The effect of spinal manipulation on biceps brachii muscle activity

By

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Dissertation submitted in partial compliance with the requirements for the Master's Degree in Technology: Chiropractic

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

…… Date: ……

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..... Date: ……

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DEDICATION

I dedicate this dissertation to:

My parents, Hannes and Jakkie Victor, and my brother, Ian Victor.

Thank you for your love, support and encouragement throughout my academic career. I am who I am today because of your influence and support.

My savior, the Lord Jesus Christ, “For God so loved the world, that He gave his only begotten Son, that whosoever believeth in Him should not perish, but have everlasting life”.

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ABSTRACT

Background:

The electromyographic response post-spinal manipulation may support the elucidation of the underlying neurophysiological mechanism of spinal manipulation on motor activity as well as on arthrogenic muscle inhibition. The literature shows conflicting evidence regarding the excitatory or inhibitory nature of the reflexive electromyographic response and the excitability of the homonymous motor neuron pool post-spinal manipulation. The current study investigated whether the electromyographic response post-spinal manipulation is affected by a facilitated golgi tendon organ Ib inhibitory di-synaptic spinal reflex as part of the convergent input on the homonymous motor neuron pool excitability.

Objectives:

The objectives of this placebo-controlled, single-blinded, repeated measures design were: 1) to determine electrical activity and muscle force of the biceps brachii muscle immediately before and after an Activator Adjusting II Instrument placebo spinal manipulation, 2) to determine electrical activity and muscle force of the biceps brachii muscle immediately before and after a C5/C6 spinal manipulation, 3) to compare the electrical activity and muscle force of the biceps brachii muscle between the control and intervention groups pre- and post-test.

Method:

Each participant performed three sets of modified stretching of the biceps brachii muscle with two minute rest intervals between each set in a single appointment, of which at a standardized fourth second during each set an intervention was applied to the ipsilateral C5/C6 segment. The first intervention (AAI 1) entailed the application of an Activator II Adjustment Instrument placebo spinal manipulation; the second intervention (AAI 2) entailed the application of an Activator II Adjustment Instrument placebo spinal manipulation; and the third intervention (SMT) entailed the application of

spinal manipulation. One-second electromyography (EMG) segments were taken during the force plateau of each set; the EMG signal was processed through Root Mean Square (RMS) analysis and the muscle force data were obtained by using the Biopac - MP 150 Data Acquisition system and AcqKnowledge analysis software.

Results:

The objective data analysis revealed a noteworthy scientific finding of a medical anomalous inverse relationship between the muscle force and EMG RMS immediately post-spinal manipulation. The immediate post-SMT intervention revealed an increase in the biceps brachii muscle force by 4.76 % and a decrease in the biceps EMG RMS by 9.03 % with a summation of percentage difference between the muscle force and EMG RMS of 13.79 %. The immediate post-placebo AAI 1 intervention showed a decrease in the biceps EMG RMS by 1.86 % and a decrease in the biceps brachii muscle force by 0.85 % with a summation of percentage difference between the muscle force and EMG RMS of 1.01 %. The immediate post-placebo AAI 2 intervention showed a decrease in the biceps EMG RMS by 0.05 % and a decrease in the biceps brachii muscle force by 1.97 % with a summation of percentage difference between the muscle force and EMG RMS of 1.92 %.

Conclusion:

Further research is warranted to add statistical significance to the inverse relationship between muscle force and EMG RMS observed immediately post-spinal manipulation. This knowledge obtained, may have clinical relevance for rehabilitation practitioners and physical therapists by providing evidence based support for the suggestion that optimal management of patients with muscle weakness suspected to be of arthrogenic nature could include the application of spinal manipulation to the segmentally innervated facet joints before traditional strength training is initiated.

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LIST OF DEFINITIONS

Acetycholine Acetycholine (Ach) is the major excitatory neurotransmitter in the peripheral nervous system. A cholinergic synapse is a chemical synapse in which ACh is the predominant ligand released (Lundy-Ekman 2013: 53; Siegel *et al*. 2010: 98).

Action potential A generated action potential or known as a nerve impulse is a brief all-or-nothing reversal in neuron membrane potential at the trigger zone (Lundy-Ekman 2013: 31, 33; Siegel *et al*. 2010: 99).

Biceps brachii The biceps brachii muscle is a musculoskeletal muscle. This muscle flexes the elbow joint and aids in shoulder flexion and consists of a long and short head. The short head attaches to the coracoid process and the long head to the supraglenoid tubercle of the scapula. Both heads insert into the radial tuberosity of the radius and the deep fascia overlying the flexor tendon. The biceps brachii is innervated by the musculocutaneous nerve originating from spinal levels C5-C6 (Diogo *et al.* 2013: 42; Muscolino 2008: 265). The brachialis muscle serves as the synergist and the triceps brachii muscle serves as the antagonist of the biceps brachii (Saladin 2007: 260).

Cyclic adenosine monophosphate system Cyclic adenosine monophosphate system is a G-protein secondary messenger system responsible for phosphorylation of proteins (Fitzgerald *et al*. 2012: 94, 95).

Central sensitization Central sensitization is a form of activity-dependent synaptic neuroplasticity and manifests as an increase in responsiveness of nociceptive neurons in the central nervous system (spinal cord and supraspinal areas) to their normal or subthreshold afferent input (Jones *et al*. 2013: 206; Simpson *et al.* 2012: 18; Malcangio 2009: 230).

Cervical facet joints Facet joints are diarthrotic joints formed by the articulation of the superior and inferior articular process of adjacent vertebrae. The facet joint is composed of a fluid-filled space bordered by hyaline cartilage over the subchondral bone and is enclosed by a synovial membrane and a thin fibrous capsular ligament known as the facet joint capsule or facet joint capsular ligament. Invaginations of the facet joint capsule form synovial

folds or menisci in the synovium deep to the facet joint capsule which fill voids, absorb shock, distribute pressure and maintain stability during facet joint articulation (Depalma 2011: 80). The facet joint capsular tissue and synovium contains mechanoreceptors and nociceptors and is well innervated by the medial branch of the dorsal rami. Each medial branch segmentally innervates two adjacent facet joints, thus providing dual innervation (Waldman 2009: 161; Manchikanti *et al.* 2002: 243). The facet joints in the cervical spine are orientated predominantly at 45 degrees in the sagittal plane and zero degrees in the coronal plane (Thamburaj 2012: 133).

Chiropractic The diagnosis and management of neuromusculoskeletal disorders with the therapeutic administration of manipulative therapy (Vernon 2010: 24; Millan *et al*. 2012: 24; Pickar 2002: 358, 359)

Depolarization The membrane potential of a neuron decreases to below -70 mV (about -50 mV or more) by the binding of an excitatory ligand (Fitzgerald *et al.* 2012: 86). An excitatory neurotransmitter can bind to and open ligand-gated sodium channels and/or ligand-gated calcium channels to cause an influx of Na^+ ions and /or Ca^{2+} ions into the target neuron to which they bind to cause depolarization of the target neuron membrane and result in graded potentials or an excitatory post-synaptic potential (Chambers *et al.* 2015: 229; Lundy-Ekman 2013: 49).

Disinhibition Disinhibition entails inhibition of an inhibitory internuncial which in turn causes inhibition of the target neuron by way of the interpolation of an additional inhibitory internuncial (Fitzgerald *et al.* 2012: 77).

End plate potential When depolarization of a sarcolemma of a muscle cell produce end plate potentials (EEPs) that reach the motor end plate threshold of about 70 mV; an action potential will be generated and propagated along the sarcolemma of the muscle fiber to bring about muscle contraction (Siegel *et al*. 2010: 98; Conn 2008: 100, 101).

Excitatory postsynaptic potential Excitatory neurotransmitters bind to their specific ligand-gated ion channels in the receiving zones of the target neuron membrane to produce graded potentials by depolarizing the target neuron membrane. When a generated graded potential reaches the trigger zone of the target neuron, the graded potential is known as an excitatory postsynaptic potential (EPSP). The EPSP can cause the generation of an action potential and firing of the target neuron if the membrane threshold was reached at the trigger zone (Lundy-Ekman 2013: 49; Siegel *et al*. 2010: 100).

Excitation When a neuron forms an excitatory synapse on a target neuron and produce an EPSP in the target neuron (Chambers *et al.* 2015: 229; Lundy-Ekman 2013: 49).

Excitatory synapse The release of predominantly excitatory neurotransmitters into a synaptic cleft produces an excitatory synapse (Lundy-Ekman 2013: 49; Siegel *et al.* 2010: 100).

Facilitation The enhancement or reinforcement of a spinal reflex activity or neuronal process by the arrival of excitatory impulses at the reflex center (Freberg 2015: 97; Lundy-Ekman 2013: 51, 52).

Gamma loop The excitation of the homonymous α motor neurons by way of the muscle spindle afferents during active or passive muscle stretch will cause reinforcement of the extrafusal muscle fibers contraction of the prime mover and its synergists in a spinal reflex arc known as the gamma loop (Haines 2013: 328; FitzGerald *et al.* 2012: 125, 126; Merletti and Parker 2004: 12).

Glutamate receptors Ionotropic glutamate receptors are found throughout the central nervous system. These glutamate-gated ion channels include AMPA receptors and kainite receptors and are collectively known as AMPA-K receptors. Glutamate binding to AMPA-K receptors causes rapid depolarization to produce an EPSP (Lundy-Ekman 2013: 59; Fitzgerald *et al.* 2012: 99).

Glutamate Glutamate is a powerful and most prevalent excitatory neurotransmitter in the central nervous system. A glutamergic synapse is a chemical synapse in which glutamate is the predominant ligand released (Lundy-Ekman 2013: 54; Fitzgerald *et al*. 2012: 99, 187, 190).

Glycine receptors Ionotropic glycine receptors are predominately found in the grey matter of the spinal cord. Glycine binding to glycine receptors (GlyR) causes rapid hyperpolarization to produce an IPSP (Fitzgerald *et al.* 2012: 102; Siegel *et al*. 2010: 143, 144).

Glycine Glycine is an inhibitory neurotransmitter found in the central nervous system and is predominantly confined to internuncial neurons in the dorsal horn and intermediate gray matter of the spinal cord. A glycinergic synapse is a chemical

synapse in which glycine is the predominant ligand released (Lundy-Ekman 2013: 54; Fitzgerald *et al.* 2012: 99).

Graded potential When a neuron membrane is depolarized, brief local changes occur in the membrane potential at the receiving zones, known as graded potentials or local potentials. These neural impulses are called graded potentials because their amplitude is directly proportional to the intensity of the stimulus applied to the target neuron. The aim of the graded potentials is to drive the trigger zone to threshold membrane potential at about -55 mV and thus to firing rate, so that an action potential can be generated of which its amplitude is no longer directly proportional to the intensity of the stimulus applied to the target neuron (Lundy-Ekman 2013: 31, 33; Siegel *et al*. 2010: 99).

Golgi tendon organs Golgi tendon organs (GTOs) are tension-sensitive mechanoreceptors found at musculotendinous junctions of skeletal muscles. They supply the central nervous system with sensory information regarding active muscle tension and force in the muscle generated via their Ib afferent fibers. The GTO receptor consists of bundles of two types of collagen fibers which are placed in series between the distal muscle fibers and tendon or aponeurosis fibers. Several extrafusal muscle fibers insert into the GTO (Mileusnic and Loeb 2006: 1789; Merletti and Parker 2004: 13). The capsule, marginal areas, proximal (muscle) and distal (tendon) ends of the GTO consist of densely packed-bypassing collagen fibers which run parallel to each other and are rarely innervated by the Ib afferent endings. The lumen of the GTO consists of loosely packed-innervated collagen fibers which run no longer parallel to each other, but give rise to a complex network; where the collagen fibrils belonging to different muscle fibers continuously divide, mix and fuse with each other. The loosely packed collagen fibers are well innervated by GTO afferent endings whose axonal branches wind back and forth between and around the collagen fibrils repeatedly to form dilations which expose large surface areas to adjacent collagen bundles (Mileusnic and Loeb 2006: 1789, 1790; Merletti and Parker 2004: 13).

Heteronymous In relevance to the context of the study the term “heteronymous” refers to the opposite side of the shared spinal segmental innervation (Naish and Court 2014: 367).

Homonymous In relevance to the context of the study the term “homonymous” refers to the same side of the shared spinal segmental innervation (Naish and Court 2014: 367).

Hyperpolarization When the membrane potential of a neuron increases beyond its resting membrane potential (above -70 mV) to about -80 mV or more by the binding of an inhibitory ligand (Fitzgerald *et al.* 2012: 86). An inhibitory neurotransmitter can bind to and open ligand-gated chloride channels to cause an influx of Cl^- ions into the target neuron they bind to by making the postsynaptic neuron membrane more permeable to these ions. The binding of inhibitory neurotransmitters to their ligand-gated ion channels can also increase the target neuron membrane permeability to K^+ ions. The influx of Cl^- ions as well as the efflux of K^+ ions will cause hyperpolarization of the target neuron membrane and result in an inhibitory post-synaptic potential (Chambers *et al.* 2015: 229; Lundy-Ekman 2013: 49).

Inhibition When a neuron forms an inhibitory synapse on a target neuron and produces an inhibitory post-synaptic potential in the target neuron (Chambers *et al.* 2015: 229; Lundy-Ekman 2013: 49).

Inhibitory synapse The release of predominantly inhibitory neurotransmitters into the synaptic cleft produces an inhibitory synapse (Lundy-Ekman 2013: 49; Siegel *et al*. 2010: 100).

Internuncial neuron Excitatory or inhibitory neurons which exist between motor and sensory neurons in the central nervous system and transmit their neuronal signals in several directions (Lundy-Ekman 2013: 26, 27; Rastogi 2006: 481, 482).

Ion channels Ion channels in the membrane of a neuron can be directly activated known as ionotropic receptors or indirectly activated known as metabotropic receptors by ligand binding. These ion channels are known as ligand-gated ion channels or transmitter-gated ion channels and serve as receptors for the neuron in which they are found (Lundy-Ekman 2013: 56; Fitzgerald *et al.* 2012: 94).

Inhibitory postsynaptic potentials The binding of inhibitory neurotransmitters to their specific ligand-gated ion channels in the receiving zones of the target neuron membrane can reduce the generation of graded potentials by hyperpolarizing the postsynaptic neuron membrane. The hyperpolarization of the target neuron membrane will produce and inhibitory postsynaptic potential (IPSP) which will drive the neuron membrane potential away from firing threshold at the trigger zone and thereby prevent the generation of an action potential and inhibit firing of the target neuron (FitzGerald *et al.* 2012: 86, 87; Siegel *et al*. 2010: 100; Rastogi 2006: 488).

Isometric contraction A voluntary contraction in which the length of the contracted muscle as well as the joints which move the contracted muscle do not change during the muscle contraction (Magee 2008: 35).

Lower motor neurons The lower motor neurons include the α motor neurons and y motor neurons which occupy Rexed laminae VIII and IX of the grey matter of the spinal cord and can be collectively referred to as a motor neuron pool (Siegel *et al*. 2010: 143, 144; Jacobson and Marcus 2011: 67).

Motor end plate The junctional folds of the sarcolemma of a muscle cell is known as motor end plates. The motor end plate contains nicotinic receptors (Siegel *et al*. 2010: 98; Conn 2008: 100, 101).

Motor neuron Neurons which send their neural impulses to the effectors in the peripheral tissues such as to a muscle or gland cell by their motor efferent fibers (Lundy-Ekman 2013: 26, 27; Rastogi 2006: 481, 482).

Motor unit A motor unit consist of an α motor neuron cell and a squad of extrafusal fibers of a skeletal muscle which are innervated by the large myelinated axon of the α motor neuron (Lundy-Ekman 2013: 192; Merletti and Parker 2004: 2).

Muscle fiber Each muscle fiber of a motor unit consists of several myofibrils which are arranged parallel to the long-axis of the muscle fiber. Each myofibril, also known as a muscle cell, is enclosed by connective tissue which makes up the membrane of the muscle cell known as the sarcolemma. An end plate potential will result in contraction of the muscle fiber. (Lundy-Ekman 2013: 187; FitzGerald *et al.* 2012: 122, 123).

Muscle spindle Within skeletal muscle there are long, thin encapsulated stretch receptors called neuromuscular spindles. Muscle spindles are enclosed in a collagenous spindle capsule and consist of numerous small and large intrafusal muscle fibers which are striated and deep to the extrafusal muscle fibers which make up the bulk of the skeletal muscle (Haines 2013: 327; FitzGerald *et al.* 2012: 122, 123; Merletti and Parker 2004: 12). In the center of the muscle spindle known as the equator; the sarcomeres of the intrafusal muscle fibers are predominantly replaced by nuclei in the form of wide fibers called nuclear bags and slender fibers called nuclear chains (FitzGerald *et al.* 2012: 122, 123; Merletti and Parker 2004: 12). This specific arrangement of equator nuclei and intrafusal muscle fibers at the end of the equator of the muscle spindle allow the equator region to be stretched which cause

activation of the muscle spindle afferents namely the Ia fibers (primary annulospiral fibers) and IIa fibers (secondary flower spray fibers). The primary afferent encodes predominantly the rate of the muscle stretch, whereas the secondary afferent encodes predominantly the degree of muscle stretch. Passive stretching of the extrafusal muscle fibers will also elongate the muscle spindle and stretch the equatorial region of the muscle spindle, which will cause activation of the muscle spindle afferents. Active stretching of the muscle will cause contraction and thus shortening of the intrafusal muscle fibers via the activated y motor neurons and in turn cause stretching of the equatorial region of the muscle spindle, which will cause activation of the muscle spindle afferents (Haines 2013: 327, 328).

Muscle tone Muscle tone or tension in a muscle is the resistance to passive stretch and is provided predominantly by titin and weak actin-myosin bonds (Lundy-Ekman 2013: 188).

Neuron The specialized cells organized in a highly specific way that make up the nervous system are known as neurons. A neuron consists of a cell body, also known as a soma, which contains the nucleus, and neural projections known as neurites or nerve fibers. The receiving zones of a neuron are the areas on the cell body or dendrites that receive information in the form of neural impulses at synapses or nerve endings. Neurites are responsible for receiving and conduction of information towards the cell body. A single long tubular neurite which conducts neural impulses away from the cell body is known as the axon (Snell 2010: 34). The neuron signal that is generated in the receiving zones produce a self-generating electrical wave that propagates from the receiving zones to the point of initiating an action potential (or spike) at a site proximal to the cell body in motor neurons known as the axon hillock or at a site distal to the cell body in sensory neurons known as the trigger zone. The neural signal in the form of the generated action potential is convoyed to the synaptic terminals down the axon by a self-propagating mechanism. The axon terminals or synaptic endings contain secretory organelles known as synaptic vesicles which contain ligands (particularly neurotransmitters) which are highly specialized molecules that convey an excitatory or inhibitory signal to the target neuron. The contact which the synaptic endings make with the target neurons is known as a chemical synapse. Between the synapse and the target neuron is a narrow gap known as the synaptic gap. In relation to the synapse, the terminal ending of a presynaptic neuron is the synaptic terminal which contains the synaptic vesicles and exerts its effects on the terminal ending of the postsynaptic neuron (also known as the target neuron). Neurotransmitters are released from synaptic vesicles in the presynaptic neuron terminal and

cross the synaptic cleft to bind to their specific receptor sites on the postsynaptic membrane terminal of the target neuron (Lundy-Ekman 2013: 25, 26; Wu 2001: 2).

