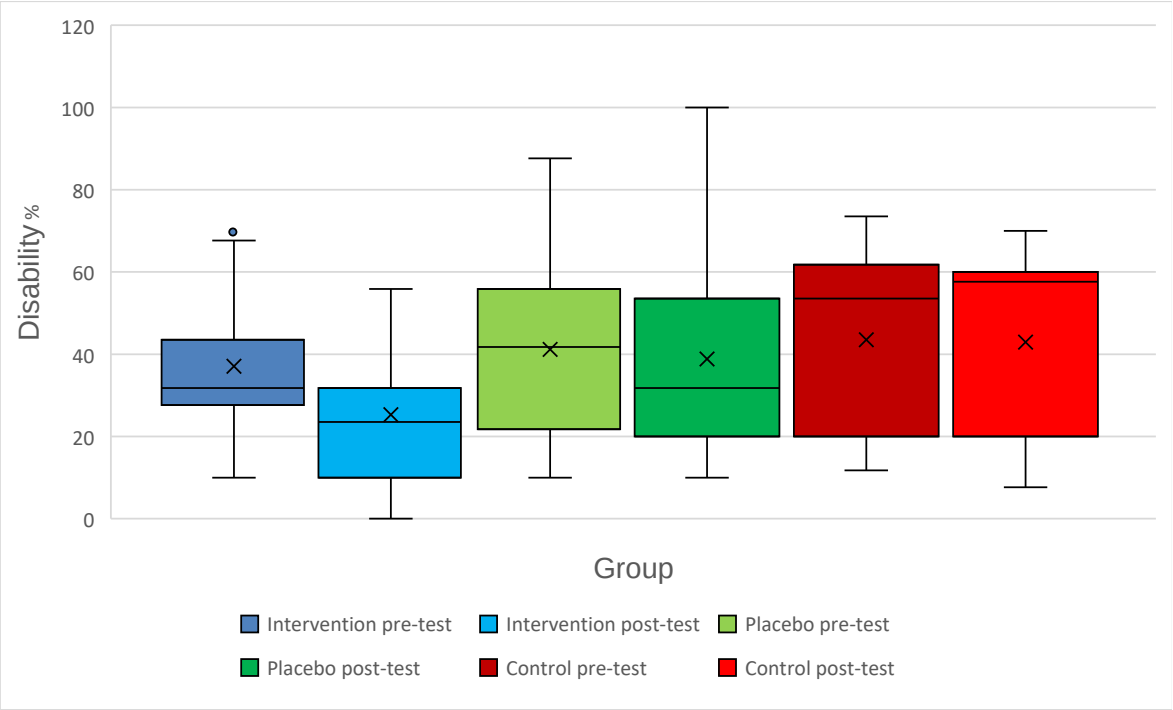


Figure 4.4: Intragroup analysis of the pre and post-test disability rating measures in the three groups

4.4.2.2 Intergroup Analysis for Disability Rating

The difference (i.e. *pre – post = difference*) in disability rating between the three groups was highly significant (p<0.001). A higher level of change equated to a greater decrease in disability rating post-test. The intervention group showed the greatest change, indicating that this group experienced a greater decrease in disability post-test. The placebo and control groups also experienced a decrease in disability post-test; however, this decrease was not statistically or clinically significant. Using, Pairwise multiple comparison tests, the intervention group was found to be different (p<0.001) from the placebo and control groups but control and placebo groups were not different (p=0.687) from each other, as shown in **Figure 4.5**. Clinically, when the groups were compared, no significant decreases for disability rating were experienced.

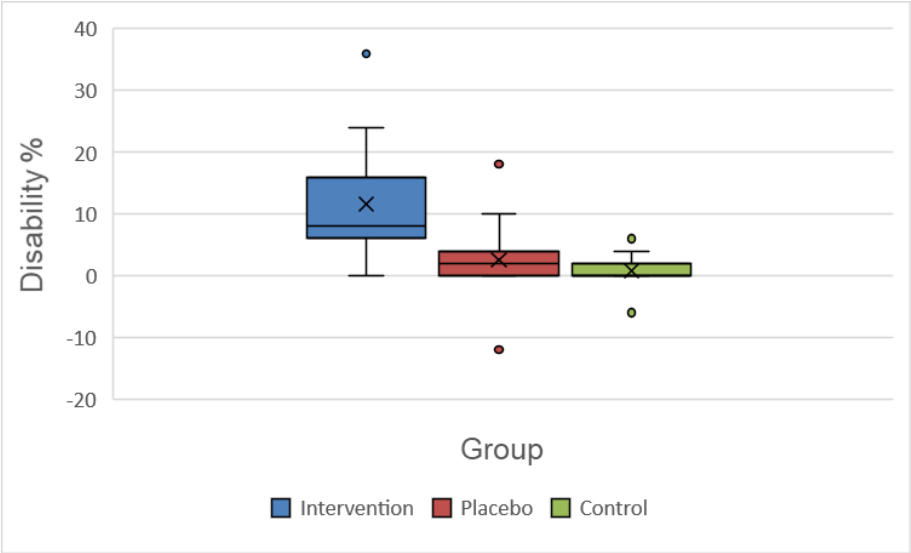


Figure 4.5: Intergroup analysis of the difference in disability rating measures between the three groups

4.4.3 Headache Frequency, Duration and Intensity

Headache frequency, duration and intensity were only recorded during the one week pre-intervention, as the results show in **Table 4.3**. At baseline (pre-test), there were no significant differences found between the three groups, when using the Kruskal-Wallis Test, for headache frequency (p=0.298), duration (p=0.746) and intensity (p=0.163). This indicates that the groups were similar at baseline in their reporting of these measures.

Table 4.3: Headache diary outcomes per participant showing baseline and interquartile ranges

HD	Intervention			Placebo			Control		
	Q1	Q2	Q3	Q1	Q2	Q3	Q1	Q2	Q3
Frequency (HA days)	0.1	0.3	0.7	0.3	0.7	1.0	0.3	0.6	0.7
Duration (min)	60.0	105.0	180.0	85.0	120.0	225.0	60.0	142.5	250.0
Intensity	1.0	2.0	2.3	2.0	2.2	3.0	1.6	2.2	2.4

(HD=Headache diary, HA=Headache, Q1= First quartile, Q2= Second quartile, Q3= Third quartile)

4.5 OBJECTIVE MEASURES: MUSCLE ACTIVITY (MV) AND FORCE OUTPUT (KG)

The objective measures were not normally distributed and this resulted in non-parametric statistical tests being utilised to analyse the data.

4.5.1 Force Output

The force output results at baseline were assessed for all three groups, for each muscle (left and right Trap, SCM and PC), and there were no significant differences found between groups at baseline ($p<0.005$).

4.5.1.1 Intragroup Analysis for Force Output

- Sternocleidomastoid (SCM)

Figure 4.6 shows that there was no significant difference, using Wilcoxon Signed Rank Test, between pre-test and post-test measures within the intervention ($p=0.112$), placebo ($p=0.363$) and control ($p=0.191$) groups for force output.

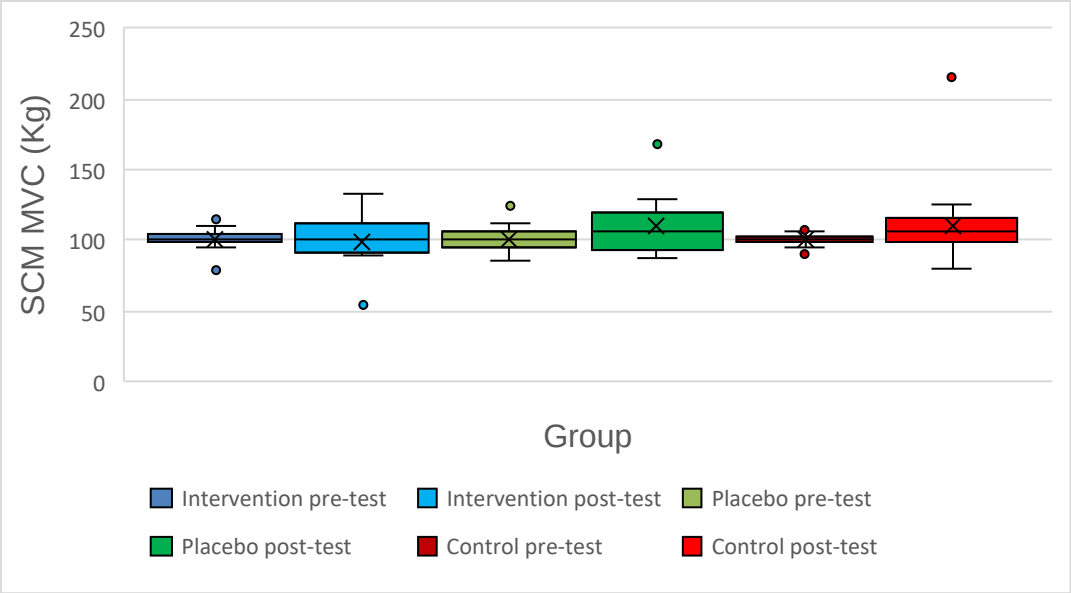


Figure 4.6: Intragroup analysis of the force output measures for the SCM in the three groups

- Posterior cervical (PC)

Figure 4.7 shows that there was no significant difference, using the Wilcoxon Signed Rank Test, between pre-test and post-test measures within the intervention ($p=0.140$), placebo ($p=0.910$) and control ($p= 0.256$) groups.

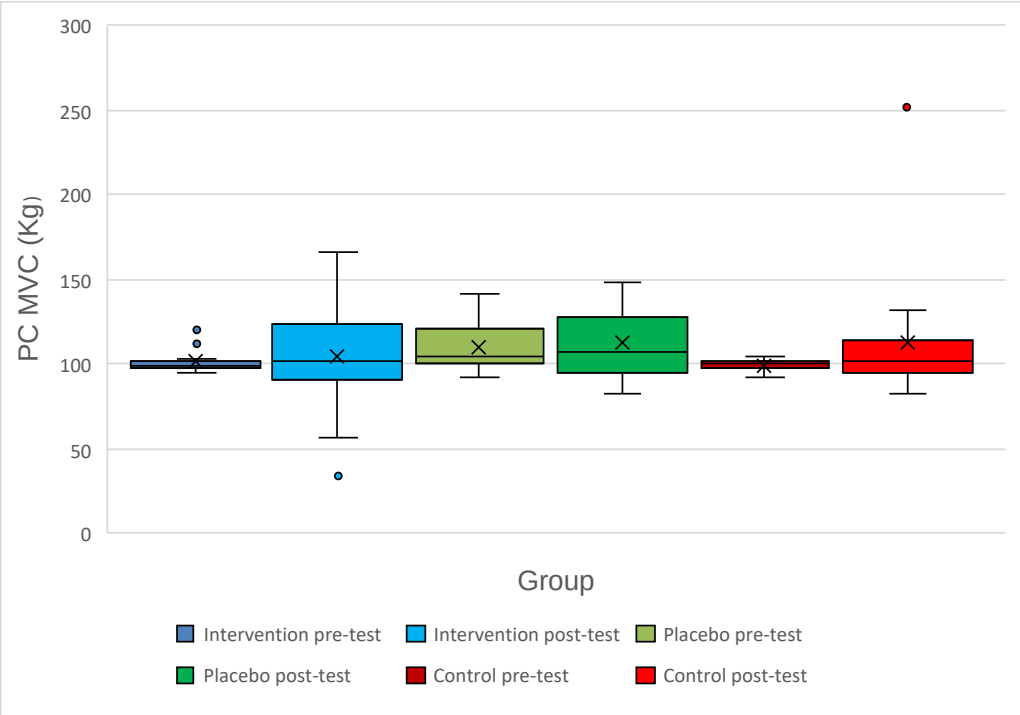


Figure 4.7: Intragroup analysis of the force output measures for the PC in the three groups

- Right Trapezius (TrapR)

Using Wilcoxon Signed Rank Test, it was found that the intragroup pre- and post-test measures were not significant for the intervention (p=0.363) and placebo (p=0.427) groups but for the control group (p=0.031), a significant increase occurred post-test as seen in **Figure 4.8**.

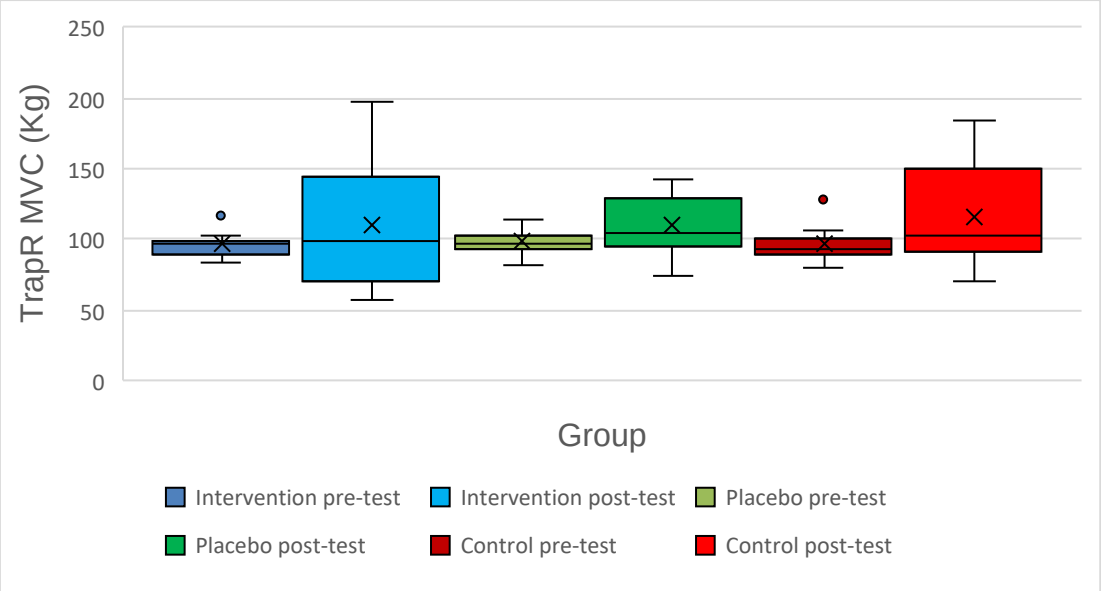


Figure 4.8: Intragroup analysis of the force output measures for the TrapR in the three groups

- Left Trapezius (TrapL)

Using the Wilcoxon Signed Rank Test, it was found that there were no significant differences between pre-test and post-test measures for the intervention group (*p*=0.925), with a significant increase being found in the placebo (*p*=0.027) and control (*p*=0.041) groups, as seen in **Figure 4.9**.

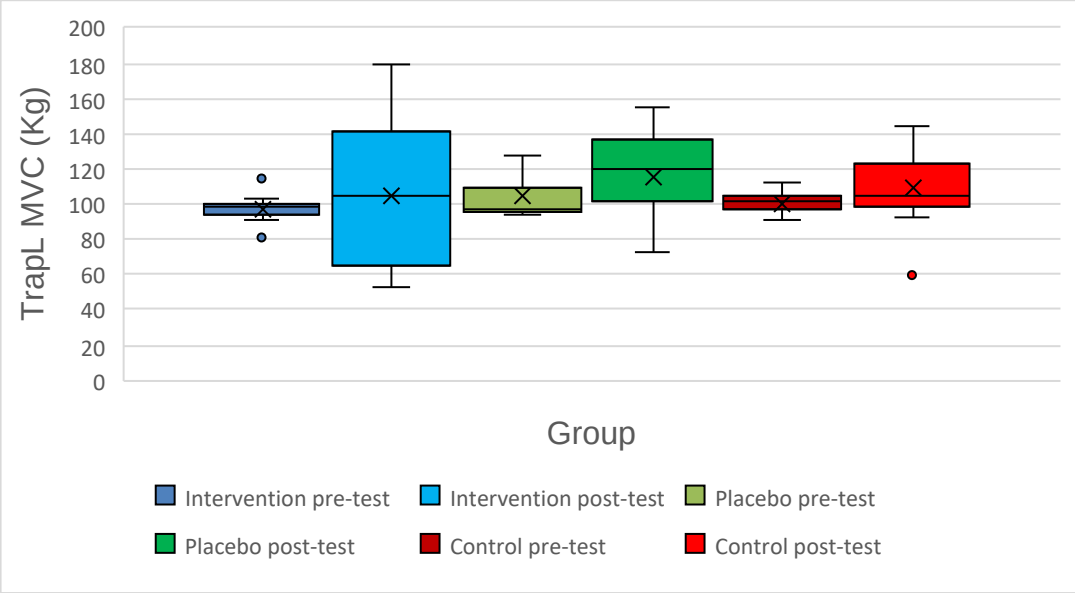


Figure 4.9: Intragroup analysis of the force output measures for the TrapL in the three groups

4.5.1.2 Intergroup Analysis for Force Output

Using the Independent-samples Kruskal-Wallis Test, no significant differences were found between the three groups for the SCM (*p*=0.997), PC (*p*=0.531), TrapR (*p*=0.290) and TrapL (*p*=0.197), as seen in Figures **4.10, 4.11, 4.12 and 4.13.**

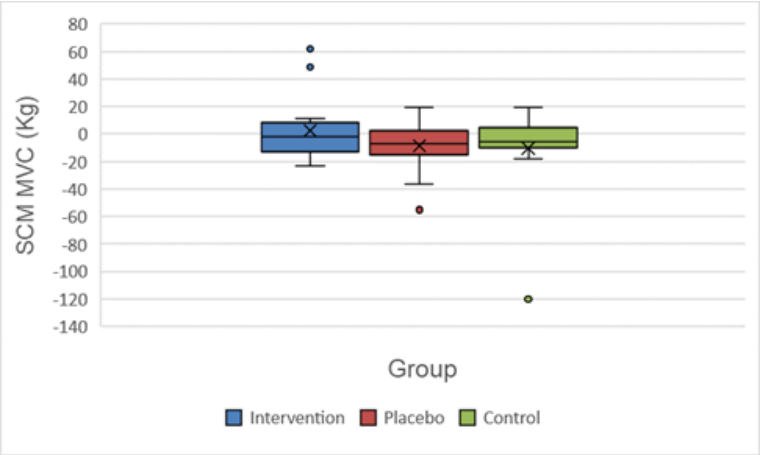


Figure 4.10: Intergroup analysis of the force output measures for the SCM between the three groups

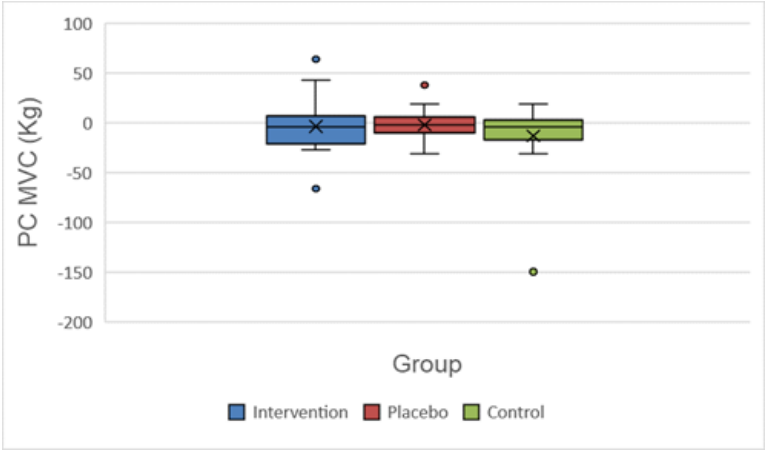


Figure 4.11: Intergroup analysis of the force output measures for the PC between the three groups

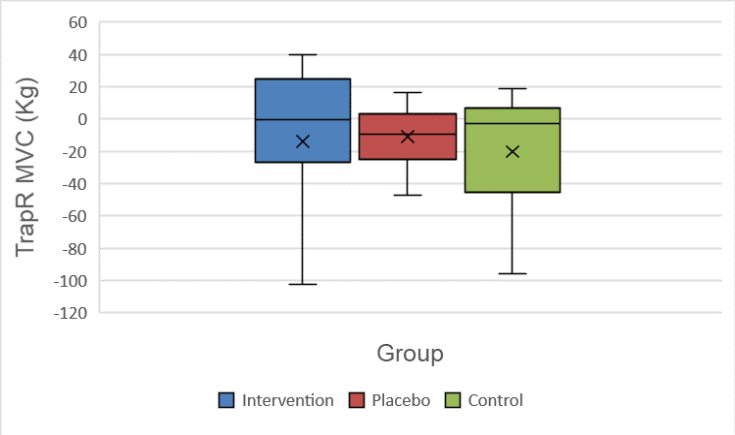


Figure 4.12: Intergroup analysis of the force output measures for the TrapR between the three groups

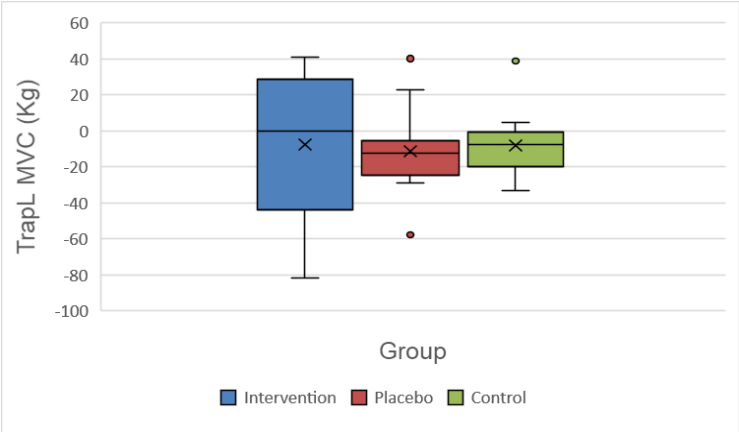


Figure 4.13: Intergroup analysis of the force output measures for the TrapL between the three groups

4.5.2 Muscle Activity

There were no significant differences between the three groups at baseline, for measures of muscle activity ($p<0.005$), indicating that the groups were comparable.