Neuronal convergence Convergence refers to a neural process by which several different neurons form synapses on a single neuron (Lundy-Ekman 2013: 36). The convergence of a neuron can include an axosomatic, axodendritic and / axoaxonic synapse(s) on the same neuron. The synapse between the axon of a presynaptic terminal and the soma of the postsynaptic neuron is called an axosomatic synapse. The synapse between the axon of a presynaptic neuron and the dendrite of a postsynaptic neuron is called an axodendritic synapse. Also, the synapse between the axon of a presynaptic neuron and the axon of a postsynaptic neuron is called an axoaxonic synapse (Lundy-Ekman 2013: 49, 51; Bear *et al.* 2007: 106, 107).

Nicotinic receptors Ionotropic nicotinic receptors are found in the muscle cell membrane at the neuromuscular junction of a skeletal muscle (Lundy-Ekman 2013: 59; Siegel *et al*. 2010: 98).

Peripheral sensitization An increase in nociceptive responsiveness and a decrease in nociceptor threshold in the periphery to stimulation of their receptive (Simpson *et al* 2012: 15) causes even non-noxious stimuli such as light touch or normal joint movement to depolarize and activate nociceptors and cause their firing (Jones *et al.* 2013: 206; Seaman and Winterstein 1998: 269).

Presynaptic inhibition Presynaptic inhibition occurs when an axon terminal of an inhibitory internuncial neuron forms an inhibitory axoaxonic synapse on an axon terminal of a presynaptic neuron in an axodendritic or axosomatic synapse (Freberg 2015: 97; Lundy-Ekman 2013: 51, 52).

Reciprocal inhibition This is a neuromodulation mechanism where branches of muscle spindle afferents synapse concomitantly on Ia inhibitory internuncials in the anterior horn and intermediate gray matter of the spinal cord to cause inhibition of the antagonist α motor neurons in a di-synaptic reflex arc (Haines 2013: 328; FitzGerald *et al.* 2012: 125, 126; Merletti and Parker 2004: 12).

Refractory When an axon of a neuron is undergoing hyperpolarization, no further wave of excitation in the form of an action potential can pass that point on the axon. The time period for recovery before a sequential action potential can be

generated at the trigger zone and propagated along the axon and thus before the resting neuron membrane potential is restored, is known as the refractory period. A continued application of stimuli to a neuron will therefore result in the generation of neuron impulses along the axon in a sequential manner (Starr and McMilan 2015: 243; Rastogi 2006: 484).

Sensory neuron Neurons which transmit information from the peripheral somatic receptors or nerve endings such as from the skin, muscles and joint by their sensory afferent fibers to the central nervous system for central processing (Lundy-Ekman 2013: 26, 27; Rastogi 2006: 481, 482).

Spinal cord The spinal cord is found in a cavity that is made of the joining of successive vertebral bodies and their vertebral arches known as the neural spinal canal (Stapleton 2002: 33). The spinal cord consists of an outer white matter that is made up of neuronal axonal pathways and an inner grey matter that is made up of neuronal nuclear groupings (Mendoza and Foundas 2007: 2, 3). The inner grey matter can be divided broadly into an anterior horn, dorsal horn, lateral horn and intermediate grey matter (FitzGerald *et al.* 2012: 38; Jacobson and Marcus 2011: 65). The grey matter of the spinal cord can also be organized in a cytoarchitectural manner; the cluster of neurons that make up the grey matter of the spinal cord can be arranged into ten zones (I-X) known as Rexed laminae (Siegel *et al*. 2010: 143; Jacobson and Marcus 2011: 65).

Spinal manipulation Spinal manipulation is a high-velocity low-amplitude thrust delivered at the end range of motion of facet joints in the spine, in the direction of the orientation of the facet joint articulation, accompanied often with an audible cracking sound (Millan *et al.* 2012: 24; Pickar 2002: 35).

Summation The superimposition of multiple spikes received at the receiving zones of a neuron and thus the superimposition of multiple generated graded potentials or EPSP in a neuron. Spatial summation refers to multiple spikes arriving simultaneously at the receiving zones of a neuron, whereas temporal summation refers to individual spikes arriving at the receiving zones of a neuron at different times (Lundy-Ekman 2013: 31, 33; Siegel *et al*. 2010: 100).

LIST OF ABBREVIATIONS AND SYMBOLS

%	percent
AAI 1	First Activator II Adjustment Instrument placebo spinal manipulation of the ipsilateral C5/C6 segment
AAI 2	Second Activator II Adjustment Instrument placebo spinal manipulation of the ipsilateral C5/C6 segment
AAI	Activator II Adjustment Instrument
ACh	Acetycholine
ACL	Anterior cruciate ligament
AMI	Arthrogenic muscle inhibition
AMPA	α-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid
AMPA-K	AMPA and kainite receptors
Ca^{2+}	Calcium
cAMP	cyclic Adenosine Monophosphate System
CDC	Chiropractic Day Clinic
Cl^-	Chloride
cm	centimeter
DUT	Durban University of Technology
EMG	Electromyography
EPP	End-plate potential
EPSP	Excitatory post-synaptic potential

GlyR	Glycine receptor
GTO	Golgi tendon organ
H-reflex	Hoffmann reflex
HVLA	High-velocity low-amplitude thrust
iEMG	Integrated electromyography
IPSP	Inhibitory post-synaptic potential
IREC	Institutional Research Ethics Committee
K^+	Potassium
kg	kilogram
LCST	Lateral corticospinal tract
M	Mean
MCN	Musculocutaneous nerve
mepp	Minature end-plate potential
ml	milliliter
mm	millimeter
ms	millisecond
N	Newton
n	Sample size or count
Na^+	Sodium
PNF	Proprioceptive neuromuscular facilitation
RMS	Root Mean Square

SD	Standard deviation
SE	Standard error
SEMG	Surface electromyography
SMT	Spinal manipulation of the ipsilateral C5/C6 segment
TMS	Transcranial magnetic stimulation
y	gamma
α	alpha
μV	microvolt

OUTLINE OF DISSERTATION

CHAPTER ONE – INTRODUCTION

The introduction provides a rationale for the study based on the relevant literature, identifies a gap or problem in the literature and states the purpose of the study.

CHAPTER TWO – LITERATURE REVIEW

This chapter conveys the knowledge required for understanding the neurophysiological mechanism underlying the methodology of this study. The literature is analyzed critically.

CHAPTER THREE – METHODOLOGY

This chapter explains and describes the manner in which the research study was conducted.

CHAPTER FOUR – RESULTS

This chapter presents the data collected in the form of tables and graphs supported by narratives.

CHAPTER FIVE – DISCUSSION

This chapter discusses the results obtained and provides proposed neurophysiological explanations thereof with possible vindications.

CHAPTER SIX – CONCLUSION

Chapter 6 presents the final conclusion of the research study conducted, as well as the final opinion of the researcher. It also provides recommendations based on the limitations of the study and new insights.

CHAPTER 1 : INTRODUCTION

1.1 INTRODUCTION TO THE STUDY

The current accepted theory for the basis of the neurophysiological mechanism underlying chiropractic spinal manipulation in symptomatic is related to the reduction of central sensitization in the spinal cord. Central sensitization has been found to induce abnormal spinal reflex arcs which can affect the homonymous motor-, nociceptive- and possibly the autonomic-neuronal pools by sensitizing mechano-insensitive nociceptors within and around the facet joint tissue (Olsen 2015: 82; Vernon 2010: 28; Pickar 2002: 358). Spinal manipulation is believed to correct alterations in the normal anatomical, physiological and / or biomechanical dynamics of individual vertebral segments to its pre-injury / normal state which lead to the induction of the central sensitization, and in doing so restores the normal functioning of the nervous system (Gutterman 2005: 281; Pickar 2002: 359). However, the neurophysiological effects of spinal manipulation on asymptomatic individuals are not known.

Marked weakness of uninjured musculature which has shared innervation with a dysfunctional joint has been found in the absence of structural damage, pain or inflammation. This is due to the on-going inhibition that prevents the weak muscle from being fully activated, a phenomena known as arthrogenic muscle inhibition (AMI) (Rice and McNair 2010: 250; Rossi *et al*. 2002: 523). Several investigators have demonstrated reduction of AMI with an associated increase in muscle activity post-spinal manipulation (Dunning and Rushton 2009: 512; Picker 2002: 364; Suter and McMorland 2002: 544). The neurophysiological mechanism responsible for the reduced AMI and improved functional capacity of the muscle after the spinal manipulation is not fully understood (Suter and McMorland 2002: 544). There is a gap in the chiropractic literature regarding the neurophysiological effect of spinal manipulation on AMI (Dunning and Rushton 2009: 509) as well as on the underlying neurophysiological mechanism of spinal manipulation on motor activity (Olsen 2015: 86; Pickar 2002: 364). This study aims to reduce this gap.

The electromyographic response post-spinal manipulation may support the elucidation of the underlying neurophysiological mechanism of spinal manipulation on motor activity (Olsen 2015: 86; Pickar 2002: 364). The literature shows conflicting evidence regarding the excitatory or inhibitory nature of the reflexive electromyographic response and the excitability of the homonymous motor neuron pool post-spinal manipulation (Olsen 2015: 86; Dunning and Rushton 2009: 508; Picker 2002: 364). The literature has revealed a gap in the EMG response post-spinal

manipulation. EMG studies (Olsen 2015: 86; Pickar 2002: 364) have solely investigated the effects of spinal manipulation on muscle activity pre- and post- maximum voluntary muscle contraction and not during the muscle contraction. Also, these studies have not yet investigated the effect of spinal manipulation on muscle activity during an induced golgi tendon organ Ib inhibitory di-synaptic reflex as part of the convergent input on the homonymous motor neuron pool excitability.

The Ib inhibitory spinal reflex arc is found to be the major spinal pathway which causes AMI (Rice *et al*. 2014: 503; Rice and McNair 2010: 256). Studies have shown that there is little specialization of Ib internuncial neurons by the type of their afferent input, as the afferent input from GTOs and from joints cause similar Ib inhibition on the homonymous α motor neuron (Greger and Windhorst 2013: 1001; Brushart 2011: 73). The current study will investigate whether the electromyographic response post-spinal manipulation is affected by a facilitated GTO Ib inhibitory di-synaptic spinal reflex as part of the convergent input on the homonymous motor neuron pool excitability.

1.2 AIM AND OBJECTIVES

1.2.1 AIM OF THE STUDY

To determine the effect of spinal manipulation of the C5/C6 segment during three sets of modified stretching of the biceps brachii on the muscle's electromyograph.

1.2.2 THE OBJECTIVES OF THE STUDY:

a) To determine electrical activity and muscle force of the biceps brachii muscle immediately before and after an Activator Adjusting II Instrument placebo spinal manipulation during the modified stretching of the biceps brachii muscle.
b) To determine electrical activity and muscle force of the biceps brachii muscle immediately before and after a C5/C6 spinal manipulation during the modified stretching of the biceps brachii muscle.
c) To compare the electrical activity and muscle force of the biceps brachii muscle between the control and intervention groups pre- and post-test.

1.3 BENEFITS OF THE STUDY

This study may provide scientific evidence for the underlying neurophysiological mechanism of spinal manipulation on motor activity.

This study also has clinical implications for rehabilitation practitioners and physical therapists. The findings of this study could provide the neurophysiological mechanism responsible for the effect of

spinal manipulation on AMI. Arthrogenic muscle inhibition presents a unique challenge to musculoskeletal therapists as it may hinder any treatment or rehabilitation outcomes (Rice and McNair 2010: 250; Rossi *et al.* 2002: 523). Treatments that specifically aids in the reduction of AMI could be an important tool to therapists in improving treatment and rehabilitation outcomes that would have been impeded by the inhibition (Rice and McNair 2010; Rossi *et al*. 2002). The findings of this study could provide evidence based support to the suggestion that for optimal management of patients with muscle weakness suspected to be of arthrogenic nature, the application of spinal manipulation to the segmentally innervated facet joints may be of benefit before traditional strength training is initiated (Dunning and Rushton 2009: 512).

CHAPTER 2 : LITERATURE REVIEW

2.1 INTRODUCTION

This chapter serves to provide the reader with the needed knowledge for understanding the neurophysiological mechanisms underlying the methodology of this study. All neuronal convergence on the homonymous motor neuron pool is of particular importance.

The literature review was conducted by means of an in depth study of the medical and chiropractic literature. The knowledge was obtained by a systematic investigation of the latest articles and anatomical, neuroscientific and chiropractic textbooks, in order to substantiate the proposed neurophysiology behind the methodology of this study. The Durban University of Technology Library, Google scholar, Google books and Amazon kindle books were also utilized.

2.2 NEUROPLASTICITY: HABITUATION

Neuroplasticity is the ability of neurons, neural pathways and their synapses to change and adapt to alterations in behavior, environment, neural processes, thinking, emotions and external noxious or abnormal stressors or stimuli (Clementi and Fumagalli 2015: 122). In general, neuroplasticity entails habituation, central sensitization, experience-dependent plasticity (learning and memory), and neuronal cellular recovery after injury to a neuron (Lundy-Ekman 2013: 67).

A form of neuroplasticity which is not associated with learning and memory in the central nervous system, but which is characteristic of a decreased response to a repeated and / or continuous benign stimulus, is known as habituation (Clementi and Fumagalli 2015: 122; Lundy-Ekman 2013: 67). Studies have recognized the presence of habituation clinically, by inducing repeatedly an involuntary spinal reflex to painful stimuli namely the withdrawal reflex (Lundy-Ekman 2013: 67). These studies found that the induced withdrawal reflex to a mildly painful stimulus reduced or ceased over time after several repetitions of the same stimulus, and concluded that this phenomenon is due to habituation. When a repetitive benign stimulus was removed; in the example a period of rest after the withdrawal reflex was repeatedly induced, the effects of habituation were completely absent or partially removed and the normal withdrawal reflex could be induced again after the rest period (Lundy-Ekman 2013: 67; French 1970: 461). Habituation can therefore cause transient neuroplasticity (Clementi and Fumagalli 2015: 122).

The exact underlying cellular mechanism for habituation is not clear. Studies have affirmed that the underlying cellular mechanism for habituation may involve a decrease in the liberation of neurotransmitters, particularly glutamate, from the presynaptic neuron terminal, as well as a decrease in free intracellular Ca^{2+} ions in the presynaptic and /or postsynaptic neuron terminal (Lundy-Ekman 2013: 67; French 1970: 461). Habituation is therefore the result of alterations of gene transcription inside the neuron. Habituation of a neuron induced by repetitive benign stimuli will cause cAMP (cyclic adenosine monophosphate system) to decrease its normal rate of activation of protein kinases involved in phosphorylation of proteins that regulate gene transcription, and result in a reduced rate of neurotransmitter synthesis and release (Fitzgerald *et al.* 2012: 97).

Prolonged repetition of a benign stimulus can also cause more permanent structural changes to occur in a neuron, as well as a decrease in the number of synapses of the neuron, resulting in a more stable neuroplasticity. Habituation can be beneficial (Lundy-Ekman 2013: 67) for example, repetitive acoustic ringing in the ear known as tinnitus can be treated by using a hearing aid. The hearing aid is designed to habituate to the tinnitus over a prolonged period of time, and results in a decrease in tinnitus (Sweetow and Sabes 2010: 461).

Occupational and physical therapists use the principles behind habituation to decrease abnormal neural responses to a stimulus by using specific techniques and exercises based on the effects of habituation (Lundy-Ekman 2013: 67, 69). For example, habituation techniques can be used in the treatment of an abnormal increase in sensitivity of skin to a stimulus known as hypesthesia caused by neuropraxia. In addition to aiding in the regeneration of an injured nerve fiber by gently stoking the hypersensitive skin and gradually increasing the intensity of stimulation over time, habituation can be achieved to the tactile stimulation and aid in the reduction of hypesthesia (Hertenstein and Weiss 2011: 422). Another example includes vertigo. Vertigo is a vestibular disorder in which normal movement cause dizziness and nausea. By inducing the movements that induce dizziness and nausea repeatedly over time, habituation of the movements can be achieved and result in a reduction of vertigo (Cesarani and Alpini 2012: 3).

It is important for chiropractors to also adopt the principles behind habituation. The current study may provide an evidence based theory for the suggestion that spinal manipulation may achieve habituation to abnormal arthrokinetic reflex arcs. The following section will explain the various elements that are significant in the process of habituation.

2.3 JOINT MECHANORECEPTORS

2.3.1 INTRODUCTION

The current accepted neurophysiological mechanism for both spinal manipulation (DePalma 2011: 81; Sterling and Kenardy 2011: 19; Vernon 2010: 27) and arthrogenic muscle inhibition entails affecting the afferent discharge of somatic receptors in joint tissues (Rice *et al*. 2014: 503; Rice and McNair 2010: 253; Hopkins *et al*. 2000: 135). All participants of this study were asymptomatic and information relating to the effects of all nociceptive afferents including those from the facet joint tissue on the homonymous motor neuron pool excitability is excluded from this review.

An increase in joint mechanoreceptor Aβ-fiber afferent discharge is strongly associated with AMI and it is postulated that joint afferent input has competing excitatory and inhibitory influences on the homonymous motor neuron pool. In a dysfunctional joint, the net effect can be inhibitory (Rice and McNair 2010: 255; Konishi *et al*. 2003: 1805). Konishi *et al.* (2003: 1805) demonstrated AMI by injecting 5 ml of local aesthetic into normal undamaged knee joints, the quadriceps femoris force output and integrated EMG during maximal voluntary isometric decreased compared to pre injection measurements.

Increased Aβ-fiber afferent input from joint mechanoreceptors can cause inhibition of the homonymous α motor neurons in the anterior grey horn of the spinal cord by synapsing on Ib internuncials in lamina VI an VII in the intermediate grey matter of the spinal cord via the Ib inhibitory di-synaptic reflex arc (Rice *et al*. 2014: 503; Greger and Windhorst 2013: 1001; Brushart 2011: 73; Rice and Mcnair 2010: 256). The facilitated Ib inhibition can decrease the excitability of the homonymous motor neuron pool at multiple spinal levels and may cause gamma loop dysfunction. Any impaired transmission of Ia afferent signals from the muscle spindles to the homonymous α motor neuron is known as gamma loop dysfunction. Gamma loop dysfunction may contribute to AMI by causing presynaptic inhibition of the Ia afferent axons near their contact points with their homonymous α motor neurons, which may increase the activation threshold and / or cause neurotransmitter depletion of the Ia afferent terminal endings (Rice *et al*. 2014: 151; Rice and Mcnair 2010: 257).

2.3.2 MECHANORECEPTORS IN THE FACET JOINT TISSUE

Joint mechanoreceptors are classified as type I-III nerve endings (Petty 2011: 14) and are supplied by large myelinated type Aβ-fibers (Petty 2011: 14; Sterling and Kenardy 2011: 19). Type I Ruffini end-organs are slowly adapting, low-threshold, static and dynamic mechanoreceptors. They are found around the collagen fibers of the superficial layers of the cervical facet capsular tissue and

are stimulated by the displacement of collagen. Type I joint mechanoreceptors signal a change in intra-articular pressure and static joint position, as well as the direction, amplitude and velocity of joint movement. They are extremely sensitive to a change in capsular stretch. Stimulation of Ruffini end-organs affect the tone of the spinal segmentally innervated muscles (Petty 2011: 14; Sterling and Kenardy 2011: 19; Yoganandan and Pintar 2000: 250; McLain 1994: 152).