4.5.2.1 Intra-Group Analysis for Muscle Activity

- Right SCM (SCMR)

Using the Wilcoxon Signed Rank Test, it was found that the intragroup pre- and post-test measures were not significant for the placebo ($p=0.140$) and control ($p=0.053$) groups, but for the intervention group ($p=0.017$), a significant increase in muscle activity was found post-test, as seen in **Figure 4.14**.

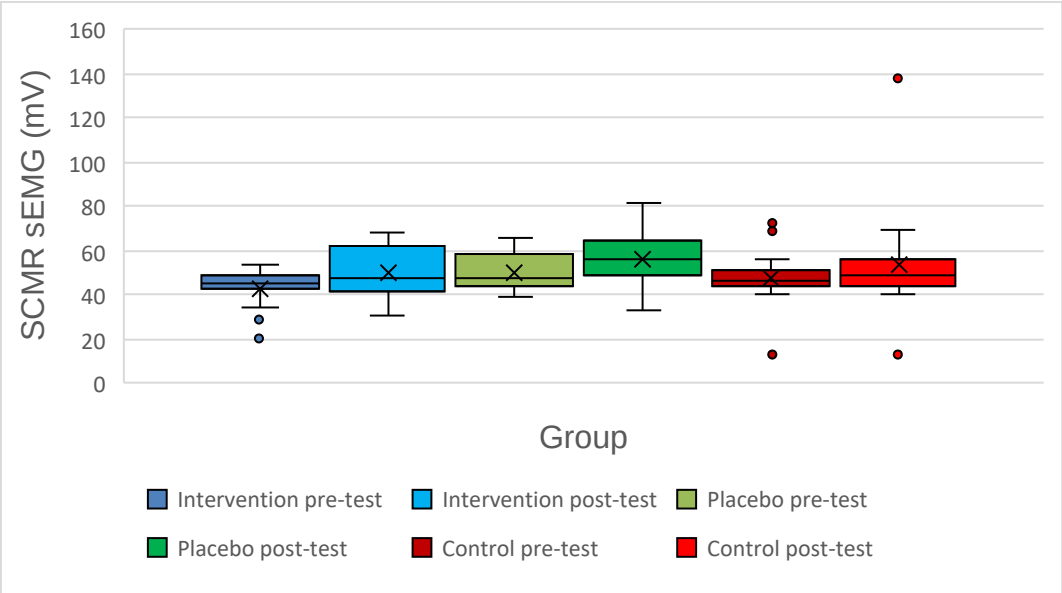


Figure 4. 14: Intragroup analysis of the muscle activity measures for the SCMR in the three groups

- Left SCM (SCML)

Intragroup pre- and post-test measures were not significant for the placebo ($p=0.156$) and control ($p=0.100$) groups, but the intervention group ($p=0.012$) showed a significant increase post-test as seen in **Figure 4.15**. The Wilcoxon Signed Rank Test was used.

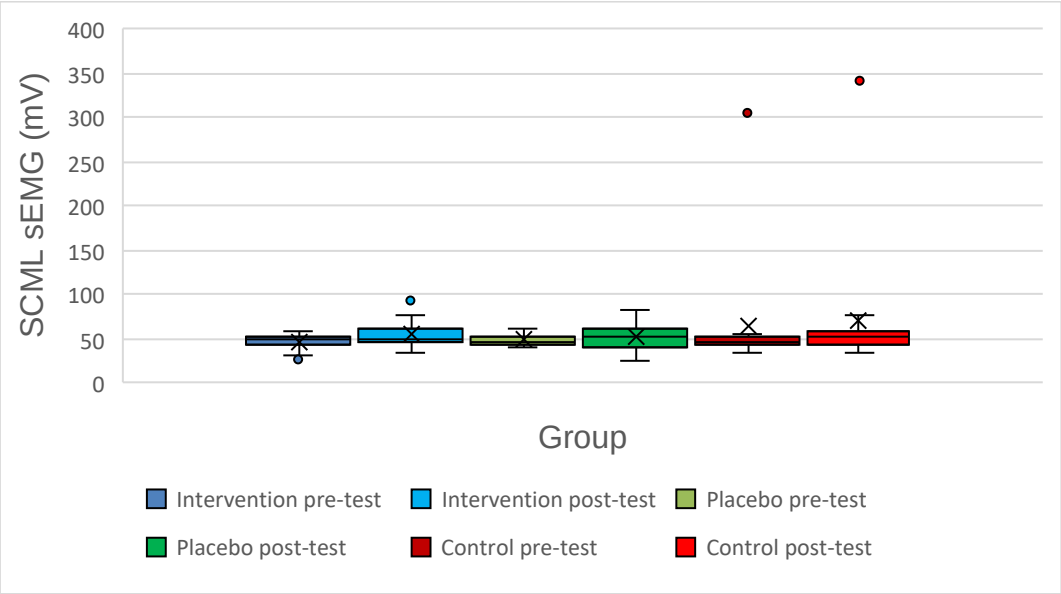


Figure 4. 15: Intragroup analysis of the muscle activity measures for the SCML in the three groups

- Right PC (PCR)

Figure 4.16 shows that there were no significant differences found for the muscle activity readings for the PCR, using the Wilcoxon Signed Rank Test, between the pre-test and post-test measures for the intervention (p= 0.281), placebo (p= 0.496) and control (p= 0.053) groups.

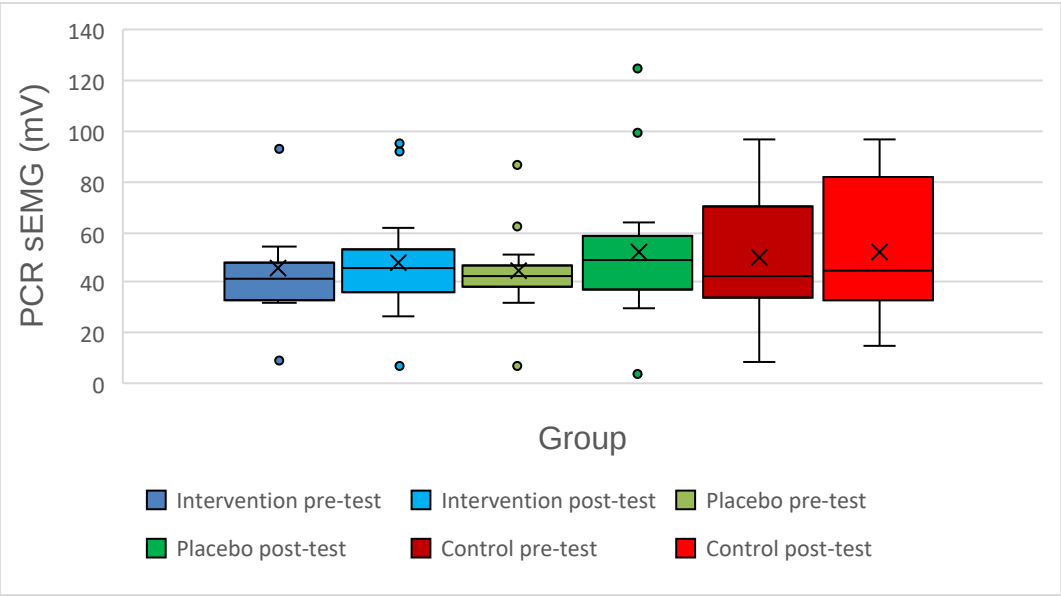


Figure 4.16: Intragroup analysis of the muscle activity measures for the PCR in the three groups

- Left PC (PCL)

Figure 4.17 shows that there was no significant difference, using the Wilcoxon Signed Rank Test, between pre-test and post-test measures with in the intervention (*p*= 0.173), placebo (*p*=0.256) and control (*p*=0.532) groups for muscle activity in the PCL.

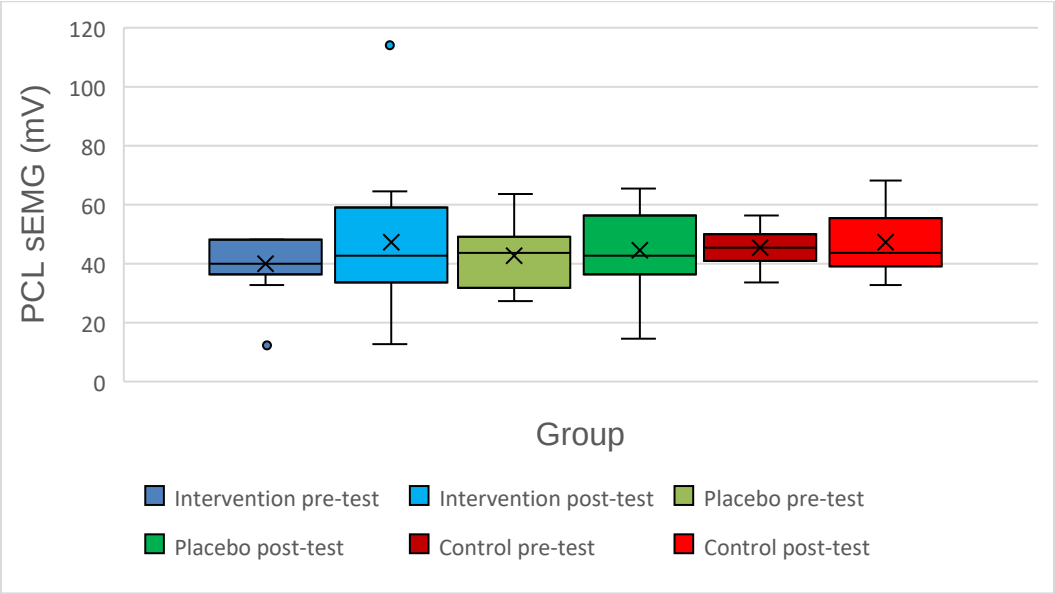


Figure 4. 17: Intragroup analysis of the muscle activity measures for the PCL in the three groups

- TrapR

Using the Wilcoxon Signed Rank Test, intragroup pre- and post-test measures were found not to be significant for the control (*p*=0.427) group, but for the intervention (*p*=0.041) and placebo (*p*=0.031) groups, there was a significant increase at the post-test measure, as seen in **Figure 4.18**.