Type II Pacinian corpuscles are rapid adapting, low threshold and dynamic mechanoreceptors. They are found in the deeper layers of the cervical facet capsular tissue. Type II joint mechanoreceptors signal dynamic joint position and a change in stress applied to the joint capsular tissue during joint movement from the start to end and during deceleration and acceleration. The Pacinian corpuscle is stimulated mainly by compression and tensile loading on the joint capsular tissue. Type II joint mechanoreceptors are sensitive to a change in capsular stretch. Activation of Pacinian corpuscles directly affects the tone of the spinal segmentally innervated muscles (Petty 2011: 14; Sterling and Kenardy 2011: 19; Yoganandan and Pintar 2000: 250; McLain 1994: 152).

Type III Golgi ending nerve endings are very slowly adapting, high threshold and dynamic mechanoreceptors (Petty 2011: 15; Sterling and Kenardy 2011: 19; Yoganandan and Pintar 2000: 250; McLain 1994: 15). These encapsulated nerve endings are found at the junction between the inner and more superficial layers of the cervical facet joint capsular ligament (Sterling and Kenardy 2011: 19; Yoganandan and Pintar 2000: 250; McLain 1994: 152). There is not much known about these joint mechanoreceptors, as some studies have not identified them in the cervical facet joint tissue (Petty 2011: 15). Type III joint mechanoreceptors signal extreme range of joint movement and are stimulated by tension in the joint capsular tissue. They are therefore also sensitive to a change in capsular stretch (Petty 2011: 15; Sterling and Kenardy 2011: 19; Yoganandan and Pintar 2000: 250; McLain 1994: 152).

Joint mechanoreceptors play an important role in maintaining articular congruity, assist in distributing the load on a joint, influence the activity of the spinal segmentally innervated muscles and relay information to the central nervous system regarding joint position sense (postural sense and kinaesthetic sense) or joint proprioception. Postural sense is not solely dependent on the joint mechanoreceptors input, because cutaneous (skin) and muscle spindle afferents also play a role in the maintenance of posture (Middleditch and Oliver 2005: 246).

Joint movement can reflexively cause activation or inhibition of the spinal segmentally innervated muscles through the arthrokinetic reflex. Joint mechanoreceptor afferent activity can exert powerful tonic facilitatory and inhibitory reflexogenic influences on the homonymous motor neuron pool during postural control and voluntary activity (Middleditch and Oliver 2005: 246). Thus when the

joint capsule is stretched during joint movement the joint mechanoreceptors will cause activation of the muscles which will reduce the joint capsular stretch and cause inhibition of the muscles which will increase the joint capsular stretch (Petty 2011: 16).

The arthrokinetic reflex, in which receptors in the joint affect muscle activity, is dependent on the type of receptor activated. It is postulated that joint type I-III nerve endings can, in addition to affecting the spinal segmentally innervated muscles directly by activating the homonymous α motor neurons, also affect the muscles by activating the homonymous y motor neuron-muscle spindle loop (Petty 2011: 16). Impairment of the arthrokinetic reflex can therefore produce abnormal patterns of spinal reflex arcs (Middleditch and Oliver 2005: 247) and result in weakness of the spinal segmentally innervated muscles (Middleditch and Oliver 2005: 247; Porter 2013: 581) known as arthrogenic muscle inhibition (Rice *et al.* 2014: 503; Rice and McNair 2010: 250, 253).

Any joint dysfunction that can affect the facet joint mechanoreceptors and cause alterations in their Aβ-fiber afferent discharge can impair the arthrokinetic reflex and joint position sense, and lead to neurogenic inhibition and weakness of the homonymous muscle group (Middleditch and Oliver 2005: 247; Hendrickson 2002: 291). Therefore, it is important to emphasize the importance of rehabilitation of joint mechanoreceptors in the treatment of any joint dysfunction (Middleditch and Oliver 2005: 247).

2.3.3 EFFECT OF SPINAL MANIPULATION

Repetitive postural strain or trauma can cause alterations in the normal anatomical, physiological and / or biomechanical dynamics of individual vertebral segments and produce relatively large vertebral motions that achieve a new position of stable equilibrium (Vernon 2010: 29; Gatterman 2005: 8). The higher energy level needed to achieve the new position of stable equilibrium can place additional mechanical stress or overload on the facet joint capsular tissue and / or cause uneven or increased unilateral facet joint loading (Vernon 2010: 29; Gatterman 2005: 8). These alterations in the vertebral segment can cause tension, pressure, stretching or irritation of the facet joint capsular tissue as well as the displacement of collagen in the facet joint capsular ligament (Vernon 2010: 29; Gatterman 2005: 8; Pickar 2002: 359), and thereby stimulate (depolarize and sensitize) mechanoreceptors within the facet joint tissue and subsequently increase their Aβ-fiber afferent discharge frequency (Vernon 2010: 29; Dunning and Rushton 2009: 512; Pickar 2002: 360; Seaman 1998: 268). The raised Aβ-fiber afferent discharge from the facet joint tissue can cause alterations in the arthrokinetic reflex and result in neurogenic inhibition and weakness of the homonymous muscle group (Middleditch and Oliver 2005: 247; Hendrickson 2002: 291).

Spinal manipulation can stimulate mechanoreceptors in the facet joint tissue and normalize their Aβ-fiber afferent discharge (Olsen 2015: 82; Millan *et al.* 2012: 24; Vernon 2010: 24; Pickar 2002: 358, 359) by reducing or normalizing the alterations in the vertebral segment to its pre-injury / normal state and in so doing reduce the efferent manifestations induced by the altered arthrokinetic reflex (Olsen 2015: 85; Vernon 2010: 28; Gutterman 2005: 281; Pickar 2002: 359). Studies have demonstrated that spinal manipulation can stimulate low-threshold and high-threshold mechanoreceptors in the facet joint tissue (Millan *et al.* 2012: 24; Pickar 2002: 35). Studies have affirmed these findings by demonstrating that type I-III nerve endings in the facet joint tissue respond to the impulse of a high-velocity low-amplitude load applied during spinal manipulation and not to loads with a slower force-time profile (Colloca *et al.* 2000: 447). Spinal manipulation is often accompanied with an audible cavitation ("cracking sound"). The accompanied audible cavitation is possibly caused by gapping of the facet joint which creates fluid cavitation within the facet joint tissue (Millan *et al.* 2012: 24; Pickar 2002: 35).

2.3.4 ARTICULAR SWELLING

Several studies have found that articular swelling can cause significant AMI even in the absence of pain, inflammation or structural damage of a joint, by infusing fluid into the undamaged joint. Direct recordings from articular nerves have shown that swelling can significantly increase the frequency of discharge and recruitment of both mechanoreceptor and nociceptor joint afferents. Moderate levels of joint swelling rarely evoke pain, making it unlikely that swelling alone stimulates a significant number of joint nociceptors. An increase in Aβ-fiber afferent discharge from joints can solely occur in minor to moderate joint swelling, whereas nociceptors are more likely to be mechanically stimulated at higher intra-articular pressures or depolarized in the presence of inflammation. It has been demonstrated that as little as 10 ml of fluid infused into joints can cause notable muscle inhibition, whereas infusions of between 20 ml and 60 ml can cause maximum muscle inhibition (Rice *et al*. 2014: 504; Rice and McNair 2010: 254; Hopkins *et al*. 2001: 123).

There is a close relationship between intra-articular pressure and the discharge of joint mechanoreceptor Aβ-fiber afferents. Articular swelling can increase the intra-articular pressure even at resting joint position; a joint effusion as small as 5 ml has been demonstrated to raise the intra-articular pressure above atmospheric pressure and induce AMI. Swelling has a strong inhibitory effect on the homonymous motor pool and even small, clinically undetectable joint effusions can cause significant AMI (Rice *et al*. 2014: 504; Rice and McNair 2010: 254; Jensen and Graf 1993: 52).

Particularly type I joint mechanoreceptors called the Ruffini end-organs are sensitive to a change in intra-articular pressure and are found around the collagen fibers of the superficial layers of the cervical facet capsular tissue (Petty 2011: 14; Sterling and Kenardy 2011: 19). Alterations in the normal anatomical, physiological and / or biomechanical dynamics of a vertebral segment can induce tension and pressure inside the facet joint capsular tissue and synovium (Vernon 2010: 29; Dunning and Rushton 2009: 512; Gatterman 2005: 510; Pickar 2002: 360; Seaman 1998: 268), thereby causing AMI by mechanically stimulating the Ruffini end organs.

2.3.5 JOINT LAXITY

Damage or stretching of joint ligaments or joint capsule can cause a greater translation of the joint surfaces during movement, which is likely to increase the activity of the joint mechanoreceptors involved in signaling the limits of the joint movement and subsequently contributing to AMI (Rice and McNair 2010: 255). Gomez-Barrena *et al.* (1999: 185) found an increase in joint mechanoreceptors afferent discharge after surgically transecting the ACL from a cat's knee joint and directly measuring the joint afferents activity. Following transection of the ACL, Gomez-Barrena *et al.* (1999: 185) surgically reconstructed the ACL and repeated the articular nerve recordings. They found that the reconstructed ACL reduced the mechanoreceptors joint afferent discharge, but to an altered discharge level which was higher than compared to before the ACL transection. Although direct comparisons cannot be made to humans, Gomez-Barrena *et al*.'s (1999: 185) findings concluded that stretching of joint ligaments or the joint capsule can cause an increase in their mechanoreceptors afferent discharge.

Joint mechanoreceptors relay information to the central nervous system via the posterior-medial lemniscal pathways reporting conscious proprioception and via the spinocerebellar pathways reporting non-conscious proprioception (Middleditch and Oliver 2005: 246). Any induced alteration in the articular mechanoreceptors discharge may affect the position sense and kinesthetic sense of the affected joint and give rise to symptoms of disequilibrium (Fitzgerald *et al.* 2012: 179; Middleditch and Oliver 2005: 247). Alterations in the normal anatomical, physiological and / or biomechanical dynamics of a vertebral segment can produce relatively large vertebral motions that achieve a new position of stable equilibrium. The higher energy level needed to achieve the new position of stable equilibrium can place additional mechanical stress or overload on the facet joint capsular tissue and synovium (Gatterman 2005: 8; Pickar 2002: 359) and hold the capsular ligament of the facet joint in a subtle stretched position (Vernon 2010: 28; Middleditch and Oliver 2005: 248, 246; Gatterman 2005: 510; Pickar 2002: 358). Although the alterations in the vertebral segment does not cause gross laxity of the facet joints (Vernon 2010: 29; Pickar 2002: 359), or may be too subtle to cause symptoms of disequilibrium; the alterations in the normal anatomical,

physiological and / or biomechanical dynamics of the vertebral segment may affect the facet joint position sense to the extent of solely increasing their mechanoreceptor Aβ-fiber afferent discharge.

2.3.6 CENTRAL NEUROPHYSIOLOGICAL MECHANISM

Joint mechanoreceptor Aβ-fiber afferents have extensive spinal and supraspinal projections and altered joint afferent discharge may affect the supraspinal centers (Rice *et al.* 2014: 502; Rice and McNair 2010: 258). In contrast, anti-nociception can occur at spinal segmental and supraspinal levels (Buijs and Swab 2013: 47; Fitzgerald *et al.* 2012: 363). AMI is mainly mediated at the associated spinal levels and are subject to their own modulatory influences, by way of the joint dysfunction (Rice and McNair 2010: 255). Therefore, anti-nociception mechanisms may not reduce the severity of AMI (Rice and McNair 2010: 250; Rossi *et al.* 2002: 523).

Studies have been conducted to quantify changes in corticospinal excitability associated with chronic knee pathology via transcranial magnetic stimulation (TMS) of the motor cortex. These studies found higher quadriceps femoris corticospinal excitability in patients with knee pathology compared to healthy subjects, despite that the quadriceps femoris EMG amplitude during maximal contractions and resting motor threshold was significantly reduced. These findings revealed increased corticospinal excitability, but the location of the observed changes involving the motor cortex versus the motor neuron pools cannot be easily determined by using a single-pulse TMS. The researchers concluded that the excitability of the motor cortex projecting to the motor neuron pools is likely to be increased and the homonymous α motor neurons are likely to be inhibited due to AMI (Heroux and Tremblay 2006: 823; On *et al*. 2004: 127).

2.4 ELECTROMYOGRAPHIC RESPONSE POST-SPINAL MANIPULATION

Several studies have provided the objective evidence that spinal manipulation can alter the excitability of the homonymous motor neuron pool and evoke spinal reflex activity (Olsen 2015: 86; Pickar 2002: 364). Studies have found varied electromyographic responses post spinal manipulation; spinal manipulation can have an inhibitory effect or an excitatory effect on the homonymous motor neuron pool excitability, as well as result in an increase in muscle strength. The neurophysiological mechanism by which spinal manipulation may reduce muscular spasm and weakness is not fully understood (Olsen 2015: 86; Pickar 2002: 365; Dishmen *et al*. 2002: 1). The most favorable proposed neurophysiological mechanism involves amplification or attenuation of the excitability of the homonymous motor neuron pool induced by the spinal manipulation (Olsen 2015: 86; Pickar 2002: 365; Dishmen *et al*. 2002: 1). The specific spinal pathways that the spinal manipulation utilizes to induce the altered motor neuron pool excitability are unknown (Olsen 2015: 86).

DeVocht *et al.* (2005: 465) investigated the effect of spinal manipulation of the lumbar spine on EMG activity of localized tight muscle bundles in the segmental paraspinal muscles. They found that in some participants there were immediate transient decreases in EMG activity after spinal manipulation. DeVocht *et al.* (2005: 465) found in other participants that spinal manipulation resulted in a short lived spike in electromyographic activity following a latent response, before returning to baseline.

Dishman *et al.* (2002: 318) investigated the effects of lumbar spine manipulation on the excitability of the lumbar motor neuron pool in participants with low back pain. They measured and recorded the amplitude of the tibial nerve Hoffmann reflex from the gastrocnemius muscle, pre- and post-the spinal manipulations. The Hoffmann reflex technique allows for an indirect measurement of the motor neuron pool excitability through direct electrical stimulation of the peripheral nerve Ia fiber which causes facilitation of the gamma loop and excludes the corticospinal influence on the homonymous motor neuron pool. Dishman *et al*. (2002: 318) found a significant transient decrease in lumbar motor neuron pool excitability immediately after lumbar spinal manipulation in participants with low back pain.

Keller and Colloca (2000: 585) investigated the electromyographic response immediately after spinal manipulation of the segmental paraspinal musculature in participants with low back pain, compared to a placebo spinal manipulation. They measured the muscle activity during lumbar spine extension isometric maximal voluntary contraction, before and after the spinal manipulation and the placebo spinal manipulation. Keller and Colloca (2000: 585) found a significant transient increase in paraspinal EMG activity post the spinal manipulation. The placebo spinal manipulation showed no significant change in the electromyographic response. Keller and Colloca (2000: 585) concluded that spinal manipulation may produce a potential short-term therapeutic effect for muscle weakness, as well as further research is warranted to determine long-term effects.

Several studies have reported latency of the electromyographic response immediately after spinal manipulation (Pickar 2002: 364). Herzog *et al*. (1999: 146) demonstrated in asymptomatic patients that spinal manipulation applied to the cervical, thoracic, lumbar spine and sacroiliac regions increased their associated paraspinal EMG activity transiently. Their findings included an electromyographic response latency occurring within 50 ms to 200 ms immediately after the HVLA thrust applied, before the transient increase in EMG. Colloca and Keller (2001: 489) confirmed these latter findings in symptomatic patients with low back pain, with an electromyographic response latency occurring within 2 ms to 3 ms immediately after the HVLA thrust applied via an Activator Adjusting Instrument. Colloca and Keller (2001: 489) also found following the electromyographic response latency, the EMG amplitude increased to reach peak amplitude within

50 ms to 100 ms, before returning to baseline. The underlying neurophysiological mechanism for the electromyographic response latency a few milliseconds immediately after the spinal manipulation is unclear (Pickar 2002: 364). The current study may provide an evidence based theory for the underlying neurophysiological mechanism of the electromyographic response latency occurring post-spinal manipulation.

2.5 NEUROPHYSIOLOGY TO SUPPORT RESEARCH METHODOLOGY

2.5.1 INTRODUCTION

Proprioceptive neuromuscular facilitation stretching is the most effective stretching technique to increase muscle flexibility and range of motion, and is commonly used in the athletic and clinical environments (Sharman *et al*. 2006: 931, 932). The underlying neural mechanisms involving PNF stretching consists of: firstly, autogenic excitation by way of the Ia muscle spindle reflex arc; secondly, autogenic inhibition by way of the golgi tendon organ Ib afferents; and thirdly, reciprocal inhibition via Ia inhibitory spinal pathways as a result of contraction of the antagonist muscle group (Bandy and Sanders 2007: 59; Sharman *et al*. 2006; 931, 932). The role of the GTOs in PNF stretching is unclear (Bandy and Sanders 2007: 59; Sharman *et al*. 2006: 932), despite the clinical proof that GTOs have an inhibitory effect on the homonymous motor neuron pool (Umphred *et al*. 2013: 209; Sharman *et al.* 2006: 932). Several authors have demonstrated that the reduced efferent (motor) drive to the muscle by way of autogenic inhibition is a major factor that assists in elongation of the targeted muscle (Umphred *et al*. 2013: 209; Sharman *et al*. 2006: 932). The autogenic inhibition phase of PNF stretching is performed by placing the targeted muscle passively in a lengthened position followed by an active low force muscle contraction to activate the Ib afferent maximally (Bandy and Sanders 2007: 59; Sharman *et al.* 2006: 932).

The behaviour of GTOs demonstrates an immediate response to muscle tension and consists of an initial dynamic response – a burst in GTO discharge within 0.5 seconds. A static response immediately follows and consists of a gradual decline to a constant GTO discharge (Plowman and Smith 2007: 575; Mileusnic and Loeb 2006: 1790, 1794-1979). Historically it was thought that GTOs only respond to high forces but several studies have demonstrated that the activation of multiple motor units simultaneously such as during high muscle force output, caused the GTOs to demonstrate non-linear summation and produced Ib afferent activity that was smaller compared to the activation of a single or two motor units. Studies have affirmed that the activation of a single or two motor units such as during very low muscle force output, caused the GTOs to demonstrate linear summation and produced higher Ib afferent activity (Mileusnic and Loeb 2006: 1790; Sharman *et al.* 2006: 932). Although the GTO behaviour depends on the generated muscle

tension that the extrafusal muscle fibers of the motor unit exert on the loosely packed innervated collagen fibrils found inside the lumen of the GTO, the response of the GTO also depends on the type of motor unit being activated (Mileusnic and Loeb 2006: 1790). Motor unit recruitment as well as the firing frequency of lower motor neurons (α motor neurons and y motor neurons) are entirely dependent on the level of force and speed of muscle contraction by the voluntary effort of the individual (Lundy-Ekman 2013: 192; Merletti and Parker 2004: 7). A low force muscle contraction will cause the recruitment of low-threshold motor units with at least one of their extrafusal muscle fibers inserting into a GTO and intertwining with the loosely packed innervated collagen fibrils inside the GTO, and result in linear summation of Ib afferent activity. As the muscular force becomes higher / faster by the voluntary effort of the individual, so the higher-threshold motor units will be recruited with at least one of their extrafusal muscle fibers inserting into a GTO and intertwining with the loosely packed innervated collagen fibrils inside the GTO, resulting in more non-linear summation of Ib afferent activity (Mileusnic and Loeb 2006: 1790).