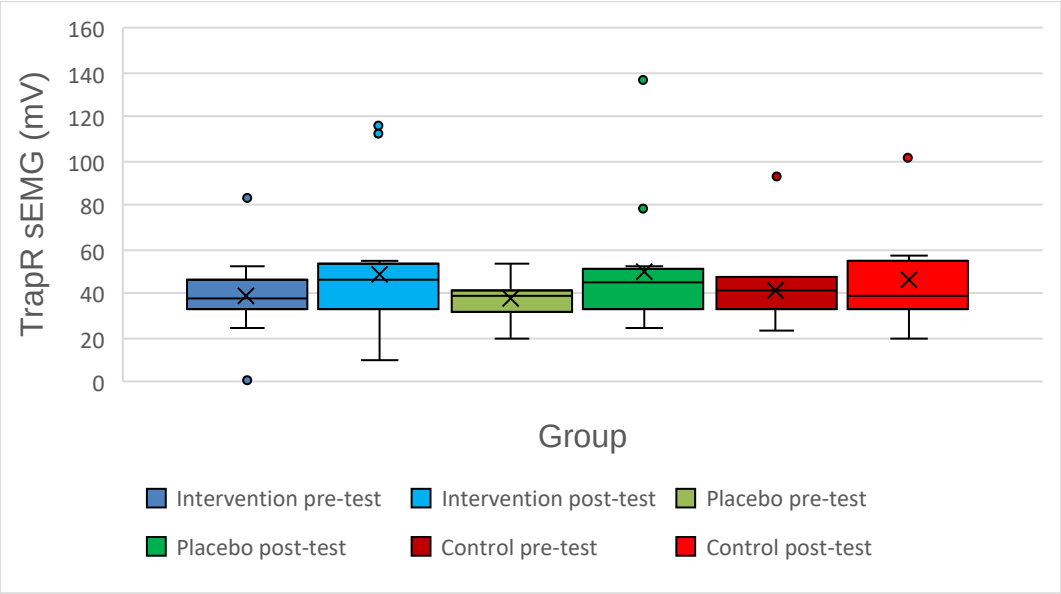


Figure 4. 18: Intragroup analysis of the muscle activity measures for the TrapR in the three groups

- TrapL

Using the Wilcoxon Signed Rank Test, intragroup pre- and post-test measures were not significant for the control (p=0.733) group, but for the intervention (p=0.041) and placebo (p=0.027) groups, a significant increase occurred post-test, as seen in **Figure 4.19**.

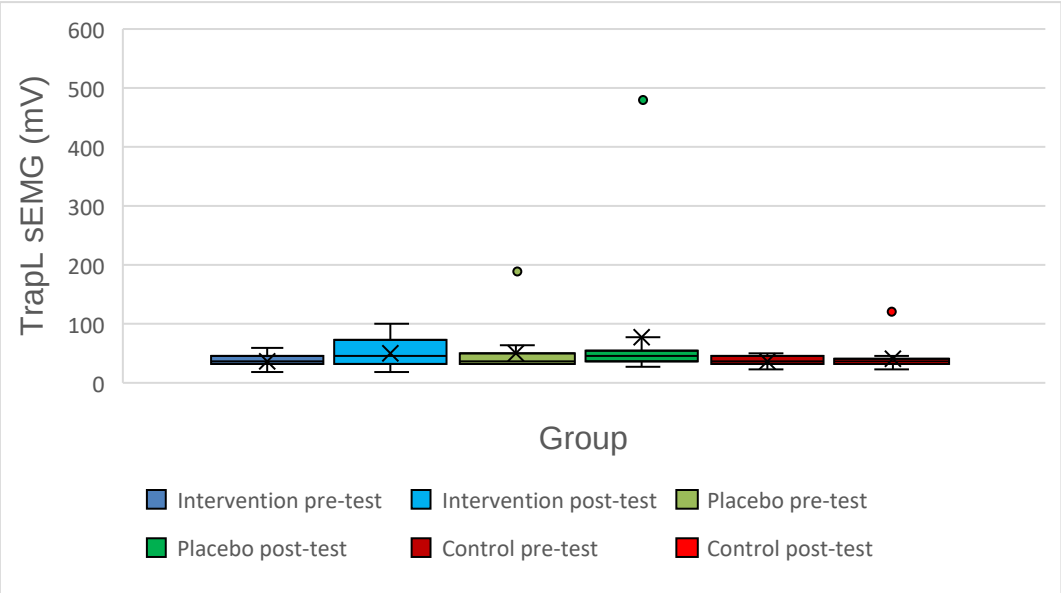


Figure 4.19: Intragroup analysis of the muscle activity measures for the TrapL in the three groups

4.5.2.2 Intergroup Analysis for Muscle Activity

Using the Independent-Samples Kruskal-Wallis Test, no significant differences were found between the three groups for muscle activity readings in the SCMR (p=0.721), SCML (p=0.876), PCR (p=0.870), PCL (p=0.706), TrapR (p=0.388) and TrapL (p=0.372), as seen in **Figures 4.20, 4.21, 4.22, 4.23, 4.24** and **4.25.**

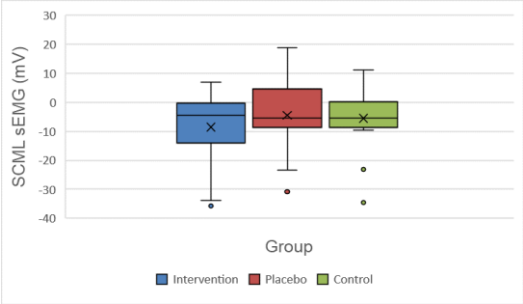


Figure 4.20: Intergroup analysis of the muscle activity measures for the SCML between the three groups

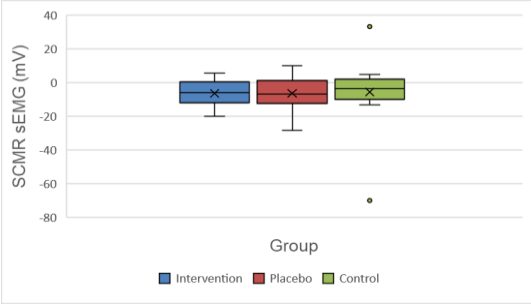


Figure 4.21: Intergroup analysis of the muscle activity measures for the SCMR between the three groups

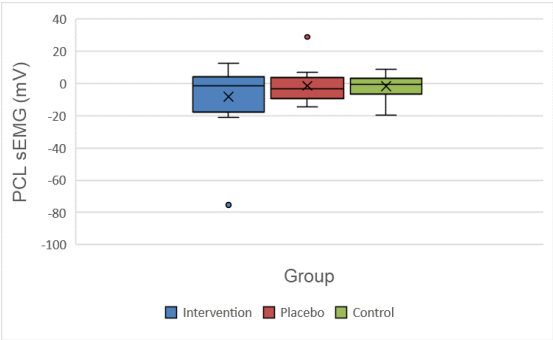


Figure 4.22: Intergroup analysis of the muscle activity measures for the PCL between the three groups

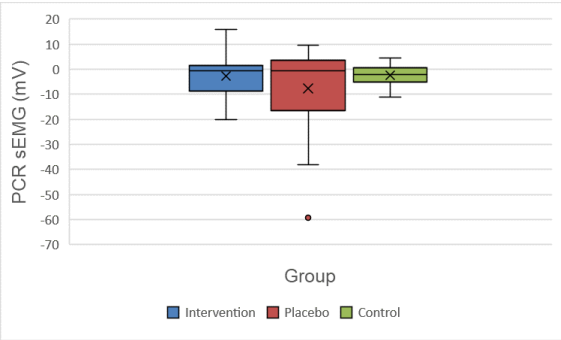


Figure 4.23: Intergroup analysis of the muscle activity measures for the PCR between the three groups

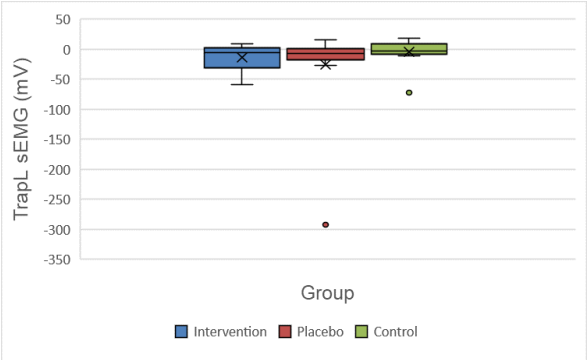


Figure 4.24: Intergroup analysis of the muscle activity measures for the TrapL between the three groups

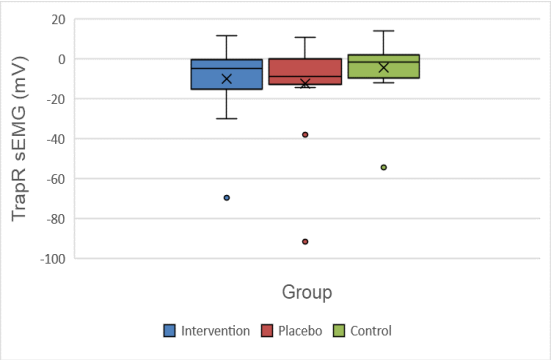


Figure 4.25: Intergroup analysis of the muscle activity measures for the TrapR between the three groups

4.5.2.3 Summary of the Subjective and Objective Measures

The subjective measurements changed over time within groups and the change over time was also significantly higher in the intervention group than the control or placebo. Therefore, the intervention was statistically significant for subjective outcomes of pain and disability. The objective measures showed some changes over time within the intervention, as well as placebo and control groups, but the changes over time were not different between the groups, and thus the intervention effect was equivalent to the control or placebo effect in these outcomes.

CHAPTER FIVE DISCUSSION

5.1 INTRODUCTION

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

5.2 DEMOGRAPHIC CHARACTERISTICS

The age range of the participants in this study was restricted to participants between 18 and 50 years of age. This excluded those under 18 years old, thereby avoiding the need for parental consent. Excluding older participants limited the likelihood of having participants in the study with spinal degeneration and pathology. Age is an important factor in a study measuring muscle activity and force output. The elderly have age-related deficits in neuromuscular functioning which can alter their maximal motor unit firing rate, force steadiness, agonist muscle activation and antagonist muscle co-activation. As a result, the elderly have decreased maximum muscular strength (force output) and increased levels of muscle activity are needed to produce the same force generated by a younger individual, further supporting the need for age to be restricted in this study (Billot *et al.* 2010; Boccia *et al.* 2015).

The mean age in this study was 32.6 years. Biondi (2005) reported a larger mean age of 42.9 years in a population suffering from chronic CGHs. This study's location at a university may have influenced participant recruitment and thus resulted in a lower mean age. Most students are between the ages of 18 to 25 years old. Since both students and staff have to attend the university daily, they may have had easier access to the research premises increasing the convenience to join the study. Therefore, they may have been more likely to respond to the advertisements and attend the consultation. When the three groups were compared at baseline, there were no significant differences (p=0.460) found between them for age, thus mitigating the effect age could have had on the results.

Force output can be affected by gender (Arjunan *et al.* 2011) and activity levels (Disselhorst-Klug *et al.* 2009; Boccia *et al.* 2015). This study did not limit inclusion based on these variables. However, both muscle activity and force output were normalised to mitigate this effect.

In South Africa and abroad, a 4:1 female predominance for CGH sufferers has been reported (Olivier, Pramod and Maleka 2018). In this study, a 3:1 female predominance occurred, supporting the literature that females are more affected by CGHs than men.

Gender may influence muscle activity and force output readings, as the ability of men to perform repetitive maximum force tasks may differ from that of women (Arjunan *et al.* 2011). Although no restriction was placed on gender in this study, it is unlikely gender influenced the results as no significant difference was found between the three groups (p=0.566). There was variability in the data possibly due to the different strengths of the participants and it is recommended that future studies control for this.

Racial diversity is prominent in South Africa, as can be observed from the broad racial and cultural groups represented in this study. A marginally significant difference (p=0.046) between the groups was found for race, yet the impact of this is unclear for the objective measures as there is a paucity of literature documenting if race effects muscle activity and force output.

5.3 SUBJECTIVE MEASURES: PAIN, DISABILITY AND HEADACHE FREQUENCY, DURATION AND INTENSITY

It has been shown that CSM decreases pain and disability as well as headache frequency, duration and intensity in CGH sufferers; however, the mechanism through which this occurs requires further investigation (Chaibi and Russel 2012; Haas *et al.* 2018). It is theorized that CSM brings about its effect through a combination of biomechanical and neurophysiological factors (Gyer 2019). One theory is the gate control theory, which suggests that CSM may alter nociceptive stimuli at a spinal cord level (Melzack and Wall 1965). This involves the large diameter myelinated neurons in mechanoreceptors processing and inhibiting inbound nociceptive stimuli and that CSM can stimulate these mechanoreceptors, thereby facilitating a decrease in pain by activating the spinal gate control mechanism (Fryer *et al.* 2004). Alternatively, it is proposed that CSM works through activating the descending inhibition of pain pathways in the CNS. The CSM activates serotonin and noradrenaline pathways of the descending pain modulation circuit of the CNS, thereby producing a hypoalgesic effect (Chaibi *et al.* 2015; Randoll *et al.* 2017; Gyer 2019).

The intervention group experienced both statistically and clinically significant decreases in pain rating from pre- to post-test (48 hours later). This result is in line with the literature showing CSM can decrease pain associated with CGH (Chaibi and Russel 2012; Hanson 2016; Haas *et al.* 2018). This may indicate that CSM can affect CGH's anatomical and neurophysiological pathophysiology. Firstly, CSM may disrupt the neurophysiological basis of CGH through the descending pain inhibition theory. The release of serotonin and noradrenaline from the pathways of descending pain modulation in the CNS may disrupt

convergence of the afferent input of the upper cervical spine nerve roots (C1-C3) with the afferent tracts of the trigeminal nerve in the trigeminocervical nucleus. This then could facilitate a hypoalgesic effect through inhibiting cervical spine nociceptive input being expressed in the sensory distribution of the trigeminal nerve. Secondly, CSM may disrupt the anatomical basis for CGH through the gate control theory. Mechanoreceptor activation, following CSM of dysfunctional upper cervical facet joints, may inhibit inbound nociceptive stimuli from the first three cervical nerves at a spinal cord level due to the close proximity of these nerves to cervical facet joint mechanoreceptors. Although statistically significant decreases in disability rating from pre- to post-test were experienced by the intervention group, none of the groups experienced a clinically significant decrease in disability rating.

Disability ratings were taken 48 hours after the intervention. It is possible that this may not have been sufficient time to determine the impact of decreased pain rating on disability. The normal pattern of CGH frequency and duration may have also influenced disability rating. In this study, most participants experienced more than one headache per week. Therefore, if a headache occurred during the 48 hour time period, the impact on disability would not have really been noticed in that short time.

CGHs produce chronic pain in its sufferers and therefore it is important to understand the concept of pain mechanisms. Pain mechanisms are broadly categorized into input, processing and output mechanisms. Input mechanisms include nociceptive pain and peripheral neurogenic pain. Processing mechanisms include central pain, central sensitization and the cognitive-affective mechanisms of pain. Output mechanisms include autonomic motor, neuroendocrine and immune systems (Gifford 1998; Cohen *et al.* 2014). It is likely that all three of these pain mechanisms are involved to some degree in the generation of a chronic pain disorder, such as CGHs.

Currently, most of the literature and therapeutic interventions have focused on input mechanisms, such as treating muscles and joints and the output mechanisms of neuromuscular motor control, as was done in this study. There is, however, increasing evidence that processing mechanisms, such as central sensitization and cognitive-affective mechanisms, may play a large role in chronic spinal disorders such as CGHs (Nijs *et al.* 2014).

Central sensitization refers to an amplification of neural signalling within the central nervous system that elicits pain hypersensitivity (Woolf 2011). This mechanism may be responsible for the hypersensitivity of the trigeminal spinal nucleus in CGHs (Chua *et al.* 2011). The cognitive-affective mechanisms of pain refer to an individual's psychological

response to chronic pain. Although in some cases chronic pain stems from an initial painful insult to the body, such as whiplash in CGHs, its progression is associated with the interaction between original injury, CNS, and cognitive-affective dysregulation (Garland *et al.* 2013). Cognitive-affective mechanisms may have been evident in this study as participants in the intervention and placebo group experienced significant decreases in pain. Participants were not screened for central sensitization. Thus, this factor cannot be accounted for in the study's results.

The nature of this study was exploratory and involved only one cervical spine manipulation. This was done in order to determine the immediate effect of CSM on muscle activity and force output of selected cervical spine muscles. Therefore, the normal treatment timelines were not adhered to for CGHs. Current literature suggests a course of three cervical spinal manipulations per week for six weeks (Hanson *et al.* 2016; Haas *et al.* 2018) for effective intervention.

In studies of a clinical nature, it is important to consider the Hawthorne effect and how it may have influenced the clinical significance of the results of the intervention, placebo and control groups. This involves the participants' awareness of being studied and its influence on their behaviour, as this awareness may provoke beliefs about the researcher's expectations, and therefore cause a participant to act in a way that he or she may believe will help the researcher (McCambridge *et al.* 2014). Participants may have reported decreased pain and disability in order to 'help' the research study. In addition, when selected to join the study, the patients were not screened for their previous exposure to SMT. Thus, it is possible that those in the control and placebo groups realised that they were not receiving the active intervention which may have skewed the results.

5.4 OBJECTIVE MEASURES: MUSCLE ACTIVITY AND FORCE OUTPUT

Spinal manipulation is theorized to initiate separation of the articular surfaces and stretching of the joint capsule leading to mechanoreceptor activation and altered afferent stimuli. A change in motor neuron excitability then occurs, with motor neuron stimuli travelling in type I and type II afferent nerve fibres to the posterior horn of the spinal cord. A synapse between the interneuron and afferent neuron then follows, resulting in motor neuron stimulation or inhibition. The motor neuron pool then increases, or decreases, in excitability when motor neuron stimuli reach muscles adjacent to the joint being manipulated. This facilitates an increase (facilitation), or decrease (inhibition), in muscle activity in order to optimize physiological function (changes in force output, pain and

disability) (Dhami and DeBoer 1992; Evans 2002; Pickar 2002; Haavik-Taylor and Murphy 2007; Pickar and Bolton 2012; Gyer 2019). Based on this theory, it was anticipated that CSM would normalize the aberrant muscle activity in the cervical spine, focusing on the upper trapezius, posterior cervicals and SCM muscles. Several studies support this theory. Sterling, Jull and Wright (2001) report that CSM in the treatment of mechanical neck pain decreases muscle activity of the SCM. This is supported by Dishman (2003), who demonstrated that CSM led to reduced muscle activity in the flexor carpi radialis.

In contrast, Dunning and Rushton (2009) report an increase in biceps' brachii resting muscle activity bilaterally post-CSM. DeVocht, Pickar and Wilder (2005) show that during the five to ten minutes after the treatment protocol of manipulation of the lumbar or low thoracic spine, both increases and decreases in muscle activity levels are observed. Therefore, arthrogenic muscle facilitation or inhibition may occur as a result of facet joint dysfunction. Spinal manipulation may disrupt this mechanism by returning aberrant muscle activity and force output to normal physiological functioning (Pickar 2002; Rice and McNair 2010).

In this study, the intra-group's analysis revealed that muscle activity increased bilaterally in the SCM for the intervention group and bilaterally in the trapezius muscles in the placebo and intervention groups only. In addition, force output only increased in the trapezius muscle bilaterally in the control group and in the placebo group, in the left trapezius muscle. These results are line with the findings of deVocht, Pickar and Wilder (2005) and Dunning and Rushton (2009), showing SMT can lead to increased motor neuron pool excitability, increasing muscle excitation.

Surface EMG measures the voltage differential occurring due to the summed effect of the many motor units depolarizing across the muscle at varying distances from the electrode. It is affected by the conduction of the signals through the muscle tissue, the different motor units and their varied firing rates (Hof 1984; Farina *et al.* 2004).

Muscle activity was recorded during a MVC of the included muscles. The manipulation group was the only group that showed intra-group increases in muscles activity in two muscles, via the trapezius and SCM, indicating that these muscles had improved muscle excitation. These findings, however, did not occur simultaneously to increases in force output. Trends observed in this study suggest that CSM, through neurophysiological effects, may alter muscle activity. The CSM group experienced improvements in pain and disability, however how these observed changes in muscle activity may relate to patient reported outcomes of pain and disability, requires further investigation.

When assessing surface EMG, ‘cross talk’ needs to be considered, i.e. signal contribution originating from other muscles (Staudenmann *et al.* 2010). This can be amplified when conducting MVCs (Disselhorst-Klug *et al.* 2009). Similarly, subcutaneous adiposity acts as an insulator between the electrode and muscles. As a result, thinner individuals, with less subcutaneous adipose, may have increased muscle activity amplitude compared to individuals with a thicker subcutaneous adipose layer (Criswell 2010). Skin fold thickness was not controlled in this study. In order to mitigate between individual skin fold thickness and other variations, the muscle activity was normalized (Nordander *et al.* 2003). It is possible that skin fold thickness may have affected the study results as it was not controlled in the participant recruitment.

This study included participants with facet joint restrictions on either the left or the right. The side of restriction, and its effect on muscle activity and force output, was not analysed. It is therefore possible that the side of restriction may have affected the results, as only the side with cervical facet dysfunction received CSM or the sham-intervention. A larger sample size may have allowed subgroup analysis to determine if the side of restriction had any effect on outcome measures; however, this was not implemented due to the small sample size.