In the current study a modified stretching of the biceps brachii muscle was performed to induce autogenic inhibition of the homonymous muscle based on the underlying mechanisms of PNF stretching (Bandy and Sanders 2007: 59). The biceps brachii muscle of the participants was passively stretched by attaching a weight of a 1 kg plate to the participant's distal forearm slowly to induce autogenic excitation of the biceps brachii muscle. The 1 kg plate was attached slowly to the participant's distal forearm in order to allow the actin-myosin cross-bridges to detach before generating resistance to the muscle stretch and in so doing reduce increased resistance to the passive muscle stretch (Lundy-Ekman 2013: 188, 190). The participant was then instructed by the researcher to perform a constant isometric biceps brachii muscle contraction against the resistance of the 1 kg plate for several seconds. The resistance against the weight of the 1 kg plate induces autogenic inhibition of the biceps brachii muscle thereby causing facilitation of the GTO Ib inhibitory di-synaptic spinal reflex arc (Bandy and Sanders 2007: 59; Sharman *et al.* 2006: 932). The following section will explain the neurophysiology of the research methodology supported by the medical literature.

2.5.2 INDUCED AUTOGENIC EXCITATION

The weight of the 1 kg weight attached to an individual's distal forearm will cause the entire belly of the biceps brachii muscle to passively lengthen, and because the extrafusal and intrafusal muscle fibers lie parallel to each other, the biceps brachii muscle spindles will also be passively stretched (FitzGerald *et al.* 2012: 125; Merletti and Parker 2004: 12). The equatorial regions of the biceps brachii muscle spindles will be subsequently stretched and cause the mechanical opening of their transduction ion channels in the surface membrane of their sensory terminals resulting in

depolarization of the biceps brachii muscle spindles equators (Lundy-Ekman 2013: 27; FitzGerald *et al*. 2012: 83, 84, 125, 126). The summation will raise the receptor membrane potential of the biceps brachii muscle spindle afferent endings of type Ia and IIa above threshold at their trigger zones and cause the generation of action potentials in the biceps brachii muscle spindle afferents in a sequential manner and causing them to fire (FitzGerald *et al*. 2012: 125, 126; Merletti and Parker 2004: 12). A single myelinated primary sensory afferent fiber type Ia supplies annulospiral wrappings around the nuclear bags and nuclear chains nuclei in the equator of the muscle spindle, known as primary annulospiral fibers. Myelinated secondary sensory afferent fibers type IIa, supply sensory endings at one or both sides of the primary sensory endings, known as secondary flower spray fibers (Fitzgerald *et al*. 2012: 122, 123).

The activated type Ia and IIa biceps brachii muscle spindle afferents will discharge their excitatory nerve impulses in the form of the generated action potentials to their first-order set of sensory neurons (Jacobson and Marcus 2011: 64) via the musculocutaneous nerve (Biller 2012: 246; Muscolino 2008: 265) and thereafter via the spinal nerves in the distal intervertebral foramina between the pedicles of successive vertebrae (Fitzgerald *et al*. 2012: 38; Steward 2012: 25) at spinal segmental levels of C5/C6 (Biller 2012: 246; Muscolino 2008: 265). The impulses from the biceps brachii muscle spindle afferents will then travel along the centripetal processes from their dorsal root ganglia to the entry zone of the spinal cord via the dorsal nerve roots (FitzGerald *et al*. 2012: 177, 178) to enter the dorsal horn of the spinal cord at the posterolateral sulcus via the medial bundle fibers of the dorsal nerve roots (Fitzgerald *et al*. 2012: 38; Steward 2012: 25, 26). The fibers of the biceps brachii muscle spindle afferents have projections via their collateral branches into lamina VIII and IX / anterior horn of the grey matter of the spinal cord to form excitatory axodendritic and / or axosomatic glutamergic synapses on the homonymous α motor neurons (Fitzgerald *et al*. 2012: 175, 176; Jacobson and Marcus 2011: 64), innervating the biceps brachii and brachialis muscles in a monosynaptic reflex arc (Haines 2013: 358; Fitzgerald *et al.* 2012: 125, 126; Merletti and Parker 2004: 12). Concomitantly, collateral braches of the biceps brachii muscle spindle afferents also have projections into the anterior horn and intermediate grey matter of the spinal cord to form excitatory axodendritic and / or axosomatic glutamergic synapses on Ia inhibitory internuncials innervating the α motor neurons of the triceps brachii muscles in a di-synaptic spinal reflex arc. The induced reciprocal inhibition of the antagonist α motor neurons will cause relaxation of the triceps brachii muscle to allow the contraction of the biceps brachii muscle (Haines 2013: 328; FitzGerald *et al*. 2012: 125, 126; Merletti and Parker 2004: 12). The impulses from the biceps brachii muscle spindle afferents will reach their synaptic terminals in the anterior horn to cause depolarization of their synaptic terminals (Fitzgerald *et al*. 2012: 175, 176; Jacobson and Marcus 2011: 64). The released glutamate neurotransmitters from the depolarized synaptic

terminals of the activated biceps brachii muscle spindle afferent will diffuse across the synaptic cleft to bind to their specific ionotropic receptors in the membrane of the homonymous α motor neurons at their receiving zones, namely the AMPA and kainite receptors (Lundy-Ekman 2013: 49; Rastogi 2006: 448). The binding of the released glutamate neurotransmitters to the AMPA-K receptors in the receiving zones of the homonymous α motor neurons will cause depolarization of them. If the summation produces excitatory post-synaptic potentials (EPSP) that reach firing threshold at the axon hillock of the depolarized homonymous α motor neurons, action potentials will be generated and cause firing of them (Lundy-Ekman 2013: 49; FitzGerald *et al.* 2012: 86, 87; Siegel *et al.* 2010: 100). The produced graded potentials in the homonymous α motor neurons induced by the activated biceps brachii muscle spindle afferents will be directly proportional to the degree to which the biceps brachii muscle spindles are passively and / or actively stretched (Haines 2013: 328).

The induced autogenic excitation via the biceps brachii muscle Ia afferent monosynaptic spinal reflex arc in response to the passive biceps brachii muscle spindles stretch will cause reinforcement of the extrafusal muscle fibers contraction of the biceps brachii and brachialis muscles (Haines 2013: 358; Fitzgerald *et al*. 2012: 125, 126; Merletti and Parker 2004: 12), in order to resist the passive stretching of the biceps brachii muscle (Bandy and Sanders 2007: 59). In addition, the facilitated biceps brachii muscle Ia afferent monosynaptic spinal reflex arc will cause an unloading effect on the biceps brachii muscle spindles and thus on the intrafusal fibers of the biceps brachii muscle, which will reinforce the muscle tension in the biceps brachii muscle via the gamma loop (Haines 2013: 328; FitzGerald *et al*. 2012: 125, 126; Merletti and Parker 2004: 12).

The excitatory synapses of the biceps brachii muscle spindle afferents on the homonymous α motor neurons may not be effective enough to drive the homonymous α motor neurons to firing threshold, but may solely increase the excitability of the homonymous α motor neurons (Mense and Gerwin 2010: 208). The increase in resistance to the passive stretching of the biceps brachii muscle and thus the increase in biceps brachii muscle tone during the passive stretching may be predominantly attributed to the viscoelastic property of the biceps brachii muscle tissue. The small quantity of slowly cycling formation of cross-bridges between the contractile proteins actin filaments and myosin heads as well as the property of the structural protein titin in the biceps brachii muscle fiber's sarcomere, may cause an increase in tensile force of the biceps brachii muscle in response to the passive stretching of the biceps brachii muscle (Lundy-Ekman 2013: 187, 188; Brodal 2010: 297; Mense and Gerwin 2010: 212).

2.5.3 INDUCED AUTOGENIC INHIBITION

2.5.3.1 ACTIVATION OF THE GOLGI TENDON ORGAN

The generated increase in tension in the biceps brachii muscle tissue induced by the facilitated biceps brachii muscle Ia afferent monosynaptic spinal reflex arc and the viscoelastic property of the biceps brachii muscle tissue during the passive stretching of the biceps brachii muscle, will result in the activation of the GTOs at the musculotendinous junctions of the biceps brachii muscle (Mileusnic and Loeb 2006: 1789; Merletti and Parker 2004: 13). The contraction of the extrafusal fibers of the biceps brachii muscle of which at least one will insert into a GTO and intertwine with the loosely packed innervated collagen fibrils inside the GTO, induced by the passive stretching of the biceps brachii muscle spindles, will in turn cause the GTOs to stretch (Mileusnic and Loeb, 2006; Merletti and Parker 2004: 13). The stretching of the GTOs will cause the mechanical opening of their transduction ion channels in the surface membrane of the sensory terminals of the biceps brachii muscle Ib afferent endings in the lumen of each GTO (Haines 2013: 329; Mileusnic and Loeb 2006: 1789, 1790; Merletti and Parker 2004: 13), and result in depolarization of the biceps brachii muscle Ib afferent endings (Lundy-Ekman 2013: 27; FitzGerald *et al.* 2012: 83, 84, 125, 126). The summation will raise the receptor membrane potential of the biceps brachii muscle Ib afferent endings above threshold at their trigger zones and cause the generation of action potentials in a sequential manner in the biceps brachii muscle Ib afferents of their unmyelinated collateral branches which intertwine among the loosely packed collagen fibers inside the GTOs, thus causing them to fire (FitzGerald *et al.* 2012: 125, 126; Merletti and Parker 2004: 12). The biceps brachii muscle Ib unmyelinated collateral branches will join each other to form larger myelinated branches which will run from the muscle end and tendon end of each GTO to form predominantly a single large myelinated Ib afferent which exits each GTO capsule near its equator (Mileusnic and Loeb 2006: 1789, 1790; Merletti and Parker 2004: 13).

The activated biceps brachii muscle GTO Ib afferents will discharge their excitatory nerve impulses in the form of generated action potentials to their first-order set of sensory neurons (Jacobson and Marcus 2011: 64) via the musculocutaneous nerve (Biller 2012: 246; Muscolino 2008: 265) and thereafter via the spinal nerves in the distal intervertebral foramina between the pedicles of successive vertebrae (Fitzgerald *et al*. 2012: 38; Steward 2012: 25) at spinal segmental levels C5/C6 (Biller 2012: 246; Muscolino 2008: 265). The impulses from the biceps brachii muscle GTO Ib afferents will travel along the centripetal processes from their dorsal root ganglia to the entry zone of the spinal cord via the dorsal nerve roots (FitzGerald *et al*. 2012: 177, 178) to enter the dorsal horn of the spinal cord at the posterolateral sulcus via the medial bundle fibers of the dorsal nerve roots (Fitzgerald *et al.* 2012: 38; Steward 2012: 25, 26). Fibers of the biceps brachii muscle

GTO Ib afferents have projections via their collateral branches into laminae VI and VII in the intermediate grey matter of the spinal cord to form excitatory axodendritic and / or axosomatic glutamergic synapses on Ib internuncials (Greger and Windhorst 2013: 1001; Brushart 2011: 73). The impulses from the biceps brachii muscle GTO afferents will reach their synaptic terminals in the intermediate grey matter to cause depolarization of their synaptic terminals (Steward 2012: 25, 26; Greger and Windhorst 2013: 1001). The released glutamate neurotransmitters from the depolarized synaptic terminals of the activated biceps brachii muscle GTO Ib afferent will diffuse across the synaptic cleft to bind to their specific ionotropic receptors in the membrane of the Ib internuncials at their receiving zones, namely the AMPA and kainite receptors (Lundy-Ekman 2013: 49; Rastogi 2006: 448). The binding of the released glutamate neurotransmitters to the AMPA-K receptors in the receiving zones of the Ib interneurons will cause depolarization of the Ib internuncials. The summation will produce EPSPs that reach firing threshold at the trigger zone of the depolarized Ib internuncials; action potentials will be generated and cause firing of them (Lundy-Ekman 2013: 49; FitzGerald *et al*. 2012: 86, 87; Siegel *et al*. 2010: 100). The produced graded potentials in the Ib internuncials induced by the activated biceps brachii muscle GTO Ib afferents will be directly proportional to the degree of generated force and active tension in the biceps brachii muscle (Mileusic and Loeb 2006: 1789; Merletti and Parker 2004: 13).

The recruited Ib internuncials in the intermediate grey matter of the spinal cord via the activated biceps brachii muscle GTO Ib afferent will cause autogenic inhibition of the homonymous α motor neurons and possible the y motor neurons innervating the biceps brachii and brachialis muscles, by forming, in turn, inhibitory glycinergic synapses on the lower motor neurons (Greger and Windhorst 2013: 1001; Brushart 2011: 73). The released glycine neurotransmitters from the depolarized synaptic terminals of the recruited Ib internuncials will diffuse across the synaptic cleft to bind to their specific ionotropic glycine receptors (GlyR) in the membrane of the homonymous α motor neurons and possibly the y motor neurons at their receiving zones (Lundy-Ekman 2013: 60, 62; Fitzgerald *et al.* 2012: 99, 100). The binding of the released glycine neurotransmitters to the GlyR in the receiving zones of the lower motor neurons will cause their hyperpolarization. The summation will produce inhibitory post-synaptic potentials (IPSP) that will drive the membrane potential at the axon hillock of the homonymous α motor neuron away from firing threshold to prevent the generation of action potentials, and thus inhibit their firing (FitzGerald *et al.* 2012: 86, 87; Siegel *et al.* 2010: 100; Rastogi 2006: 488). The produced IPSPs in the homonymous α motor neurons induced by the activated biceps brachii muscle GTO Ib afferents, will also be directly proportional to the degree of generated force and active tension in the biceps brachii muscle (Mileusic and Loeb 2006: 1789; Merletti and Parker 2004: 13). Facilitation of the GTO Ib inhibitory

di-synaptic reflex arc will result in a decrease in the excitability of the homonymous α motor neurons (Mense and Gerwin 2010: 208).

2.5.3.2 LINEAR SUMMATION OF Ib AFFERENT ACTIVITY

2.5.3.2.1 ACTIVATION OF THE LATERAL CORTICOSPINAL TRACT

A voluntary constant low force isometric contraction of the biceps brachii muscle for several seconds during the passive stretching of the biceps brachii muscle by resisting against the weight of a 1 kg plate will activate the biceps brachii muscle GTOs maximally by inducing linear summation of the biceps brachii muscle Ib afferent activity (Bandy and Sanders 2007: 59; Mileusnic and Loeb 2006: 1790; Sharman *et al*. 2006: 932). Voluntary contraction of the biceps brachii muscle is mediated by several highly interconnected adjacent motor areas in the frontal lobe of the cerebral cortex of the brain including the premotor cortex on the lateral surface of the hemisphere, the supplementary motor area on the medial surface of the hemisphere and the primary motor cortex in the precentral gyrus. These motor areas also receive regulatory input from the cerebellum and basal ganglia in the brain via their relays in the thalamus, as well as from the somatic sensory regions of the parietal lobe (FitzGerald *et al.* 2012: 187; Snell 2010: 155; Mendoza and Foundaz 2007: 10). The axons of the corticospinal tracts which serve as the major voluntary descending motor pathway originate mainly from the upper motor neurons in the primary motor cortex known as the Giant cells of Betz. The corticospinal tract will descend through the corona radiata and posterior limb of the internal capsule to enter the cerebral peduncle at the base of the midbrain in the brainstem. These fibers continue their descent through the basilar pons to reach the ventral surface of the medulla oblongata where they will form the medullary pyramids. Above the spinomedullary junction 90% of the pyramidal fibers will cross the midline in a pyramidal decussation to form the lateral corticospinal tract which occupies the lateral column of the spinal cord on the contralateral side (FitzGerald *et al*. 2012: 187; Snell 2010: 155; Mendoza and Foundaz 2007: 11). The LCST fibers have projections to the intermediate grey matter and anterior horn of the spinal cord approximately at spinal levels of C5/C6 to form excitatory glutamergic synapses on lower motor neurons innervating the biceps brachii and brachialis muscles and on Ia inhibitory internuncials which in turn form inhibitory synapses on α motor neurons innervating the triceps brachii muscle. During voluntary contraction of the biceps brachii muscle the LCST will first excite the Ia inhibitory internuncials to cause relaxation of the triceps brachii muscle, to permit the contraction of the biceps brachii muscle. Immediately thereafter, the LCST will cause co-activation of the α motor neurons and y motor neurons innervating the biceps brachii muscle to allow the function of the prime mover (FitzGerald *et al*. 2012: 188; Snell 2010: 156; Mendoza and Foundaz 2007: 11). The impulses in the LCST generated by the voluntary effort will reach their synaptic

terminals in the intermediate grey matter and anterior horn to cause depolarization of their synaptic terminals (Steward 2012: 25, 26; Mendoza and Foundaz 2007: 11). The released glutamate neurotransmitters from the depolarized synaptic terminals of the LCST fibers will diffuse across the synaptic cleft to bind to their specific ionotropic receptors in the membrane of the lower motor neurons at their receiving zones, namely the AMPA and kainite receptors (Lundy-Ekman 2013: 49; Rastogi 2006: 448). The binding of the released glutamate neurotransmitters to the AMPA-K receptors in the receiving zones of the lower motor neurons will cause their depolarization. The summation will produce EPSPs that reach firing threshold at the trigger zone of the depolarized lower motor neurons and action potentials will be generated which will cause them to fire (Lundy-Ekman 2013: 49; FitzGerald *et al*. 2012: 86, 87; Siegel *et al*. 2010: 100). The produced graded potentials in the lower motor neurons induced by the activated LCST fibers will be directly proportional to the effort of the individual to voluntary contract his or her biceps brachii muscle, that is to say, during the active stretch of the biceps brachii muscle (Fitzgerald *et al*. 2012: 178; Snell 2010: 155; Mendoza and Foundaz 2007: 10).

The force generated in the actively stretched biceps brachii muscle is modulated by a combination of motor unit recruitment and firing frequency (rate coding) of the α motor neurons innervating the biceps brachii muscle (Lundy-Ekman 2013: 192; Merletti and Parker 2004: 6). The nervous system does not control the firing rate of an individual α motor neuron, but rather modulates the motor neuron pool as a whole in a uniform fashion. The nervous system excites or inhibits the entire motor neuron pool to achieve the demand for force modulation, known as the common drive. When a motor neuron pool is depolarized during voluntary muscle contraction it is believed that there is a specific sequence of motor unit recruitment in order of increasing size of the α motor neurons, known as the size principle (Lundy-Ekman 2013: 192; Merletti and Parker 2004: 6, 7). This is because the firing threshold of an α motor neuron is determined by its total electrical resistance, which is inversely proportional to its surface area. Therefore, during depolarization of a motor neuron pool the smaller α motor neurons of low-threshold motor units which are characterized by more high fatigue-resistant muscle fibers (type I and IIa) will be first recruited, before the larger α motor neurons of higher-threshold motor units which are characterized by more high fatigable muscle fibers (type IIb and IIa) will be recruited (Haines 2013: 326; Lundy-Ekman 2013: 192; Merletti and Parker 2004: 7). When an α motor neuron is activated there is synchronized depolarization of all its extrafusal muscle fibers in an all-or-nothing phenomenon (Merletti and Parker 2004: 2, 7). Thus, when an α motor neuron fires, an end-plate potential will always result in the motor end plates of all of the extrafusal muscle fibers innervated by the recruited motor unit in normal conditions, in an all-or-nothing phenomenon (Bear and Conners 2007: 110, 111).