Interestingly, it is the effect observed in the placebo group, as they received a sham intervention in which the dysfunctional upper cervical facet joint was taken to the end range of motion and held there for five seconds with no thrust being applied. The placebo group showed increased left trapezius force output and muscle activity and also reported a reduction in pain. It is possible that this procedure mobilized the dysfunctional upper cervical facet joint and may have stimulated cervical mechanoreceptors and thereby lead to increased muscle activity. Lascurain-Aguirrebeña *et al.* (2019) report that cervical mobilization increases the muscle activity of the SCM and upper trapezius. Yet this effect, in this study, was isolated to the trapezius muscle, unlike the intervention group, where both the SCM and trapezius muscles were affected.

Achieving adequate placebo interventions in studies investigating manipulation has been problematic (Koes 2004). The placebo effect occurs when a positive physiological or psychological change occurs with the use of inert medications, sham procedures, or therapeutic symbols during a healthcare encounter (Miller *et al.* 2005). The ‘hands on’ nature of the treatment makes it difficult to mimic. The placebo intervention utilised in this study has been used by Haavik and Murphy (2007; 2008; 2010 and 2012). However, it appears from the results of this study, that it may have had a treatment effect.

The control group in this study also resulted in significant changes in force output, but not muscle activity, in the trapezius muscles. This group remained in the supine position in a resting state, as opposed to the other two groups, where their cervical spines were placed in a position to illicit or mimic a CSM. It could be postulated that a 'learnt effect' may be responsible but all participants were allowed to practice MVC s before the pre-test to ensure accurate execution during testing. Thus, this effect would have occurred across the groups, and would have affected all muscles and not just the trapezius muscle group.

Besides for the intra-group effects observed, there were no significant differences found between the three groups for muscle activity or force output in the posterior cervical, trapezius and SCM muscles bilaterally, in participants with CGHs following the various interventions. Pires *et al.* (2015) find no statistically significant intergroup differences in outcome measures of SCM muscle activity between intervention and control groups in women with chronic neck pain. Similarly, Cardinale *et al.* (2015a) find SMT to have no statistically significant effect on force output of asymptomatic participants. This study was designed to assess if there were effects following CSM on muscles other than the SCM, which has been well explored in the literature (Falla *et al.* 2002; Jull, O'Leary and Falla 2008; Jull and Falla 2016). The results have indicated that further studies into the biomedical and biopsychosocial paradigms are required to determine the role of CSM in reducing pain and disability in CGH suffers.

5.5 POTENTIAL BIAS IN THE STUDY

Bias in a research setting may occur from the randomization process, due to deviations from intended interventions, due to missing outcome data, in measurement of the outcome and in selection of the reported results (Sterne *et al.* 2019). No blinding was applied in this study and the researcher alone was involved in all components of the research procedure. Bias may have occurred during sample allocation as although randomized and concealed from participants, the researcher was aware into which group the participants were randomized into and no research assistant was utilised, however this was mitigated by the fact that, at baseline, no statistically significant differences were found between the three groups for age and gender and measures of pain, disability, muscle activity and force output.

Bias in this study may have also occurred due to deviations from intended interventions. The majority of participants were unaware what group they were allocated into, however some participants were chiropractic students and may have been aware that they were receiving a control or placebo intervention instead of CSM due to their familiarity with spinal manipulation. Only four of the 45 participants were chiropractic students which

somewhat mitigates the potential bias and statistically significant results were still found for the placebo and control groups for measures of muscle activity and force output.

Measurement of subjective and objective measures could have differed between groups as a result of human error and participants failing to follow the researcher's instructions. This is one possible reason for the variability seen in muscle activity and force output data. Furthermore, a high number of statistical tests were utilised in this study, which increases the risk of mistakes occurring when analysing and reporting on the results of the study. Therefore bias may have occurred in measurement of outcomes and in selection of reported results. The research procedure was specific, detailed and followed as closely as possible with all participants, however due to the lack of blinding and a research assistant, bias may have been present in this study.

5.6 CONCLUSION

The results of this study demonstrate that CSM is effective in decreasing CGH pain and disability. Trends suggest that increased muscle activity in the SCM and trapezius muscles during a maximum voluntary contraction show greater muscle activation following CSM. According to the researcher's knowledge, this is the first study investigating the effect of CSM on muscle activity and force output of the SCM, posterior cervical and trapezius in CGH sufferers. Therefore, further research into this field would be recommended. Most of the literature to date has focused on CSM decreasing pain, disability, headache frequency, duration and intensity and the mechanism through which this occurs. Considering that this was a pre-post study, future studies with long-term follow-ups may provide more reliable results about the long-term effectiveness of this type of treatment, taking into account that a CGH is a chronic condition.

CHAPTER SIX CONCLUSION, LIMITATIONS AND RECOMMENDATIONS

6.1 CONCLUSION

The aim of this study was to determine the relative effect of upper CSM, compared to a placebo and a control in terms of pain rating, disability, muscle activity and force output of the trapezius, posterior cervical and sternocleidomastoid muscles in the treatment of CGH. A pre-test post-test design was utilized in this study.

The null hypothesis was rejected in favor of the alternate hypothesis, for the subjective outcomes of pain and disability, in the intervention group, as both had statistically significant intragroup and intergroup decreases when recorded after 48 hours. Clinically, only the intervention group experienced a significant decrease in pain from pre- to post-test. None of the groups experienced a clinically significant decrease in disability rating from pre- to post-test, possibly due to the short duration of the follow up session.

Muscle activity and force output measures showed some statistically significant intragroup increases over time within the intervention, placebo and control groups; however, no significant intergroup differences were found between the three groups in terms of the objective measures. Therefore, the results of this study did not provide sufficient evidence to support the alternate hypothesis that stated that there would be a significant difference ($p<0.05$) between the group receiving CSM and the placebo and control groups in terms of objective outcomes. A trend of effect was observed for the intervention group as muscle activity of the SCM and trapezius increased during a maximum voluntary contraction following CSM. Thus, greater muscle activation was observed in these muscles post-CSM.

Therefore, CSM can be used by manual therapists to assist patients with CGH to have an improvement in their pain. Future studies should elaborate on this design by assessing the observed effects over a period of CSM treatment and to control for central sensitization, gender and skin fold thickness.

6.2 LIMITATIONS

During the study, the following limitations were recognized:

1. Although an *a priori* power analysis for sample size (n=45) was conducted, a larger sample size would allow for variability in the data.
2. Subcutaneous adipose thickness was not controlled in this study and, as it acts as an insulator between electrode and the muscle (Criswell 2010; Cooper *et al.* 2014), it can influence the muscle activity data.
3. The velocity and amplitude of the CSM thrust could not be standardized or verified, although performed by the same researcher. Future studies should include a measure of these variables.
4. Effort was taken for each muscle, to demonstrate the correct position and technique when performing MVC s. The researcher, however, could not control if the participant was contracting at maximum effort due to the subjective nature of participants performing MVC s.
5. The headache diary was only used seven days prior to the study and not during the study. Therefore, the effect of CSM on headache frequency, duration and intensity could not be quantified.
6. This study examined the subjective and objective effects of a single dose of CSM compared to a placebo and a control. Therefore, only the immediate effect of CSM on objective measures was documented. Future studies should assess the effect of CSM over a course of manual therapy treatment.
7. The use of pain medication throughout the study should be monitored.
8. Bias may have been present in this study. No blinding was used and this study lacked a research assistant which could have influenced the research results.
9. The varying duration of headache and the influence of chronic pain and central sensitisation may have influenced the results.

6.3 RECOMMENDATIONS

6.3.1 Recommendations for Future Studies

The following recommendations are made for future studies based on the findings of this study:

1. A larger sample size should be used in future studies in order to further examine the trends observed in this study.

2. A research assistant may be used in future studies in order to remove any potential bias; to deliver the intervention or conduct the subjective and objective measures.
3. A headache diary should be used seven days prior to the study, during and after the study, in order to assess the effect of CSM on headache frequency, duration and intensity.
4. Skin fold thickness should be controlled for in future studies and should be included as part of the participant inclusion criteria.
5. A future study should include the level and side of the joint fixations which were manipulated, allowing for subgroup analysis to improve understanding of the effect of CSM on muscle activity and force output.
6. Future studies should treat the CGH patients over a course of manipulative treatments. The literature indicates that three cervical spinal manipulations per week, for six weeks, is the most effective course (Haas *et al.* 2018).

6.3.2 Recommendations for the Manual Therapy Professions

In an era of evidenced-based medicine, it is prudent that manual therapy professions continue investigating the effect of the treatments they offer. Studies like this one contribute to a greater understanding of a popular treatment modality – spinal manipulation. The evidence suggests that CSM decreases pain in CGH suffers. There is a growing understanding of how these mechanisms work but further research into the exact mechanism, together with an increased understanding of the role of biopsychosocial paradigm, is necessary.

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APPENDICES

Appendix A: Approval from the Clinic Director

MEMORANDUM

To : Prof Adam
Chair: IREC

From : Dr Laura O'Connor
HoD: Chiropractic

Dr Desiree Varatharajullu
Clinic Director: Chiropractic Day Clinic: Chiropractic

Date : 09.05.2019

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

---

Permission is hereby granted to:

Mr William Brann (Student Number: 21349499)

Research title: " The effect of upper cervical spine manipulation, compared to a placebo and a control, on neck muscle activity, pain and disability in participants with cervicogenic headache".

Mr Brann, is requested to submit a copy of his FRC / IREC approved proposal along with proof of his M.Tech:Chiropractic registration 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 his seeing patients.

Thank you for your time.

Kind regards

Dr Laura O'Connor
HoD: Chiropractic

Dr Desiree Varatharajullu
Clinic Director: Chiropractic Day Clinic

Cc: Mrs Linda Twiggs: Chiropractic Day Clinic
Dr A Abdul-Rasheed: Research coordinator
Dr L O'Connor: Supervisor
Dr. Y. Venketsamy: Research co-supervisor

Appendix B: IREC Approval

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

P O Box 1334, Durban, South Africa, 4001

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

www.dut.ac.za

24 May 2019

Mr W Brann
8 Madura Grove
Westridge
Berea

Dear Mr Brann

The relative effect of upper cervical spine manipulation, placebo and a control, on neck muscle activity, pain and disability in participants with cervicogenic headache.

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

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

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

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

Yours Sincerely

Professor J K Adam
Chairperson: IREC

DUT DURBAN UNIVERSITY OF TECHNOLOGY
2019 -05- 24
INSITUTUTIONAL RESEARCH ETHICS COMMITTEE
P O BOX 1334 DURBAN 4000 SOUTH AFRICA

Appendix C: Advertisement

Do you suffer from Headaches?

Are you between the ages of 18-50?

Research is being carried out at the Durban University of Technology Chiropractic Day Clinic and you may be eligible for

treatment!!