A voluntary constant isometric contraction of the biceps brachii muscle during the passive stretching of the biceps brachii muscle by resisting against the weight of a 1 kg plate, will cause co-activation and recruitment of both the α motor neurons of low-threshold motor units innervating the extrafusal fibers of the biceps brachii muscle, as well as the y motor neurons innervating the intrafusal fibers of the biceps brachii muscle (Lundy-Ekman 2013: 192; Merletti and Parker 2004: 7). There will be no unloading effect of the extrafusal fibers on the intrafusal fibers of the biceps brachii muscle, due to the co-activation of the α motor neurons and y motor neurons (Fitzgerald *et al*. 2012: 125, 126; Merletti and Parker 2004: 12). The lower motor neurons myelinated efferent fibers innervating the biceps brachii muscle in the anterior grey horn of the spinal cord will discharge their excitatory nerve impulses in the form of generated action potentials in a sequential manner at the anterolateral surface of the spinal cord via the ventral nerve roots (Fitzgerald *et al.* 2012: 38; Steward 2012: 25). The generated and propagated action potentials in the fibers of the low motor neurons efferent will travel along the ventral nerve roots to join the dorsal nerve roots and continue as the spinal nerves at the distal intervertebral foramina between the pedicles of successive vertebrae (Fitzgerald *et al*. 2012: 38; Steward 2012: 25) at spinal segmental levels of C5/C6 (Biller 2012: 246; Muscolino 2008: 265), and thereafter travel along the anterior rami division of spinal nerves before exiting the intervertebral foramina (Fitzgerald *et al*. 2012: 38; Steward 2012: 25). The spinal nerves and ventral rami of spinal nerves are thus mixed nerves and contain both the somatic sensory afferent and motor efferent fibers (Fitzgerald *et al.* 2012: 38; Steward 2012: 25). The ventral rami of spinal nerves originating at spinal segmental levels of C5/C6 give rise to the superior trunk followed by the superior anterior division and lateral cord of the brachial plexus. The musculocutaneous nerve extends from the lateral cord of the brachial plexus opposite the lower border of the pectoralis minor muscle, pierces the coracobrachialis muscle and descends laterally between the biceps brachii and brachialis muscles to the lateral side of the arm. The MCN innervates the coracobrachialis muscle, both the heads of the biceps brachii and the brachialis muscles during its descent (Biller 2012: 246; Hansen 2010: 305, 309; Loukas and Aqueelah 2005: 101). The axons of the α motor neurons innervating the biceps brachii muscle via the MCN branch within the belly of the biceps brachii muscle to form nerve plexuses from which groups of axons can emerge to innervate the motor end plates of the individual extrafusal muscle fibers of their motor units at about halfway along the biceps brachii muscle. At the myoneural junction, the axons of the α motor neurons innervating the biceps brachii muscle lose their myelin sheath to divide into several axonal branchlets which form synaptic terminals containing synaptic vessels loaded with acetylcholine (ACh) (FitzGerald *et al*. 2012: 121, 122; Siegel *et al*. 2012: 98). The axons of the y motor neurons (also known as the fusimotor fibers) innervating the biceps brachii muscle divide within the spindles of the biceps brachii muscle to

supply the intrafusal muscle fibers at both ends of the spindles deep to the extrafusal fibers of the biceps brachii muscle (Fitzgerald *et al*. 2012: 122, 123).

2.5.3.2.2 CO-ACTIVATION OF THE ALPHA AND GAMMA MOTOR NEURONS

The generated impulses from the excited α motor neurons innervating the biceps brachii muscle by the voluntary effort, will reach their synaptic terminals at the neuromuscular junction of all of the biceps brachii muscle extrafusal fibers of the recruited motor units to cause depolarization of their terminal endings (Lundy-Ekman 2013: 192; Merletti and Parker 2004: 2, 7). The released ACh neurotransmitters from the depolarized synaptic terminals of the excited biceps α motor neurons efferent fibers will diffuse across the synaptic clefts to bind to their specific ionotropic nicotinic cholinergic receptors in the crest of the junctional folds in the sarcolemma of each recruited biceps brachii muscle extrafusal fiber (Lundy-Ekman 2013: 49; Conn 2008: 100, 101; Bear and Conners 2007: 110, 111; Rastogi 2006: 448). The binding of the released ACh neurotransmitters to the nicotinic cholinergic receptors at their neuromuscular junctions will depolarize the motor end plates of each recruited biceps brachii muscle extrafusal fiber (Fitzgerald *et al.* 2012: 86). Each ACh ligand binding to its nicotinic cholinergic receptor will generate a small postsynaptic potential change of a fixed size at each depolarized motor end plate, known as the miniature end-plate potential (mepp). The binding of several ACh neurotransmitters to their nicotinic cholinergic receptors will produce mepp summation and result in the generation of a larger depolarization of each motor end plate, to generate an end-plate potential (EPP). When the EPP is large enough and reaches the threshold, the generation of an action potential will result and cause contraction of each recruited biceps brachii muscle extrafusal fiber (Siegel *et al*. 2012: 98; FitzGerald *et al*. 2012: 122. 123). The produced EPPs in the recruited biceps brachii muscle extrafusal fibers induced by the activated LCST fibers will be directly proportional to the effort of the individual to voluntary contract his or her biceps brachii muscle during the active stretch of the biceps brachii muscle (Fitzgerald *et al*. 2012: 178; Snell 2010: 155; Mendoza and Foundaz 2007: 10).

Active stretching of the biceps brachii muscle will cause contraction and thus shortening of the biceps brachii muscle intrafusal fibers via the activated fusimotor fibers and in turn cause stretching of the equatorial region of the biceps brachii muscle spindles thereby causing the activation of the biceps brachii muscle spindle afferents (Haines 2013: 327, 328). Actively stretched muscle spindles of the biceps brachii muscle will reinforce the excitation on the homonymous α motor neurons via the gamma loop with reciprocal inhibition of the α motor neurons innervating the triceps brachii muscles (FitzGerald *et al.* 2012: 190; Snell 2010: 156, 157; Gupta and Pasley 1996: 116). The gamma loops from the passively stretched spindles of the

triceps brachii muscle are not activated due to these muscle spindles still being refractory (FitzGerald *et al*. 2012: 190; Snell 2010: 156, 157; Gupta and Pasley 1996: 116).

2.5.4 NEURONAL CONVERGENCE ON THE HOMONYMOUS MOTOR NEURON POOL

The α motor neuron is the final point of summation for all the descending motor pathways and spinal reflex arcs (Merletti and Parker 2004: 2). Thousands of axons synapse on an α motor neuron cell. To put the convergent input on an α motor neuron in perspective, the extensive dendritic tree of an α motor neuron can allow about 50 000 individual synapses on it (Jacobson and Marcus 2011: 67) via multiple, independent spinal pathways (Rice and McNair 2010:256) which can be excitatory or inhibitory in nature (Jacobson and Marcus 2011: 67). The α motor neuron at each segmental spinal level can receive input from descending motor pathways, type Ia and IIa muscle spindle afferents, type Ib GTO afferent, joint afferents of type I-IV nerve ending, Ia inhibitory internuncials, Ib inhibitory internuncials, excitatory internuncials and from Renshaw cells (Fitzgerald *et al.* 2012: 186, 187; Merletti and Parker 2004: 2). In addition, excited or facilitated nociceptive afferents from the skin, homonymous muscle, heteronymous muscle, and from a dysfunctional joint can produce an excitatory or inhibitory influence on the homonymous motor neuron pool excitability (Mense and Gerwin 2010: 229, 230). In addition, the viscoelastic property of the biceps brachii muscle tissue can influence the contractile activity of the biceps brachii muscle, and thereby produce alterations in the homonymous motor neuron pool excitability (Lundy-Ekman 2013: 229; Mense and Gerwin 2010: 221). Studies have suggested that alterations in the motor neuron pool excitability may be partly due to a subconscious adjustment in voluntary effort in the form of an emotional component such as excitement, fear thereby causing more pain or damage to a body part. Studies that investigate changes in muscle activation rely on the voluntary effort of free will by the participant. By including asymptomatic participants, one may exclude the subconscious adjustment in extra voluntary effort caused by pain (Rice and McNair 2010: 259; Engelhardt *et al*. 2001: 75). In the current study the participants were asymptomatic and the effects of the nociceptive spinal pathways including the effects of the nociceptive afferents from the skin, homonymous muscle, heteronymous muscle and from joints on the homonymous motor neuron pool excitability could be excluded from the study.

The final induced convergent spatial summation on the homonymous α motor neurons from the various synapses on them will determine whether the firing thresholds of the axon hillock of each homonymous α motor neuron is reached so as to generate action potentials and cause firing of the homonymous α motor neurons (Fitzgerald *et al.* 2012: 87) in reference to the whole motor neuron pool in a uniform fashion from low to high threshold motor unit recruitment, depending on the level of force and speed of muscle contraction by the voluntary effort of the individual (Mileusnic and

Loeb 2006: 1790; Merletti and Parker 2004: 2, 6). Therefore, the net membrane current induced in an α motor neuron will determine the firing frequency and the electrical activity of its motor unit (Fitzgerald *et al*. 2012: 186, 187; Merletti and Parker 2004: 2).

Because the wiring of the α motor neuron entails multiple spinal segmental and supraspinal inputs, a single input may be negligible to trigger the firing threshold of the homonymous α motor neurons and may solely influence the excitability of them. The summate balance between all these influences may decide if the homonymous α motor neurons increase or decrease their discharge frequency or excitability (Mense and Gerwin 2010: 229). The linear GTO Ib afferent activity of the biceps brachii muscle induced by the modified stretching may produce IPSPs in the homonymous α motor neurons and thereby reduce the excitability state of the homonymous motor neuron pool (Bandy and Sanders 2007: 59; Mileusnic and Loeb 2006: 1790; Sharman et al. 2006: 932) to facilitate the elongation of the biceps brachii muscle during the stretching (Umphred *et al*. 2013: 209; Sharman *et al*. 2006: 932). A decrease in the EMG amplitude (Root Mean Square) along with a decrease in biceps brachii muscle force may result (Lundy-Ekman 2013: 192; Merletti and Parker 2004: 6, 7).

2.6 THE EFFECT OF SPINAL MANIPULATION ON BICEPS BRACHII MUSCLE ACTIVITY

The neurophysiological effects of spinal manipulation on AMI is unclear despite the objective evidence of the reduction of AMI and increase in muscle EMG activity post spinal manipulation (Dunning and Rushton 2009: 512; Picker 2002: 358, 364, 365; Suter and McMorland 2002: 541). Suter and McMorland (2002: 541) applied spinal manipulation to C5/C6/C7 segments with evidence of motor inhibition of the biceps brachii muscle using an interpolated twitch technique and EMG. Their results showed a significant reduction in biceps brachii muscle inhibition and an increase in biceps brachii muscle force post spinal manipulation. The mechanism responsible for the improved functional capacity of the biceps brachii muscle after the spinal manipulation is not fully understood (Suter and McMorland 2002: 544). There is a gap in the chiropractic literature regarding the neurophysiological effect of spinal manipulation on AMI (Dunning and Rushton 2009: 509) as well as on the underlying neurophysiological mechanism of spinal manipulation on motor activity (Olsen 2015: 86; Pickar 2002: 364).

Spinal manipulation can improve muscle functioning either by facilitation or disinhibition of the involved neural pathways (Dunning and Rushton 2009: 511, 512; Pickar 2002: 359, 360; Suter and McMorland 2002: 543, 544). Dunning and Rushton (2009: 508) found a significant increase in biceps brachii muscle activity post spinal manipulation of the ipsilateral C5/C6 segment in

asymptomatic patients for neck pain and bilateral upper extremity pain, and based the underlying neural mechanism on an excitatory effect of spinal manipulation on the motor activity. Suter *et al*. (2000: 385) found a significant decrease in inhibition of the knee extensor muscles post manipulation of the ipsilateral sacroiliac joint in symptomatic patients with sacroiliac syndrome, anterior knee pain and EMG evidence of motor inhibition of the knee extensor muscles, and based the underlying neural mechanism on disinhibition of the involved neural pathways.

CHAPTER 3 : METHODOLOGY

3.1 INTRODUCTION

This chapter describes the manner in which the research study was conducted.

3.2 STUDY DESIGN

A single-blinded, repeated measures design was employed on asymptomatic individuals (n=20) (Dunning and Rushton 2009: 508).

3.3 POPULATION

Any individual who was asymptomatic for neck pain and bilateral upper extremity pain and presented himself or herself to the Durban University of Technology Chiropractic Day Clinic in response to the advertisement (Appendix G) was subjected to a screening based on inclusion and exclusion criteria (below). Advertisements were placed on notice boards at the DUT including all the campuses and residences, CDC and around the DUT Berea and City campuses. Active recruitment by word of mouth was also utilized. The prospective participants were requested to contact the researcher telephonically for more information.

All of the prospective participants were informed that this was the preliminary selection and further inclusion and exclusions would be applied during the first consultation. The prospective participants were asked questions during the telephonic conversation to see if they qualified (Appendix H). If any of the participants did not meet the telephonic preliminary selection, they were excluded from participating in this study.

3.3.1 INCLUSION CRITERIA

Only participants between the ages of 18 and 40 were selected. No parental consent was required as all the participants were over the age of 18. Degenerative changes in the intervertebral disc and posterior facet articulations commonly occur after the age of 40 (Kelly *et al*. 2012: 1).

Inclusion criteria:

- Participants fluent (write or speak) in English or Afrikaans were selected, to enable them to read and sign the informed consent form and to follow the verbal instructions of the researcher.

- Participants asymptomatic for neck pain and bilateral upper extremity pain were selected.
- Participants must have completed and signed an informed consent form (Appendix A).

3.3.2 EXCLUSION CRITERIA

- A history of neck pain in the past two weeks (Yoshimizu *et al*. 2012: 97).
- A history of surgery to the cervical spine or to the upper extremities (Macrae 2001: 88).
- A history of acute injury or trauma to the cervical spine or to the upper extremities in the past two months, such as a whiplash injury to the cervical spine or a biceps brachii muscle strain or rupture (Macrae and Davies 1999: 125).
- Any shoulder joint dysfunction or pain and any elbow joint dysfunction or pain.
- Participants who exhibited signs of upper or lower motor neuron disease / lesion and any peripheral neuropathy (Fitzgerald *et al*. 2012: 191, 192).
- Any contraindication for spinal manipulation therapy (Puentedura *et al*. 2012: 66). The clinical presence of any contraindication for spinal manipulation or any indication for further special investigations would have excluded the patient from the study. This was assessed during the case history (Appendix B), physical examination (Appendix C) and the cervical spine regional examination (Appendix D).

3.4 SAMPLE SIZE AND STRATEGY

A sample of 20 participants, regardless of race and gender, who met the inclusion and exclusion criteria, participated in the study (Carpenter 2015). The sample size was chosen based on other similar studies in the physiological literature (Dunning and Rushton 2009: 508). Participants served as their own controls. The data recorded pre-intervention for each participant served as the control. Each participant received all interventions in a single appointment.

3.5 INSTRUMENTS

The dependent variables included muscle force and EMG RMS. The instruments used to measure these variables are described below. The independent variables were delivered using a Biopac - Bionomadix complete wireless research system with four channel EMG recording with Biopac EL509 electrodes, a Biopac - TSD121C Hand Dynamometer 100kg and amplifier, and an Activator Methods®– ii Activator Adjusting Instrument.

3.5.1 SURFACE ELECTROMYOGRAPHY

Surface EMG is a non-invasive technique for measuring muscle electrical activity that occurs during cycles of muscle contraction and relaxation (Merletti and Parker 2004: 6, 7). A Biopac -

Bionomadix complete wireless research system with four channel EMG recording, and Biopac EL509 electrodes of 27 mm x 36 mm in dimension with Ag/AgCl laminated carbon composition contacts and incorporated electrode gel cavity (16 mm diameter and 1.5 mm deep) were used. Signal to noise ratio, distortion of the EMG signal and cross talk susceptibility between adjacent muscles can influence the fidelity of the EMG signal recorded (De Luca 2002: 3, 6).

By using the stated small electrode size and a standardized inter-detection surface spacing of 1 cm, the cross-talk can be minimized. The average nerve conduction velocity of skeletal muscles is 4.0 m/s. By using high quality electronic components, intelligent circuit design and construction techniques, a bandwidth of SEMG frequency between 20 Hz and 450 Hz can be used to capture the full frequency spectrum of the electromyographic signal and to suppress the distortion of the signal and noise at higher frequencies, as well as to suppress the quasi-random nature of the firing rate of the motor units at lower frequencies (Dunning and Rushton 2009: 509; Quach 2007: 25, 26; Suter and McMorland 2002: 542). The AcqKnowledge software was used to process the electromyographic signals to obtain the digital values for the identified variables which were stored on the hard drive directly of the researcher's personal computer (LENOVA G50 Laptop).

The Integrated EMG (iEMG) is defined as the area under the curve of the rectified EMG signal; the mathematical integral of the absolute value of the raw EMG signal. When the absolute value of the EMG signal is taken, noise will make the mathematical integral have a constant increase. Integrated EMG splits up the EMG signal into fixed-width time slices and resets the integral at the start of each time slice. The iEMG is measured in microvolts. In addition to the true iEMG, the AcqKnowledge Root Mean Square analysis routine outputs a second waveform for the maximum value of the iEMG signal in each time slice by using the Integrate transformation in a RMS Average over Samples configuration. This maximum iEMG is easier to interpret visually and approximates the envelope of the iEMG signal. The EMG RMS is also measured in microvolts (BIOPAC® System, Inc. 2016). The EMG RMS is considered to provide the most insight on the amplitude of the EMG signal, because it gives a measurement of the power of the signal and produces a waveform that is easily analyzable (Merletti and Parker 2004: 172; Suter and McMorland 2002: 542).

3.5.2 DYNAMOMETRY

A dynamometer is an instrument used for measuring the force of muscular contraction in kilograms. Studies have shown that a dynamometer has high levels of both intra- and inter-reliability for testing muscle strength (Maayah *et al.* 2012: 123). The dynamometer (Biopac - TSD121C Hand Dynamometer 100 kg and amplifier) was used to quantify the constant muscle

force output during the modified stretching of the biceps brachii muscle in order to reduce the effect of extra voluntary effort by the participant's response to the SEMG readings.

3.5.3 ACTIVATOR ADJUSTING INSTRUMENT

An Activator Adjusting Instrument (AAI) (Activator Methods® – ii) is a hand held instrument used to deliver a high velocity-low amplitude mechanical force, to produce a spinal manipulation. The AAI with a force setting of zero force can be used to deliver a placebo spinal manipulation. There are no validity or reliability issues regarding the Activator II Adjusting Instrument. Several studies have used the AAI as a placebo spinal manipulation (Humphries *et al.* 2013: 153; Huggins *et al.* 2012: 53).