For more information contact William at 0786639934 or the DUT Chiropractic clinic at 0313732205 for more information.

Appendix D: Case History

DUT DURBAN UNIVERSITY OF TECHNOLOGY | DEPARTMENT OF CHIROPRACTIC AND SOMATOLOGY

CHIROPRACTIC PROGRAMME

CHIROPRACTIC DAY CLINIC CASE HISTORY

Patient: _____ Date: _____

File #: _____ Age: _____

Sex: _____ Occupation: _____

Student: _____ Signature _____

FOR CLINICIANS USE ONLY:

Initial visit

Clinician: Signature:

Case History:

Examination:

Previous: Current:

X-Ray Studies:

Previous: Current:

Clinical Path. lab:

Previous: Current:

CASE STATUS:

PTT: Signature: Date:

CONDITIONAL:

Reason for Conditional:

Signature: Date:

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

Case Summary signed off: Date:

Page 1 of 4

Student's Case History:

1. **Source of History:**

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

3. **Present Illness:**

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

4. **Other Complaints:**

5. **Past Medical History:**

General Health Status

Childhood Illnesses

Adult Illnesses

Psychiatric Illnesses

Accidents/Injuries

Surgery

Hospitalizations

6. Current health status and life-style:

Allergies

Immunizations

Screening Tests incl. x-rays

Environmental Hazards (Home, School, Work)

Exercise and Leisure

Sleep Patterns

Diet

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

Tobacco
Alcohol
Social Drugs

7. Immediate Family Medical History:

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

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

8. Psychosocial history:

Home Situation and daily life
Important experiences
Religious Beliefs

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

General

Skin

Head

Eyes

Ears

Nose/Sinuses

Mouth/Throat

Neck

Breasts

Respiratory

Cardiac

Gastro-intestinal

Urinary

Genital

Vascular

Musculoskeletal

Neurologic

Haematological

Endocrine

Psychiatric

Appendix E: Physical Examination

DUT DURBAN UNIVERSITY OF TECHNOLOGY

DEPARTMENT OF CHIROPRACTIC AND SOMATOLOGY

CHIROPRACTIC PROGRAMME

PHYSICAL EXAMINATION: SENIOR

Patient Name: _____ **File no:** _____ **Date:** _____

Student: **Signature:**

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

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

SYSTEM SPECIFIC EXAMINATION:

CARDIOVASCULAR EXAMINATION

RESPIRATORY EXAMINATION

ABDOMINAL EXAMINATION

NEUROLOGICAL EXAMINATION

COMMENTS

Clinician: **Signature:**

Page 1

Appendix F: Cervical Regional Exam

DUT DURBAN UNIVERSITY OF TECHNOLOGY — DEPARTMENT OF CHIROPRACTIC

REGIONAL EXAMINATION – CERVICAL SPINE

Patient: ..... File No: .....

Date: ..... Student: .....

Clinician: ..... Sign: .....

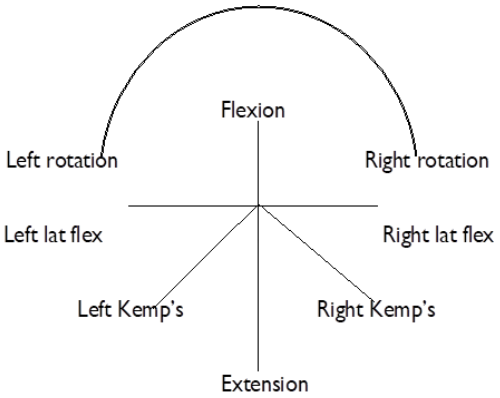
OBSERVATION:

Posture
Swellings
Scars, discolouration
Hair line
Body and soft tissue contours

Shoulder position
Left:
Right:
Shoulder dominance (hand):
Facial expression:

RANGE OF MOTION:

Extension (70°):
L/R Rotation (70°):
L/R Lat flex (45°):
Flexion (45°):



PALPATION:

Lymph nodes
Thyroid Gland
Trachea

MYOFASCIAL ASSESSMENT

Tenderness		Right	Left
Trigger Points:	SCM		
	Scalenii		
	Post Cervicals		
	Trapezius		
	Lev scapular		

ORTHOPAEDIC EXAMINATION:

	Right	Left		Right	Left
Adson's test			Halstead's test		
Brachial plexus test			Hyper-abduction test		
Cervical compression			Kemp's test		
Cervical distraction			Lateral compression		
Costoclavicular test			Lhermitte's sign		
Dizziness rotation test			Shoulder abduction test		
Doorbell sign			Shoulder compression test		
Eden's test					

Page 1 of 2

NEUROLOGICAL EXAMINATION:

Dermatones	Left	Right	Myotomes	Left	Right	Reflexes	Left	Right
C2			C1			C5		
C3			C2			C6		
C4			C3			C7		
C5			C4					
C6			C5					
C7			C6					
C8			C7					
T1			C8					
			T1					
Cerebellar tests:			Left		Right			
Dysdiadochokinesis								

VASCULAR:	Left	Right		Left	Right
Blood pressure			Subclavian arts.		
Carotid arts.			Wallenberg's test		

MOTION PALPATION & JOINT PLAY:

Left: Motion Palpation:
Joint Play:
Right: Motion Palpation:
Joint Play:

BASIC EXAM: SHOULDER:

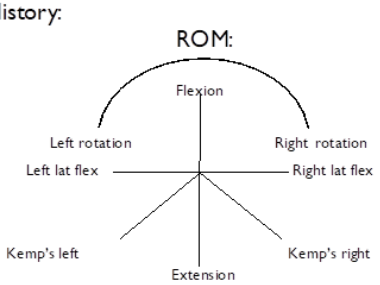
Case History:

ROM: Active:
Passive:
RIM:
Orthopaedic:
Neuro:
Vascular:

BASIC EXAM: THORACIC SPINE:

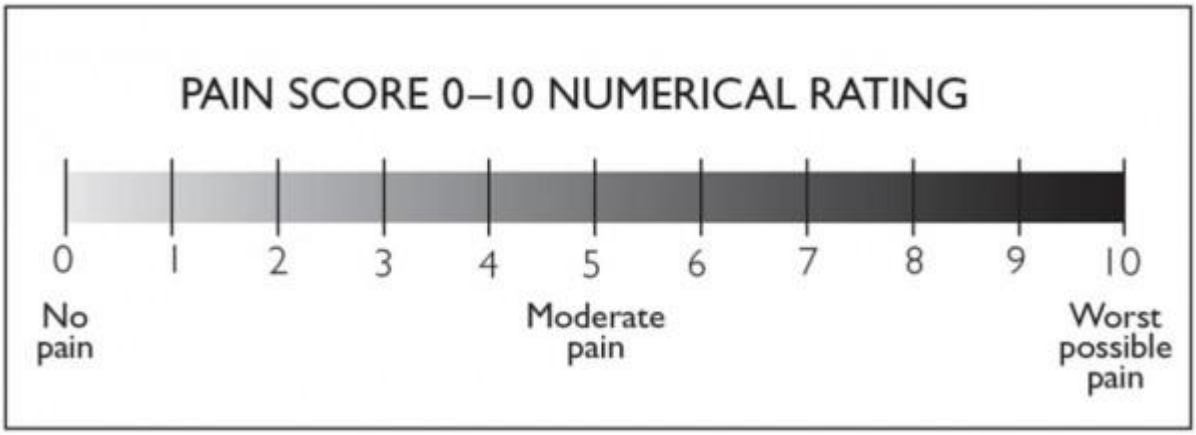
Case History:

ROM:



Motion Palpation:	
Orthopaedic: Neuro:	
Vascular:	
Observ/Palpation:	
Joint Play:	

Appendix G: Numerical Pain Rating Scale



Appendix H: Headache Disability Index

HEADACHE DISABILITY INDEX

NAME: _____ DATE: _____ AGE: ____ SCORES TOTAL: ____; E____; F____
(100) (52) (48)

INSTRUCTIONS: Please CIRCLE the correct response:

1. I have headache: [1] 1 per month [2] more than but less than 4 per month [3] more than one per week.
2. My headache is: [1] mild [2] moderate [3] severe

INSTRUCTIONS: ***PLEASE READ CAREFULLY:*** The purpose of the scale is to identify difficulties that you may be experiencing because of your headache. Please check off "YES", "SOMETIMES", or "NO" to each item. Answer each item as it pertains to your headache only.

	YES	SOMETIMES	NO
E1. Because of my headaches I feel handicapped.			
F2. Because of my headaches I feel restricted in performing my routine daily activities.			
E3. No one understands the effect my headaches have on my life.			
F4. I restrict my recreational activities (e.g. sports, hobbies) because of my headaches			
E5. My headaches make me angry.			
E6. Sometimes I feel that I am going to lose control because of my headaches			
F7. Because of my headaches I am less likely to socialize.			
E8. My spouse/significant other, or family and friends have no idea what I am going through because of my headaches.			
E9. My headaches are so bad that I feel I am going to go insane.			
E10. My outlook on the world is affected by my headaches.			
E11. I am afraid to go outside when I feel a headache is starting.			
E12. I feel desperate because of my headaches.			
F13. I am concerned that I am paying penalties at work or at home because of my headaches.			
E14. My headaches place stress on my relationships with family or friends.			
F15. I avoid being around people when I have a headache.			
F16. I believe my headaches are making it difficult for me to achieve my goals in life.			
F17. I am unable to think clearly because of my headaches.			
F18. I get tense (e.g. muscle tension) because of my headaches.			
F19. I do not enjoy social gatherings because of my headaches.			
E20. I feel irritable because of my headaches.			
F21. I avoid traveling because of my headaches.			
E22. My headaches make me feel confused.			
E23. My headaches make me feel frustrated.			
F24. I find it difficult to read because of my headaches.			
F25. I find it difficult to focus my attention away from my headaches and on other things.			

Reference: Jacobson Gary P., Ramadan NM, et al., The Henry Ford Hospital Headache Disability Inventory (HDI). Neurology 1994; 44:837-842
FORM 502

Appendix I: Permission to use HDI

Dear Steven

I know we have asked permission before for the use of the attached HDI and you had responded indicating that you were happy with a master's student utilising this questionnaire for purposes of their research study.

However, for formality, we are required to ensure that the student has requisite permission and has attempted to seek permission.

In this case Mr William Brann is looking to investigate the effect of upper cervical spine manipulation compared to placebo and a control on neck muscle activity, pain and disability in participants with cervicogenic headache.

Your assistance is very much appreciated

Charmaine

Dr Charmaine Korporaal

M.Tech: Chiropractic, CCFC, CCSP, ICSSD

Registered Chiropractor

Senior lecturer: Chiropractic Programme and Clinic Director: Chiropractic Day Clinic

Durban University of Technology, P.O. Box 1334, Durban, 4000

Tel: +27 31 3732611

Fax: + 27 866486360

E-mail: charmak@dut.ac.za

National Co-ordinator: Chiropractic Internship

Member: ICRC

Hi Dr Korporaal:

In review of the version of the HA Q attached to your email, it is formatted differently from the Henry Ford / Gary Jacobson 1994 referenced questionnaire. Your version includes a severity grading system that I have only seen published for the Oswestry/NDI tools but using different cut-offs.

Do you have a reference (or know its origin) for the severity grading system that is included on the bottom of the HDI tool you have attached above?

If desired, I'm attaching a version that more replicates what I have published in my text which includes the Jacobson reference at the bottom. It might be wise to use this version with the reference at the bottom since this is what Gary Jacobson granted permission to me to share.

Thanks, and please let me know if you have any questions.

Steve

Appendix J: Headache diary

Headache Diary

Participant Code: _____

There are two questions that need to be completed. Please complete the following questions for the next 9 days.

Day: _____ Date:_____

Question 1

The time chart below, represented in the form of blocks, shows one complete day from 6:00am to 6:00am the next day. Each block represents 30minutes. If you get a headache, please mark in the time block below when the headache occurred.

Original version

Day:

6:00 am	6:30 am	7:00 am	7:30 am	8:00 am	8:30 am	9:00 am	9:30 am	10:00 am	10:30 am	11:00 am	11:30 am	12:00 noon	12:30 pm	1:00 pm	1:30 pm	2:00 pm	2:30 pm	3:00 pm	3:30 pm	4:00 pm	4:30 pm	5:00 pm	5:30 pm

Night:

6:00 pm	6:30 pm	7:00 pm	7:30 pm	8:00 pm	8:30 pm	9:00 pm	9:30 pm	10:00 pm	10:30 pm	11:00 pm	11:30 pm	12:00 pm	12:30 am	1:00 am	1:30 am	2:00 am	2:30 am	3:00 am	3:30 am	4:00 am	4:30 am	5:00 am	5:30 am

Question 2

How much did your headache hurt, or how strong was the pain? Please mark a box below:

Mild	Moderate	Fairly severe	Severe

Appendix K: Trial Registration

19 June 2019

To Whom It May Concern:

RE: The relative effect of upper cervical spine manipulation, placebo and a control, on neck muscle activity, pain and disability in participants with cervicogenic headache.

As project manager for the Pan African Clinical Trial Registry (www.pactr.org) database, it is my pleasure to inform you that your application to our registry has been accepted. Your unique identification number for the registry is **PACTR201906717506525**.

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

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

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

Yours faithfully,

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

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

Appendix L: Table of Cervical muscles

Summary of relevant cervical muscles (Moore, Dalley and Agur, 2014 and Middelditch and Oliver, 2005).

Muscle	Attachment	Innervation	Action
Sternocleidomastoid	Lateral surface of mastoid process of temporal bone and lateral half of the superior nuchal line. Sternal head attaches to anterior surface of manubrium of sternum and clavicular head on superior surface of medial third of clavicle.	Motor supply by the accessory nerve; sensory supply ventral rami C2 and C3	Extend or rotate or laterally flex the head and neck; flex or laterally flex the neck
Trapezius	Medial third of superior nuchal line, external occipital protuberance, nuchal ligament, spinous processes of C7-T12 and lumbar and sacral spinous processes attaching to lateral	Motor supply by the accessory nerve; sensory supply ventral rami of C3 and C4	Laterally flex head and neck, with shoulders fixed; bilateral contraction extends the neck; unilateral contraction produces lateral flexion to the same side
Obliquus capitis inferior	Proximally to the transverse processes of the atlas onto the spinous process of axis and adjacent lamina.	Dorsal ramus of C1	Rotate the head on the neck
Obliquus capitis superior	Proximally between the superior and inferior nuchal lines of occipital bone onto the transverse processes of the atlas.	Dorsal ramus of C1	Extend the head on the neck
Rectus capitis posterior major	Spinous process of axis ascending to lateral part	Dorsal ramus of C1	Extend the head on the neck

	of inferior nuchal line.		
Rectus capitis posterior minor	Tubercles on the posterior arch of atlas ascending to medial part of inferior nuchal line.	Dorsal ramus of C1	Extend the head on the neck
Semispinalis cervicis	Transverse processes of T1-T5 and attaching to spinous processes of C2-C5.	Dorsal rami of adjacent cervical spinal nerves	Rotate the neck
Splenius capitis	Lateral aspect of mastoid process and lateral third of superior nuchal line to the inferior half of nuchal ligament and spinous processes of T1-T6.	Dorsal rami of C3-C5	Extend or rotate or laterally flex head and neck
Splenius cervicis	Spinous processes of T3-T6 passing upwards to attach to the posterior tubercles of the transverse process of C1-C3.	Dorsal rami of C5-C7	Extend or rotate or laterally flex head and neck
Longus coli	Anterior tubercle of atlas, bodies of C1-C3 and transverse processes of C3-C6 descending to bodies of C5-T3 and transverse processes of C3-C5.	Ventral rami of C3, C4, C5 and C6	Flex the neck

Appendix M: Letter of Information

Letter of information

Dear Participant,

Thank you for your interest in this research study.

Title of study: The relative effect of upper cervical spine manipulation, placebo and a control, on neck muscle activity, pain and disability in participants with cervicogenic headache.

Principle investigator: William Brann (B Tech Chiropractic)

Co-investigators/Supervisors:

Dr L. O'Connor (M.Tech Chiropractic)

Dr Y. Venketsamy (M.Tech Chiropractic)

Brief introduction and purpose of this study: You have been selected to participate in a study to investigate the effects of cervical spine manipulation on pain, disability and muscle activity of the neck in participants who suffer from cervicogenic headaches. The surface electromyography (sEMG) machine will be used to assess the electrical activity (excitability) of the muscles, while the strength will be measured using a dynamometer.

Procedure: You will be required to attend an appointment at the Durban University of Technology during which you will be randomly put into one of three groups (either the intervention, control or placebo group). Please note you have a one in three chance of being allocated into the intervention group. Prior to the first consultation, participants will be required to record their headache intensity, frequency and duration in a headache diary. A full history, physical exam and regional evaluation of your neck will be performed by the researcher. Anything found that contraindicates treatment will be noted by the researcher, and you will be excluded from the study. If no contraindications are found, you will then be screened in an effort to identify any fixations in the cervical spine. The researcher will then take the time to explain to you how the sEMG and dynamometer works. You will be allocated time to practice the movements you will be asked to perform. Three pre-tests for all muscles will then be taken. The intervention will then be performed and three post-tests for each muscle will be done. The data will be recorded and securely filed. Participants will be phoned 48 hours later to rate their pain and disability.

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

Benefits and Remuneration: 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 increasing the efficacy of treatment provided for cervicogenic headaches. On completion of your participation you will be eligible for a voucher for one free treatment at the chiropractic day clinic (CDC) at the Durban University of Technology. The voucher is only valid for three months.

Reasons why the participant may be withdrawn from the study: If you present with contraindications to the usage of the surface electromyography machine (sEMG) including, but not

limited to: open wounds, rashes, psoriasis, skin irritation's, or skin conditions of any kind in the region of electrode placement (the_researcher will point out these locations); you will be excluded from the study. You may not receive any form of chiropractic manipulative treatment on your neck during the study (including chiropractic students.) If you do, you will be withdrawn from the study. Participants suffering from other types of headaches such as migraine and tension type headaches will be removed from the study.

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

Confidentiality: 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 made available.

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

Persons to Contact in the Event of Any Problems or Queries: Please contact the researcher (0786639934), my supervisor, Dr Laura O'Connor (084 848 0620) or the Institutional Research Ethics administrator (Ms Lavisha Deonarain) on 031 373 2375 or lavishad@dut.ac.za. Complaints can be reported to the DVC: Prof C Napier on 031 373 2326/2577 or carinn@dut.ac.za.

Appendix N: Informed Consent

CONSENT

Statement of Agreement to Participate in the Research Study:

I hereby confirm that I have been informed by the researcher, William Brann, about the nature, conduct, benefits and risks of this study - Research Ethics Clearance Number: 032/19

I have also received, read and understood the above written information (Participant Letter of Information) regarding the study.

I am aware that the results of the study, including personal details regarding my sex, age, date of birth, initials and diagnosis will be anonymously processed into a study report.

In view of the requirements of research, I agree that the data collected during this study can be processed in a computerized system by the researcher.

I may, at any stage, without prejudice, withdraw my consent and participation in the study.
I have had sufficient opportunity to ask questions and (of my own free will) declare myself prepared to participate in the study.

I understand that significant new findings developed during the course of this research which may relate to my participation will be made available to me.

Full Name of Participant	**Date /**	**Time**	**Signature**
Thumbprint		**Right**	

I, William Brann

hereby confirm that the above participant has been fully informed about the nature, conduct and risks of the above study.

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

Appendix O: Voucher

DUT DURBAN UNIVERSITY OF TECHNOLOGY

DEPARTMENT OF CHIROPRACTIC

This voucher entitles the bearer to one free chiropractic treatment session at the DUT Chiropractic Day Clinic. This voucher is valid for six months following the day of research participation.

Date of participation:_____

Expiry date:_____

DUT DURBAN UNIVERSITY OF TECHNOLOGY

DEPARTMENT OF CHIROPRACTIC

This voucher entitles the bearer to one free chiropractic treatment session at the DUT Chiropractic Day Clinic. This voucher is valid for six months following the day of research participation.

Date of participation:_____

Expiry date:_____

DUT DURBAN UNIVERSITY OF TECHNOLOGY

DEPARTMENT OF CHIROPRACTIC

This voucher entitles the bearer to one free chiropractic treatment session at the DUT Chiropractic Day Clinic. This voucher is valid for six months following the day of research participation.

Date of participation:_____

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DUT DURBAN UNIVERSITY OF TECHNOLOGY

DEPARTMENT OF CHIROPRACTIC

This voucher entitles the bearer to one free chiropractic treatment session at the DUT Chiropractic Day Clinic. This voucher is valid for six months following the day of research participation.

Date of participation:_____

Expiry date:_____

Appendix P: Permission to place advertisement

Letter requesting permission to place advertisements

To whom it may concern

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

Title of study: The relative effect of upper cervical spine manipulation, placebo and a control, on neck muscle activity, pain and disability in participants with cervicogenic headache.

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

Statement for permission to place advertisements:

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

Name.....

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

Researcher's name.....

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

Witness' name.....

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

Appendix Q: Clinical significance for pain and disability

Clinical significance was determined for pain rating as a decrease of 1.3 in pain perception and for disability rating as a 29-point change (95% confidence interval) or greater in the disability total score from test-retest as shown in **Table 5**.

Table 5: Mean difference (pre minus post-test) in pain and disability ratings from pre to post-test per group

	CSM	**Placebo**	**Control**
Pain	1,87	0,57	0,13
Disability	11,6	4,1	1,53