3.6 INTERVENTIONS

3.6.1 MODIFIED STRETCHING OF THE BICEPS BRACHII MUSCLE

Each participant was instructed by the researcher to sit on a chair in front of an adjustable small table. The participant was further instructed by the researcher to place his or her arm on the table. The adjustable table was set by the researcher so that the participant's arm was horizontal to his or her shoulder. The elbow was extended and the forearm supinated passively by the researcher. The extended elbow remained in contact with the table and the distal supinated forearm hung off the table. A 10 mm by 2 m tie down strap was strapped to the distal forearm of the participant by the researcher. The pull side of a Biopac - TSD121C Hand Dynamometer 100 kg (the proximal part to the distal forearm) was attached to the tie down strap. Following this, a second 10 mm by 2 m tie down strap was used to attach the push side of the dynamometer (the distal part to the distal forearm) to a 1 kg plate, which hung below the distal forearm of the participant. The tie down strap attached to the 1 kg plate was only attached to the push side of the dynamometer and not directly to the distal forearm (Plate 3.1). The weight of the 1 kg plate fully extended the elbow and passively stretched the biceps brachii muscle to elicit the autogenic excitation phase of the modified stretching of the biceps brachii muscle (Bandy and Sanders 2007: 59; Sharman *et al*. 2006: 932). The participant was then instructed by the researcher to resist against the weight of the 1 kg plate for 10 seconds without flexing their elbow to perform an isometric contraction of their biceps brachii muscle and thereby to elicit the autogenic inhibition phase of the modified stretching. The participant's elbow must remain stationary to hold the biceps brachii muscle in a passively stretched position (Bandy and Sanders 2007: 59; Mileusnic and Loeb 2006: 1790; Sharman *et al*. 2006: 932). The participant was also instructed to maintain the same constant

biceps brachii muscle contraction for the whole duration of the 10 seconds of resistance to rule out most unwanted extra voluntary effort by the participant.

Each participant performed three sets of modified stretching of the biceps brachii muscle with two minute rest intervals between each set. At the fourth second during the first set of modified stretching of the biceps brachii muscle an Activator II Adjustment Instrument placebo spinal manipulation was applied to the ipsilateral C5/C6 segment (AAI 1) of each participant. At the fourth second during the second set of modified stretching of the biceps brachii muscle an Activator II Adjustment Instrument placebo spinal manipulation was applied to the ipsilateral C5/C6 segment (AAI 2) of each participant. At the fourth second during the third set of modified stretching of the biceps brachii muscle spinal manipulation was applied to the ipsilateral C5/C6 segment (SMT) of each participant.

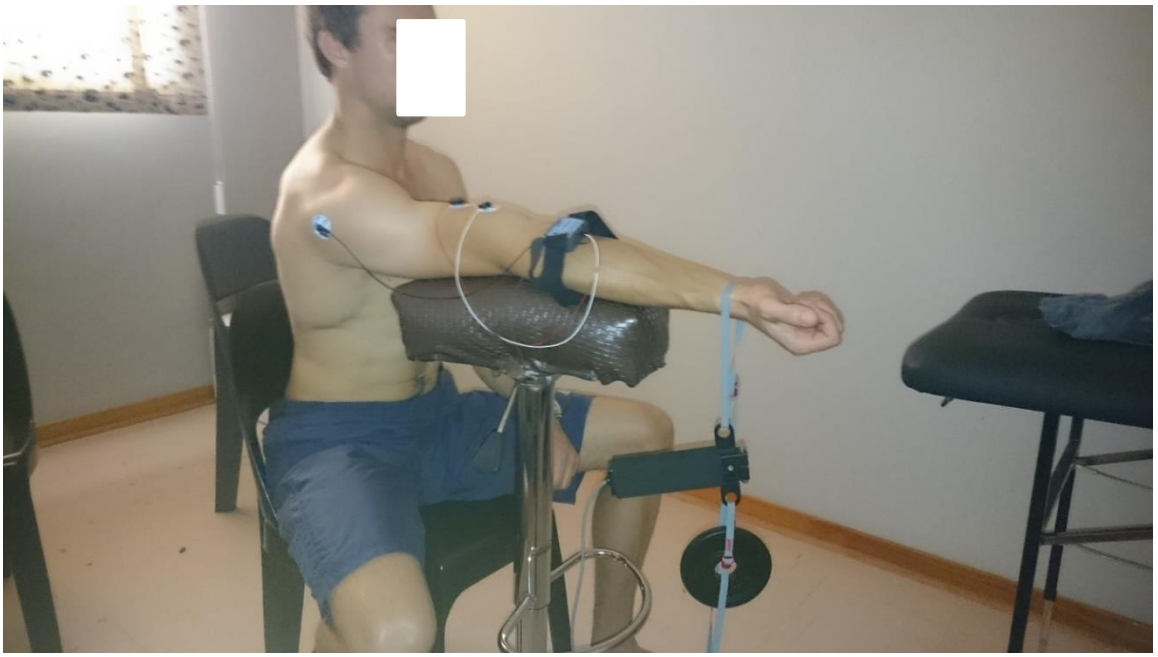


Plate 3.1: The setup of the modified stretching of the biceps brachii muscle

3.6.2 ACTIVATOR ADJUSTING II INSTRUMENT PLACEBO SPINAL MANIPULATION

During the first and second sets of modified stretching of the biceps brachii muscle, the researcher placed the AAI against the ipsilateral posterolateral surface of the articular pillar of the superior vertebra of C5/C6 spinal levels in an anterior rotational vector along the facet joint planes, at the 4th second. The researcher then pulled immediately the trigger of the AAI with a force setting of zero force at the 4th second to deliver an AAI placebo spinal manipulation (Humphries *et al.* 2013: 153). Although the AAI was set to a force setting of zero, the placebo AAI intervention still caused an audible cavitation type sound. The researcher removed the contact of the AAI from the participant immediately after the placebo spinal manipulation.

An AAI placebo spinal manipulation cannot stimulate mechanoreceptors in the facet joint capsular tissue and synovium (Humphries *et al*. 2013: 153; Huggins *et al.* 2012: 53) and may therefore have no effect on the facilitated GTO Ib inhibitory di-synaptic spinal reflex arc as part of the convergent input on the homonymous motor neuron pool excitability during the modified stretching of the biceps brachii muscle. The AAI placebo spinal manipulation was also used to account for the placebo effect to provide a quantitative assessment of the effects of the C5/C6 spinal manipulation (Temple 2003: 1130).

3.6.3 SPINAL MANIPULATION OF THE C5/C6 SEGMENT

During the third set of modified stretching of the biceps brachii muscle, the researcher contacted the contralateral posterior head of the participant with his non-adjusting hand for support and the ipsilateral posterolateral surface of the articular pillar of the superior vertebra of C5/C6 spinal levels with the dorsolateral aspect of his contact index finger (adjusting hand), at the 4th second. The researcher then rotated the facet joints in an anterior rotational vector along the facet joint planes to its end range of motion to lock up the C5/C6 facet joint. Once the barrier was engaged at end range of motion, the researcher then applied a high-velocity low-amplitude thrust in an anterior rotational vector along the facet joint planes at the 6th second to deliver the C5/C6 spinal manipulation (Bergmann and Peterson 2010: 752; Redwood and Cleveland 2003: 257). A two second time interval was given to lock up the C5/C6 facet joint. The researcher then re-positioned the participant's head to the starting position and removed any contact by the researcher from the participant immediately.

The C5/C6 spinal manipulation can stimulate mechanoreceptors in the facet joint capsular tissue and synovium (Olsen 2015: 85; Dunning and Rushton 2009: 509; Suter and Mcorland 2002: 544; Pickar 2002: 363) and may therefore have an effect on the facilitated GTO Ib inhibitory di-synaptic spinal reflex arc as part of the convergent input on the homonymous motor neuron pool excitability during the modified stretching of the biceps brachii muscle.

3.7 PROCEDURE FOR DATA COLLLECTION

After the participant met the criteria via telephonic screening, an initial appointment was scheduled at the CDC. The participant was given a verbal explanation of the research procedure on arriving for the initial appointment and was given a letter of information and informed consent (Appendix A) to read and sign. Time was given to the participant to ask the researcher any questions about this research. A case history (Appendix B), a physical examination (Appendix C) and a cervical spine regional examination (Appendix D) were performed by the researcher on each participant after the informed consent was signed.

Once the participants met the inclusion and exclusion criteria they were accepted into the study. A follow up appointment within two weeks was scheduled at the CDC, in which each participant received all the interventions. A full description of the procedure was explained verbally to each participant. Time was then given for each participant to ask any questions regarding the procedure.

The participants were told that they would perform three sets of modified stretching of the biceps brachii muscle by resisting against a 1 kg weight plate for 10 seconds with the biceps brachii muscle in a passively stretched position with two minute rest intervals between each set, and during each set of modified stretching they would receive an intervention at the fourth second. The first and second interventions would be an Activator II Adjusting Instrument. The third intervention would be spinal manipulation of the C5/C6 segment.

Before the modified stretching of the biceps brachii muscle commenced and the interventions were applied, the researcher correctly identified the C5/C6 spinal level by using specific palpation (Magee 2008: 180; Benzel 2012: 958). The researcher stood behind the participant. The researcher then palpated the occiput of the skull of the participant. The researcher descended from the occiput in the posterior midline of the participant's neck and the C2 spinous process was palpated as the first bump. The next spinous processes which were most palpable were C6 and C7. The researcher differentiated between C5, C6 and C7 spinous process by passively flexing and extending the participant's neck. With the flexion and extension movements the C5 and C6 spinous process moved in and out and the C7 spinous process remained stationary (Magee 2014: 180; Benzel 2012: 958). The C5/C6 spinal levels were marked on the participant's skin by using a non-permanent marker pen. Horizontal lines were then drawn on the participant's skin over C5 and C6 spinous processes.

Frequencies below 20 Hz and above 450 Hz were filtered out to reduce noise in the EMG signal (Dunning and Rushton 2006: 509). Devices such as Trans Electrical Nerve Stimulation, Interferential Current and ultrasound units / machines were not allowed in the vicinity of the room in which the interventions took place to exclude electrical noise (De Luca 2002: 3, 6). Participants were asked to switch off their cell phones. The lights in the room were switched off to reduce the noise. The door and windows in the room were closed to reduce electrical noise.

The dynamometer was correctly calibrated with an attached 1 kg plate to 0 kg before the modified stretching of the biceps brachii muscle commenced in order to also exclude the weight of the one kg plate during the dynamometer recordings.

Skin over the biceps brachii muscle was prepared at the sites of the SEMG electrode placement before the tie down strap with the attached dynamometer and 1 kg plate was strapped to the participant's distal forearm during the first set of modified stretching of the biceps brachii muscle. The researcher ensured that the skin was clean from dirt and dry by using a towel. The skin was then gently swiped with fine sand paper abrasion tape to remove dead skin cells that could impede the EMG signal, prior to wiping the site with a cleansing wipe and allowing the site to air dry (Quach 2007: 18; De Luca 2002: 8, 9). The stated small Surface EMG electrodes were then placed on the biceps brachii muscle of interest. Two SEMG electrodes were placed on the longitudinal midline of the biceps brachii muscle mid-way between the origin and insertion of the muscle with an inter-detection surface spacing of 1 cm. The longitudinal axes of the electrodes were aligned perpendicular to the length of the biceps brachii muscle fibers to allow intersection of most of the same muscle fibers by both electrodes and provide an EMG signal that reflected the activity of a fixed set of muscle fibers (Plate 3.2) (Quach 2007: 21, 22; De Luca 2002: 8, 9). A reference electrode was placed on the posterior aspect of the ipsilateral deltoid muscle.

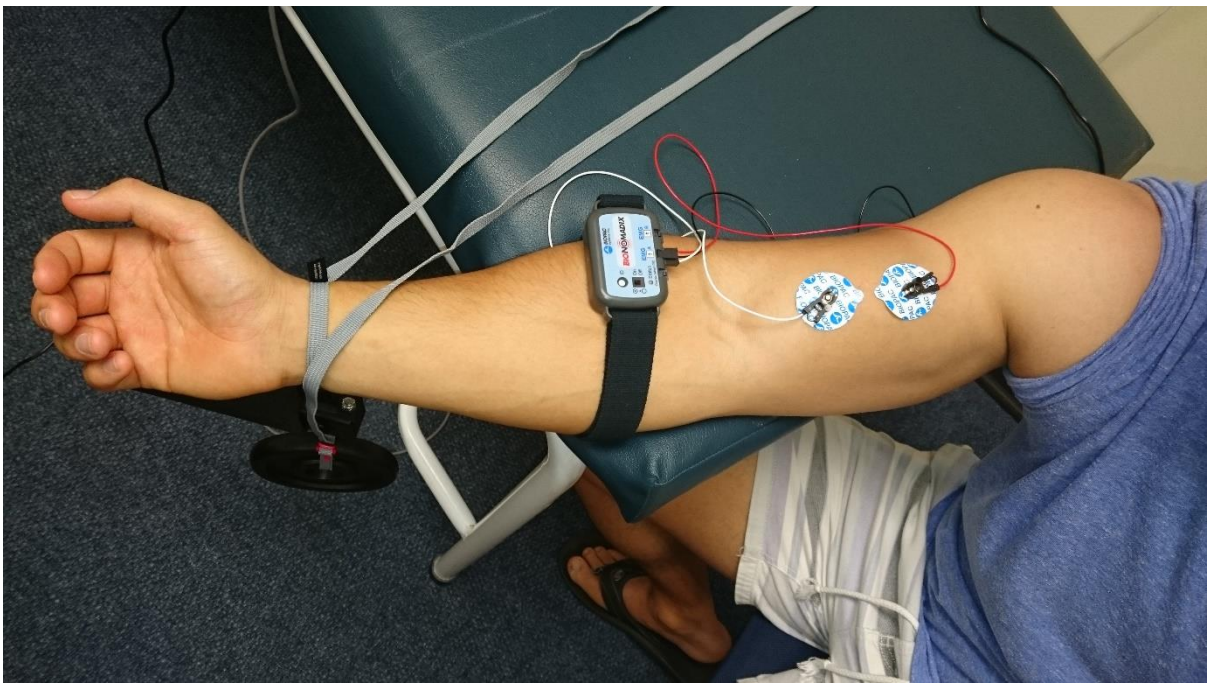


Plate 3.2: Electrode placement on the biceps brachii muscle

A stop watch was placed in front of the researcher. The participant performed the first set of modified stretching of the biceps brachii muscle. The first intervention of the placebo, AAI 1, was applied to the participant at the fourth second of the 10 seconds of resistance against the weight of the 1 kg plate during the first set of modified stretching of the biceps brachii muscle.

After the 10 seconds of resistance against the weight of the 1 kg plate during the first set of modified stretching the biceps brachii muscle, the tie down strap with the attached dynamometer and 1 kg plate was unstrapped from the participant's distal forearm by the researcher. The

participant was then instructed to rest for two minutes. The participant was also instructed to stay in the seated position for the full two minute duration of rest. The SEMG electrodes were not removed from the participant's biceps brachii muscle during the rest period.

After the two minutes of rest the participant performed the second set of modified stretching of the biceps brachii muscle. The second intervention of the placebo, AAI 2, was applied to the participant at the fourth second of the 10 seconds of resistance against the weight of the 1 kg plate during the second set of modified stretching of the biceps brachii muscle.

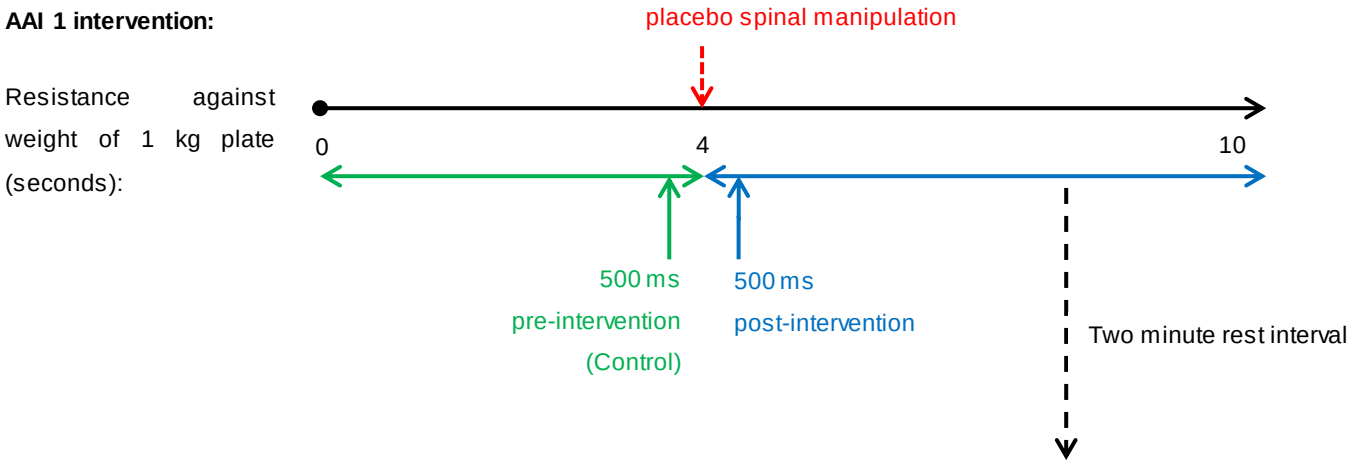
After the 10 seconds of resistance against the weight of the 1 kg plate during the second set of modified stretching of the biceps brachii muscle, the tie down strap with the attached dynamometer and 1 kg plate was unstrapped from the participant's distal forearm by the researcher. The participant was then instructed to rest for two minutes again. The participant was also instructed to stay in the seated position for the full two minute duration of rest. The SEMG electrodes were not removed from the participant's biceps brachii muscle during the rest period.

After the two minutes of rest the participant performed the third set of modified stretching of the biceps brachii muscle. The third intervention of SMT was applied to the participant at the fourth second of the 10 seconds of resistance against the weight of the 1 kg plate during the third set of modified stretching of the biceps brachii muscle.

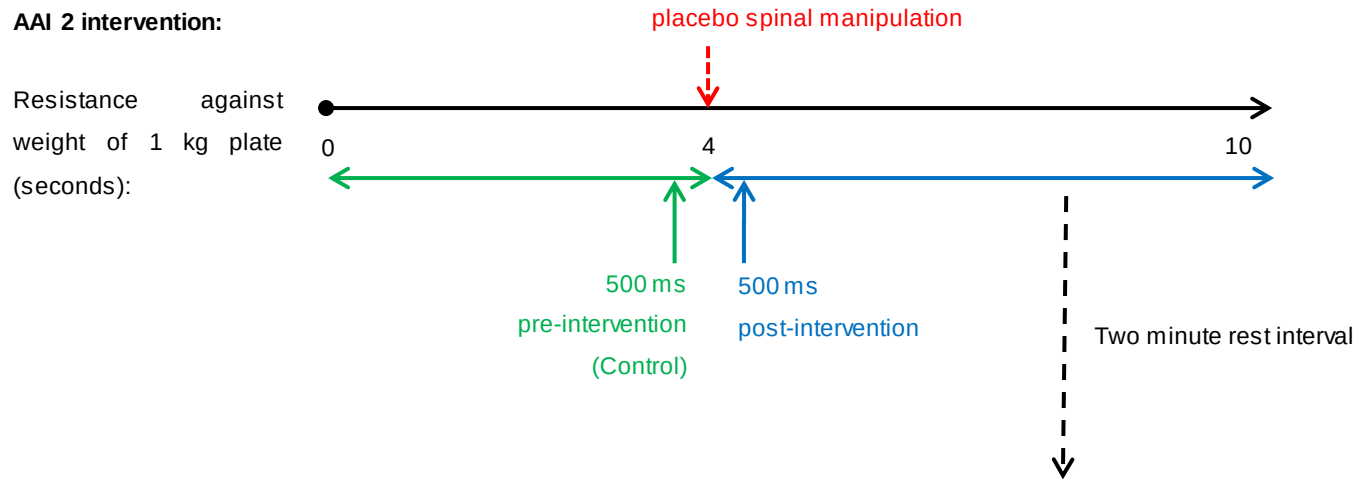
After the third set of modified stretching of the biceps brachii muscle, the researcher unstrapped the tie down strap with the attached dynamometer and 1 kg plate from the participant's distal forearm. The SEMG electrodes were then safely and correctly removed from the participant's biceps brachii muscle and the participant was thanked for his or her participation in the study. Figure 3.1 illustrates the procedure for data collection from each participant.

The raw EMG and dynamometry readings were recorded simultaneously during the entire duration of muscle activity during each set of the modified stretching of the biceps brachii muscle and stored on the hard drive directly and processed to obtain the digital values for the identified variables. Electromyographic signals were collected using the Biopac – MP 150 Data Acquisition system and processed using the AcqKnowledge software. One-second EMG segments were taken during the force plateau and the EMG signal was processed through Root Mean Square analysis using the Biopac - MP 150 Data Acquisition system and AcqKnowledge analysis software. The mean scores of all variables pre- and post-interventions were recorded (Appendix F).

First set of modified stretching of the biceps brachii muscle:



Second set of modified stretching of the biceps brachii muscle:



Third set of modified stretching of the biceps brachii muscle:

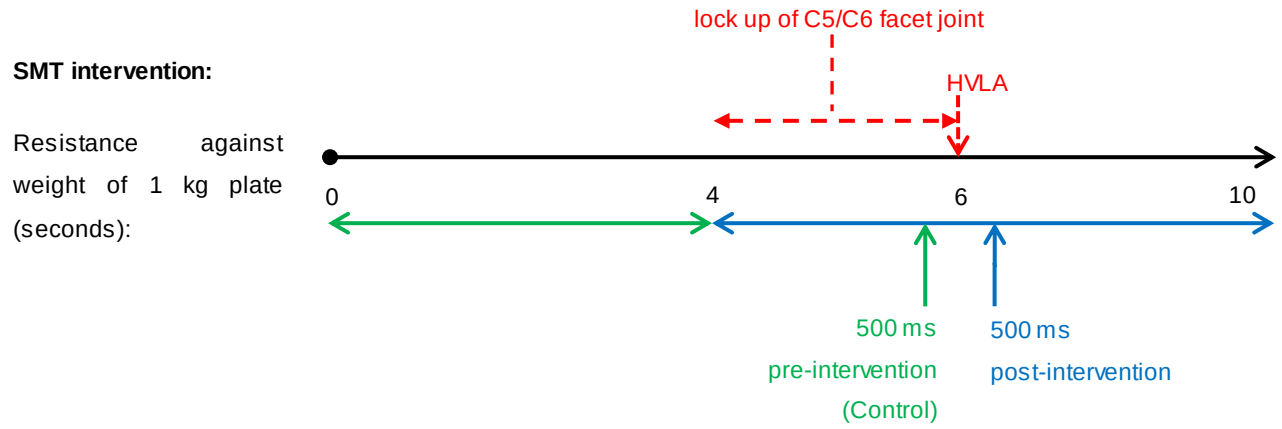


Figure 3.1: Schematic diagram illustrating the procedure for data collection for each participant

3.8 DATA ANALYSIS

The objective analysis was conducted using Stata 13.1 and consisted of descriptive statistics to analyze the data followed by bivariate and multivariate analyses to examine the effects of the intervention. The analyses made use of the mean values of each variable for the 500 millisecond period before and the 500 millisecond period after the placebo AAI 1 intervention and the placebo AAI 2 intervention at the fourth second during the modified stretching of the biceps brachii muscle. The analysis made use of the mean values of each variable for the 500 millisecond period before and the 500 millisecond period after the high-velocity low-amplitude thrust at the sixth second during the SMT intervention, during the modified stretching of the biceps brachii muscle. These values were also subtracted from one another to calculate change scores for each variable.

The bivariate analyses consisted of independent t-tests, adjusted for unequal variance where appropriate. All analyses were performed with a confidence interval of $\alpha = 0.05$, as well as an optimal α to account for the lack of power inherent in small sample designs as per Mudge *et al.*'s (2012: 32734) recommendations.

The optimal αs were determined separately for the two sample comparisons between the placebo AAI 1 and placebo AAI 2 interventions, optimal $\alpha = 0.232$, and those between the SMT intervention and both placebo AAI interventions (AAI 1 + AAI 2), optimal $\alpha = 0.203$. The calculation optimized α to find a medium effect size of $d = 0.5$ which was an equal weight for the cost of errors and a prior probability ratio of 1. The optimal α for the placebo AAI intervention comparison yielded a power of 0.647, a large improvement from that of the conventional α, power = 0.338. Similarly, the optimal α for the SMT intervention and placebo AAI interventions (AAI 1 + AAI 2) comparison yielded a power of 0.707, a large improvement from that of the conventional α, power = 0.429.

3.9 ETHICAL CONSIDERATIONS

All the participants who partook in this study were required to read and sign information and consent form specific to this study (Appendix A). The information and consent form provided the details of the researcher, supervisor and co-supervisor, and outlined the purpose, procedure and risks / benefits of this study.

All objective measurements, interventions and procedures utilized in this study, were non-invasive and were applied within the safety procedure parameters. The modified stretching of the biceps brachii muscle may lead to transient delayed-onset muscle soreness of the biceps brachii muscle. The participants were advised that this was a normal response to the modified stretching of the biceps brachii muscle, and usually abates after 48-72 hours (Hackney 2008: 1602). Side effects of

spinal manipulation may occur and include transient local soreness, discomfort, stiffness, headaches and nausea. The participants were advised that side effects of spinal manipulation are uncommon and usually abate 48-72 hours after the spinal manipulation (Ernst 2007: 330). There was no compensation in the event of an injury. If the participant developed pain or any adverse effect; the participant was withdrawn from the study immediately, the DUT Head of Department of Chiropractic was contacted and the adverse event was reported to IREC. The participant was allowed to contact his or her personal general medical practitioner; alternatively, the participant was referred to the closest local hospital with a referral letter.

Some of the participants were fellow chiropractic or DUT students and / or friends. The measurements utilized in this study were objective, and any relationship between the researcher and the participants would not affect the results. Participants were not forced to participate in the study, they did so by free will and there was no financial benefit. The participants were permitted to leave the study of their own free will at any stage without prejudice.

The rights and the welfare of the participants were protected. The participants were given codes and their names were not used on the data sheets. Only the clinic receptionist and researcher had access to the names and the codes. The SEMG readings and dynamometer readings were recorded and stored on a computer hard drive directly and processed to obtain the digital values for the identified variables via the Biopac - MP 150 Data Acquisition system and AcqKnowledge software. All of the electronic data obtained were burned on a CD. The electronic data obtained were completely removed from the hard drive, after the electronic data was burned on the CD. All of the research data will be stored in the patient file at the CDC with the electronic data obtained in the form of a CD for a period of 5 years. After the 5 year period the research data will be shredded and the CD will be destroyed.

CHAPTER 4 : RESULTS

4.1 INTRODUCTION

This chapter presents the data collected in the form of tables and graphs supported by narratives.

The following analyses were performed:

1. Demographic data: age, height, weight, gender and treatment intervention distribution; and
2. Objective data: surface electromyography and dynamometry.

4.2 DEMOGRAPHIC PROFILE

4.2.1 AGE, HEIGHT AND WEIGHT DISTRIBUTION

Participants of this study had an age range of between 19 and 31years with a mean age of 26 years (±3.3 years). The participants ranged in height from 1.57 m to 1.83 m with a mean height of 1.7 m (±0.1 m). The participants weighed between 45 kg and 100 kg with a mean weight of 70.45 kg (±13.7 kg).

4.2.2 GENDER DISTRIBUTION

The 20 participants in this study included 12 males (60 %) and 8 females (40 %).

4.2.3 DISTRIBUTION OF TREATMENT INTERVENTIONS WITH AUDIBLE CAVITATION

Each participant received two placebo AAI interventions and one SMT intervention in a single appointment. The 60 interventions applied to the 20 participants compromised 20 SMT interventions (33.3 %) and 40 placebo AAI interventions (66.7 %). For the placebo AAI interventions; 40 experienced an audible cavitation (100 %) and null experienced a non-audible cavitation (0 %). For the SMT interventions; 17 experienced an audible cavitation (85 %) and 3 experienced a non-audible cavitation (15 %).

4.3 OBJECTIVE DATA ANALYSIS

4.3.1 THE ELECTRICAL ACTIVITY AND MUSCLE FORCE IMMEDIATELY PRE- AND POST-INTERVENTIONS

The mean scores of the placebo AAI 1 and placebo AAI 2 interventions immediately pre- and post-interventions are fairly similar across the variables for EMG RMS and muscle force (Table 4.1). While differences are present between the placebo AAI 1 and placebo AAI 2 interventions they are generally much smaller than the differences between them and the SMT intervention immediately pre- and post-interventions, although the standard deviation of the variables in relevance to the differences between the interventions are also fairly large.

The objective data analysis reveals a noteworthy scientific finding. The placebo AAI 1 and placebo AAI 2 interventions show a decrease in the EMG RMS along with the decrease in muscle force, whereas the SMT intervention reveals a medical anomalous increase in the muscle force and decrease in EMG RMS immediately post-interventions (Table 4.1). Figure 4.1 shows no notable change in the biceps brachii muscle force immediately post-placebo AAI interventions and a transient large increase in the biceps brachii muscle force during the SMT intervention. Figure 4.2 shows a transient small decrease in the biceps EMG RMS immediately post-placebo AAI interventions and a transient larger decrease in the biceps EMG RMS immediately post-SMT intervention.

Table 4.1: The electromyography RMS and muscle force of the biceps brachii muscle immediately pre- and post-interventions (M ±SD)

	Baseline (500 ms pre-intervention)	Post-intervention (first 500 ms)	Percentage difference (%)	
RMS (µV)				
Placebo AAI 1	41,98 ± 20,83	41,21 ± 20,27	1.86	
Placebo AAI 2	37,41 ± 20,19	37,4 ± 22,36	0.05	
SMT	49,53 ± 23,48	45,25 ± 23,93	9.03	p = 0.39
Force (kg)				
Placebo AAI 1	$2{,}397^{-1} \pm 2{,}238^{-2}$	$2{,}377^{-1} \pm 2{,}304^{-2}$	0.85	
Placebo AAI 2	$2{,}369^{-1} \pm 2{,}153^{-2}$	$2{,}323^{-1} \pm 2{,}162^{-2}$	1.97	
SMT	$2{,}327^{-1} \pm 3{,}69^{-2}$	$2{,}440^{-1} \pm 5{,}419^{-2}$	4.76	p = 0.155

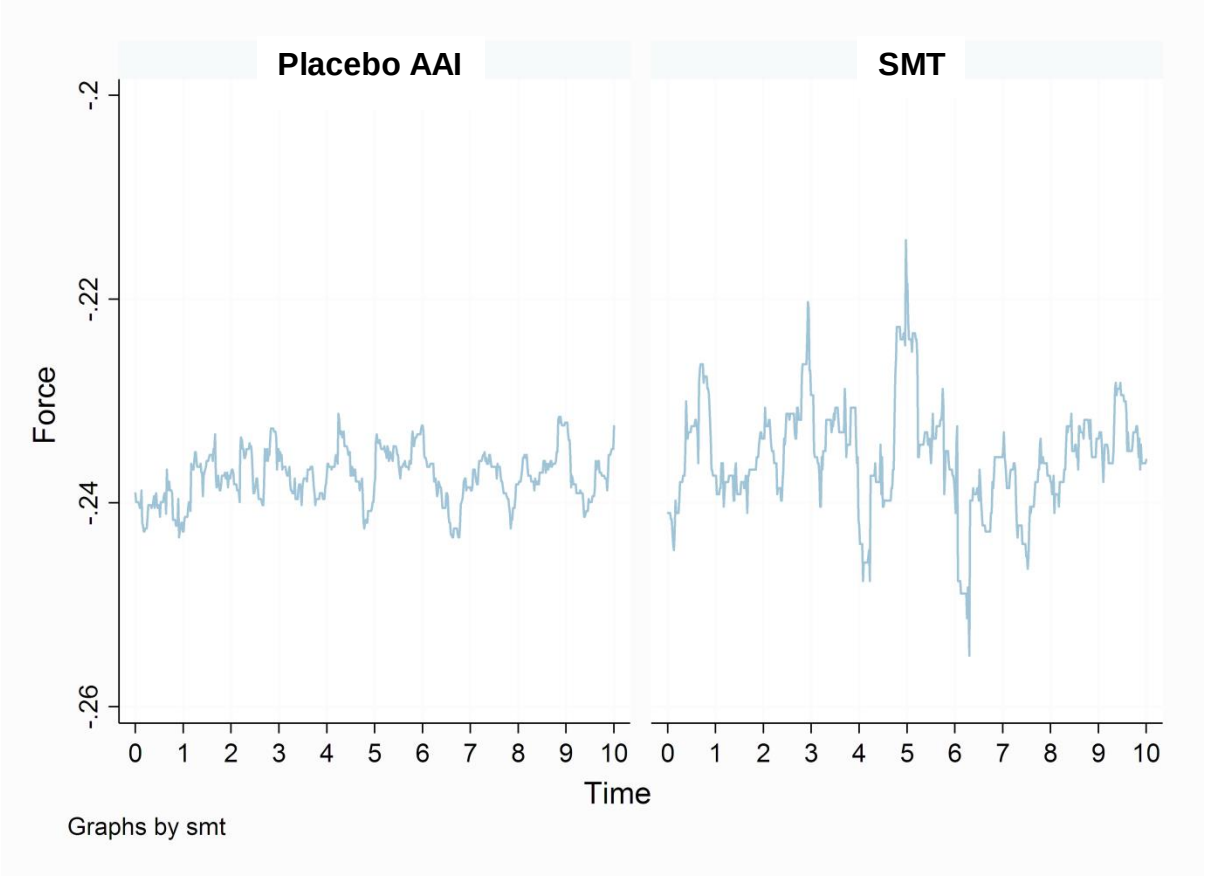


Figure 4.1: The mean biceps brachii muscle force. The interventions were applied at a standardized 4th second during the modified stretching of the biceps brachii muscle (Placebo AAI = AAI 1 + AAI 2 interventions, SMT = SMT intervention)

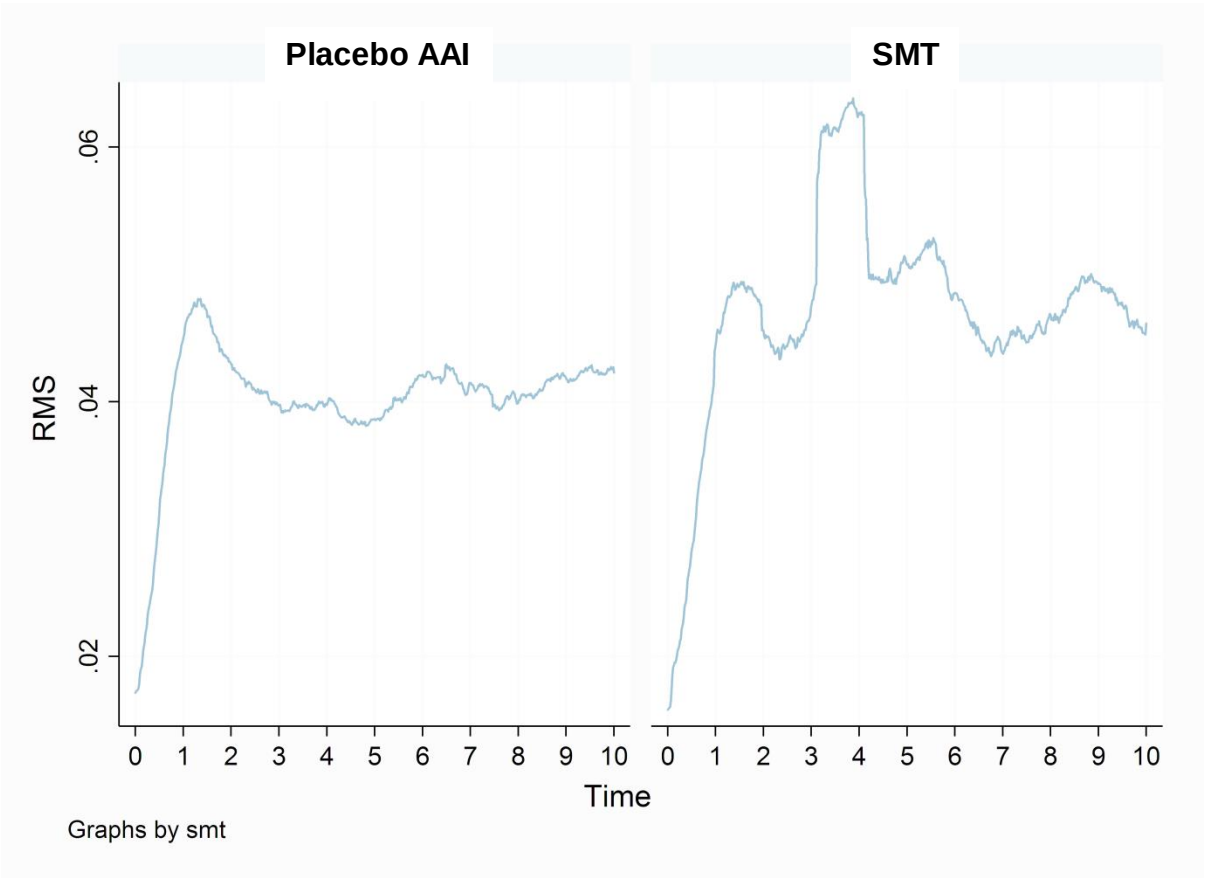


Figure 4.2: The mean biceps electromyography RMS. The interventions were applied at a standardized 4th second during the modified stretching of the biceps brachii muscle (Placebo AAI = AAI 1 + AAI 2 interventions, SMT = SMT intervention)

4.3.2 STATISTICAL DIFFERENCE BETWEEN THE PLACEBO AAI 1 AND PLACEBO AAI 2 INTERVENTIONS

In an analysis of the similarity of EMG RMS between the placebo AAI 1 and placebo AAI 2 interventions, no significant difference was found at both a conventional alpha level, $t(38) = 0.5344$, $p > 0.05$ ($p = 0.5962$), and an optimal alpha level, $t(38) = 0.5344$, $p > 0.232$. See Table 4.2.

Table 4.2: Two-sample t test with equal variances on change in electromyography RMS (placebo AAI 1 vs placebo AAI 2)

Intervention	Observation	Mean (µV)	Standard error	Standard deviation	[95% Confidence interval]
Placebo AAI 1	20	$7{,}734^{-1}$	1,067	4,771	3,006 - 1,459
Placebo AAI 2	20	$1{,}88^{-2}$	$9{,}253^{-1}$	4,138	1,955 - 1,918
combined	40	$3{,}961^{-1}$	$6{,}996^{-1}$	4,425	1,811 - 1,019
Difference		$7{,}547^{-1}$	1,412		3,614 - 2,104

4.3.3 STATISTICAL DIFFERENCE BETWEEN THE PLACEBO AAI (AAI 1 + AAI 2) AND SMT INTERVENTIONS

In an analysis of the difference in EMG RMS between the placebo AAI interventions (AAI 1 + AAI 2) and the SMT intervention no significant difference was found at a conventional alpha level, $t(57) = 0.881$, $p > 0.05$ ($p = 0.3895$), or an optimal alpha level, $t(57) = 0.881$, $p > 0.203$. See Table 4.3.

Table 4.3: Two-sample t test with unequal variances on change in electromyography RMS (SMT vs placebo AAI)

Intervention	Observation	Mean (µV)	Standard deviation	Standard error	[95% Confidence Interval]
Placebo AAI	40	$3{,}96^{-1}$	$7{,}0^{-1}$	4,42	1,81 - 1,02
SMT	19	4,28	4,35	19	13,4 - 4,87
combined	59	1,65	1,47	11,3	4,6 - 1,31
Difference		3,88	4,41		5,35 - 13,1

In an analysis of the similarity in muscle force between the placebo AAI interventions (AAI 1 + AAI 2) and the SMT intervention no significant difference was found at the conventional alpha level, $t(57) = 1.482$, $p > 0.05$ ($p = 0.1549$), but displayed one at the optimal alpha level, $t(57) = 1.482$, $p < 0.203$. See Table 4.4.

Table 4.4: Two-sample t test with unequal variances on change in muscle force (SMT vs placebo AAI 1)

Intervention	Observation	Mean (kg)	Standard error	Standard deviation	[95% Confidence Interval]
Placebo AAI	40	$3,33^{-3}$	$1,630^{-3}$	$1,03^{-2}$	$3,04^{-5}$ - $6,63^{-3}$
SMT	19	$1,13^{-2}$	$9,768^{-3}$	$4,26^{-2}$	$3,19^{-2}$ - $9,18^{-3}$
combined	59	$1,4^{-3}$	$3,4^{-3}$	$2,6^{-2}$	$8,2^{-3}$ - $5,41^{-3}$
Difference		$1,47^{-2}$	$9,904^{-3}$		$6,06^{-3}$ - $3,54^{-2}$

CHAPTER 5 : DISCUSSION

5.1 INTRODUCTION

This chapter discusses the results obtained and provides proposed neurophysiological explanations thereof with possible vindications. In addition, this chapter reviews limitations to the study.

5.2 DEMOGRAPHIC DATA

There were a total of 20 participants in this study with an age range between 19 and 31, the mean age being 26 years (± 3.3 years). The participants ranged in height from 1.57 m to 1.83 m with a mean height of 1.7 m (±0.1 m) and weighed between 45 kg and 100 kg with a mean weight of 70.45 kg (±13.7 kg). Of the 20 participants 12 were male (60 %) and 8 were females (40 %). A total of 40 placebo AAI interventions (66.7 %) were applied to the 20 participants of which all experienced an audible cavitation (100 %). A total of 20 SMT interventions were applied to the 20 participants of which 17 experienced an audible cavitation (85 %).

Dunning and Rushton (2009: 511) investigated the audible cavitation effect of spinal manipulation of the C5/C6 segment on the resting biceps brachii muscle activity. Their findings revealed a significant difference in the resting EMG amplitude immediately post-spinal manipulation for both the cavitation group ($P = 0.0001$) and non-cavitation group ($p = 0.014$). Dunning and Rushton (2009: 511) concluded that the high-velocity low-amplitude thrust delivered during the spinal manipulation can stimulate mechanoreceptors in the facet joint tissue and cause changes in the homonymous motor neuron pool excitability whether or not the facet joint demonstrated the cavitation phenomena.

5.3 OBJECTIVE DATA

Although the SMT intervention showed a much larger difference than the placebo AAI 1 and placebo AAI 2 interventions for the EMG RMS and muscle force of the biceps brachii muscle immediately pre- and post-interventions; this cannot be quantified in terms of statistical significance due to the large standard deviation of the variables relative to the differences between the interventions and possibly due to the small population group. The interferential statistical analysis revealed no significant difference between the placebo AAI interventions (AAI 1 + AAI 2)

and the SMT intervention for the EMG RMS at a conventional alpha level of $t(57) = 0.881$, $p = 0.3895$ ($p > 0.05$) or at an optimal alpha level of $t(57) = 0.881$ ($p > 0.203$), and for the muscle force at a conventional alpha level of $t(57) = 1.482$, $p = 0.1549$ ($p > 0.05$) but displayed one at an optimal alpha level of $t(57) = 1.482$ ($p < 0.203$) of the biceps brachii muscle immediately post-interventions. In addition, no significant difference was found between the placebo AAI 1 intervention and placebo AAI 2 intervention at both a conventional alpha level of $t(38) = 0.5344$, $p = 0.5962$ ($p > 0.05$) and at an optimal alpha level of $t(38) = 0.5344$ ($p > 0.232$) immediately post-interventions.

The large standard deviation is caused by each participant contracting their biceps brachii muscle at their own intensity by their free will, as the participants in this study did not perform a voluntary maximal contraction of their biceps brachii muscle during the modified stretching. The variables of EMG RMS and muscle force are entirely dependent on the voluntary effort of the participant and could therefore not be standardized by depending on the participants performing a constant low force contraction of the biceps brachii muscle during the modified stretching (Lundy-Ekman 2013: 192; Merletti and Parker 2004: 6, 7). Although the participants were instructed to keep a constant isometric contraction of their biceps brachii muscle against the weight of the 1 kg plate during the modified stretching in order to exclude unwanted extra voluntary effort by the participants, they clearly contracted their biceps brachii muscle at a higher muscle force during the SMT intervention, as can be seen in Table 4.1 and Figure 4.1. The participants were instructed to perform a constant isometric contraction of their biceps brachii muscle against the weight of the 1 kg plate during the modified stretching; an increase in voluntary effort by the participants during the SMT intervention can cause the same electromyographic response post-spinal manipulation. An increase in motor unit recruitment and firing frequency of α motor neurons caused by an increase in the voluntary effort by the individual will cause an increase in the EMG RMS (Lundy-Ekman 2013: 192; Merletti and Parker 2004: 6, 7). Suter and McMorland (2002: 542, 543) found a transient increase in EMG RMS of the biceps brachii muscle immediately post-spinal manipulation of C5/C6 segmental levels.

The objective data analysis revealed a noteworthy scientific finding. The immediate post-SMT intervention revealed an increase in the biceps brachii muscle force by 4.76 % and a decrease in the biceps EMG RMS by 9.03 % with a summation of percentage difference between the muscle force and EMG RMS of 13.79 %, whereas for both the immediate post-placebo AAI 1 intervention and post-placebo AAI 2 intervention the biceps EMG RMS decreased along with the biceps brachii muscle force. The immediate post-placebo AAI 1 intervention showed a decrease in the biceps EMG RMS by 1.86 % and a decrease in the biceps brachii muscle force by 0.85 % with a summation of percentage difference between the muscle force and EMG RMS of 1.01 %. The

immediate post-placebo AAI 2 intervention showed a decrease in the biceps EMG RMS by 0.05 % and a decrease in the biceps brachii muscle force by 1.97 % with a summation of percentage difference between the muscle force and EMG RMS of 1.92 % (Table 4.1). This finding is noteworthy because the motor unit recruitment and firing frequency of α motor neurons are entirely dependent on the level of force and speed of muscle contraction by the voluntary effort of the individual. There is a direct relationship between the muscular force generated and the EMG amplitude RMS in the normal non-pathological state. The greater the number of motor units recruited and their discharge frequency rate, the greater the muscular force will be. Thus, if the muscle force increases the EMG RMS should also increase and if the muscle force decreases the EMG RMS should also decrease in relation to the voluntary effort of the individual (Lundy-Ekman 2013: 192; Merletti and Parker 2004: 6, 7).

5.4 THE INVERSE RELATIONSHIP BETWEEN MUSCLE FORCE AND ELECTROMYOGRAPHY RMS IMMEDIATELY POST-SPINAL MANIPULATION

5.4.1 AN INCREASE IN BICEPS BRACHII MUSCLE FORCE IMMEDIATELY POST-SMT INTERVENTION

A large transient increase in the mean biceps brachii muscle force during the SMT intervention can be seen in Figure 4.1 and immediately post the SMT intervention (Table 4.1); this is possibly due to the mean participant contracting their biceps brachii muscle at a higher muscle force by their free will. A subconscious adjustment in voluntary effort by the participants may be the mechanism responsible due to the emotional component such as fright, excitement or fear experienced (Rice and McNair 2010: 259; Engelhardt *et al.* 2001: 75) during the SMT intervention. A significant difference in biceps brachii muscle force was found between the placebo AAI interventions (AAI 1 + AAI 2) and the SMT intervention at the optimal alpha level, $t(57) = 1.482$, $p < 0.203$ immediately post-interventions. The transient increase in voluntary effort by the participants immediately post the spinal manipulation will cause an increase in the motor unit recruitment and fire frequency of the α motor neurons innervating the biceps brachii muscle via the lateral corticospinal tract (pyramidal motor pathway) and thereby should result in the transient increase in the biceps EMG RMS (Lundy-Ekman 2013: 192; Merletti and Parker 2004: 7) and an increase in the biceps brachii muscle force (Lundy-Ekman 2013: 187; FitzGerald *et al.* 2012: 122, 123). On the contrary, the biceps EMG RMS decreased during the transient increase in voluntary effort by the participants immediately post the SMT intervention as can be seen in Table 4.1, Figure 4.1 and Figure 4.2.

5.4.2 A DECREASE IN ELECTROMYOGRAPHY RMS IMMEDIATELY POST-SMT INTERVENTION

5.4.2.1 EXCLUSIONS

Prior to suggesting that spinal manipulation caused or contributed to the decrease in EMG RMS observed in the inverse relationship between muscle force and EMG RMS immediately post the SMT intervention; it is vital to explore and consider all other causes or contributors to the decrease in EMG RMS observed immediately post the SMT intervention. The following section will provide an in depth discussion of the possible causes or contributors which must be excluded prior to making the deduction on a neurophysiological basis from the results obtained from this study that spinal manipulation caused or contributed to the decrease in EMG RMS observed in the inverse relationship between muscle force and EMG RMS immediately post the SMT intervention.

5.4.2.1.1 GOLGI TENDON ORGAN Ib INHIBITION

Historically the function of the GTO was thought to be a protective reflex in which a strong and potentially damaging muscle force from excessive loading will reflexively inhibit the muscle via autogenic inhibition to cause lengthening of the muscle instead of trying to maintain the muscle force and risking damage (Khurana 2014: 829; Lundy-Ekman 2013: 194; Carpenter and Reddi 2012: 100). The effect of GTO input to the homonymous motor neuron pool is not on its own powerful enough to inhibit voluntary muscle contraction (the LCST), because maximal GTO Ib afferent activity occurs before 50% of maximal voluntary muscle contraction. GTO activation cannot therefore on its own elicit sufficient Ib inhibition of the homonymous α motor neurons to cause reflexive relaxation of overloaded muscles. Nor can GTO inhibition explain muscle relaxation following maximal voluntary muscle contraction, because the GTO Ib afferents firing rate decreases when the muscle stops contracting (Lundy-Ekman 2013: 194; Fitzgerald *et al.* 2012: 188, 189; Mense and Gerwin 2010: 228, 229). Although the GTOs provide some protection by way of the autogenic inhibition, most of the protection is considered to be provided by the lateral cortical spinal tract which causes presynaptic inhibition of the homonymous actively stretched prime mover muscle spindle afferents close to the contact points with their α motor neurons in the anterior horn of the spinal cord by way of the interpolation of inhibitory internuncials in the intermediate grey matter of the spinal cord. The extent of the suppression of the active stretch reflex by the LCST is dependent on the particular motor program being executed, during postural muscle activity or voluntary effort (Fitzgerald *et al.* 2012: 188, 189). The role of GTO resides in the regulation of small alterations in muscle tension during normal muscle activity by inducing autogenic inhibition on the homonymous motor neuron pool in concert with other proprioceptive

signals and upper motor neuron control (Khurana 2014: 829; Lundy-Ekman 2013: 194; Carpenter and Reddi 2012: 100).

The transient large increase in biceps brachii muscle force possible caused by the increase in voluntary effort by the participants immediately post the SMT intervention caused the activation of α motor neurons of higher-threshold motor units of which at least one of their extrafusal fibers inserted into the biceps brachii muscle GTO and intertwined with the loosely packed innervated collagen fibrils inside the GTO and resulted in peak linear or more non-linear summation of biceps brachii muscle GTO Ib afferent activity (Mileusnic and Loeb 2006: 1790). The peak linear / non-linear summation of biceps brachii muscle GTO Ib afferent activity will decrease the excitability of the homonymous α motor neurons innervating the biceps brachii muscle and contribute to the decrease in the biceps brachii muscle EMG RMS immediately post the SMT intervention. It is unlikely though, that the peak linear / non-linear biceps brachii muscle GTO Ib afferent activity immediately post the SMT intervention on its own can cause an inverse relationship between the muscle force and EMG RMS of the biceps brachii muscle immediately post the SMT intervention, except for in the setting of a pathological state such as clasp-knife reflex. Clasp-knife reflex refers to an exaggerated form of the GTO Ib inhibitory di-synaptic spinal reflex in upper motor neuron disease or lesion (Khurana 2014: 829; Carpenter and Reddi 2012: 100).

Several studies have vindicated the observation that the GTO inhibitory input to the homonymous motor neuron pool during voluntary maximal muscle contraction is not solely powerful enough to cause the inverse relationship between the muscle force and EMG RMS observed immediately post-spinal manipulation. These EMG studies did not apply spinal manipulation during the recorded muscle activity, but did so between the pre- and post-recorded muscle activity of voluntary maximal muscle contraction. Suter *et al*. (1999: 149) investigated the effect of spinal manipulation of the sacroiliac joint on the activity and strength of the knee extensor muscles in participants with sacroiliac joint dysfunction. Immediately pre and post the spinal manipulation; the strength of the knee extensor muscles was measured during voluntary maximal isometric contractions by using a Cybex dynamometer, as well as the activity of the knee extensor muscles by using a surface electromyograph. Suter *et al*. (1999: 149) found a transient increase in both the EMG amplitude and the muscle force of the knee extensor muscles immediately post the spinal manipulation. Du Plessis (2014) investigated the effect of spinal manipulation of the C5/C6 segment on the activity and strength of the biceps brachii muscle in participants with chronic neck pain. Immediately pre and post the spinal manipulation strength was measured during voluntary maximal biceps brachii muscle contraction against a Baseline Hydraulic Push-Pull dynamometer, as well as the activity of the biceps brachii muscles by using a surface EMG. Du Plessis (2014) found a significant ($p < 0.05$) transient increase in the mean biceps brachii muscle force as well as

a significant ($p < 0.05$) transient increase in the mean biceps EMG amplitude immediately post the spinal manipulation.

5.4.2.1.2 NOCICEPTORS

Although participants in the current study were asymptomatic, nociceptors in the cutaneous tissue (skin) and facet joint capsular tissue and synovium may have also been stimulated due to the propulsive thrust applied during the SMT intervention (Millan *et al.* 2012: 24; Pickar 2002: 359) in addition to activating the mechanoreceptors in the facet joint tissue (Olsen 2015: 85; Dunning and Rushton 2009: 509; Suter and McMorland 2002: 544; Pickar 2002: 363). It is unlikely though that an increase in nociceptors Aδ- and / or C-fiber afferent discharge caused the inverse relationship between the muscle force and EMG RMS immediately post the SMT intervention. Recent medical literature has shown that activated nociceptors afferent from joints and cutaneous tissue are more likely to cause an excitatory influence on the homonymous motor neuron pool to result or contribute in the induction of a spasm of the homonymous muscle group (Steward 2012: 214; Mense and Gerwin 2010: 222, 229; Mense and Skippar 1991: 201). An excitatory influence on the homonymous motor neuron pool will contribute to an increase in the EMG RMS of the homonymous muscle group during voluntary muscle contraction by inducing disinhibition of the homonymous α motor neurons if the induced depolarization on the homonymous α motor neurons produce EPSPs that reach firing threshold at the axon hillock of the depolarized α motor neurons (Fitzgerald *et al.* 2012: 87; Mense and Gerwin 2010: 227, 229; Merletti and Parker 2004: 6, 7). Facilitation of an increased state of firing of the homonymous α motor neurons will not cause an increase in muscle strength (Mense and Gerwin 2010: 229), but may cause a decrease in muscle strength during voluntary muscle contraction if the muscle spasm is severe enough (Page *et al*. 2010: 50). The facilitation of increased excitability of the homonymous motor neuron pool can cause alterations in the motor unit recruitment, lower the homonymous α motor neurons activation threshold, or lower their irritability threshold (Page *et al.* 2010: 50).

In addition, studies have shown that the input of Aβ-fiber afferents of mechanoreceptors is more powerful than the input of Aδ- and / or C-fiber afferents of nociceptors into the central nervous system. Branches of large Aβ-fibers of mechanoreceptors afferent of joint tissue which has shared innervation with the homonymous wide dynamic range neurons in the dorsal horn of the spinal cord, enter the posterior column-medial lemniscal pathway in the white matter of the spinal cord. Stimulation of the posterior column by way of the Aβ-fibers of mechanoreceptors in joint tissues sends antidromic conducted action potentials via collateral braches into the dorsal horn of the spinal cord which in turn stimulate the enkephalinergic interneurons that inhibit the transmission of nociceptive signals via the anterolateral system. This is the physiological basis for the gate theory

of pain (Steward 2012: 214; Haines 2012: 259). Further, none of the participants of this study reported experiencing pain during the SMT intervention.

5.4.2.1.3 MECHANORECEPTORS IN CUTANEOUS TISSUE

Mechanoreceptors in the cutaneous tissue could have also been stimulated during the placebo AAI interventions and the SMT intervention (Millan *et al.* 2012: 24; Pickar 2002: 359) in addition to activating the mechanoreceptors in the facet joint tissue (Olsen 2015: 85; Dunning and Rushton 2009: 509; Suter and McMorland 2002: 544; Pickar 2002: 363). Activated mechanoreceptors in cutaneous tissue can exert an excitatory or inhibitory influence on the homonymous motor neuron pool and could have contributed to the decrease in EMG RMS immediately post the SMT intervention (Steward 2012: 214; Mense and Gerwin 2010: 222, 229; Merletti and Parker 2004: 6, 7). However, it is unlikely that the stimulation of mechanoreceptors in cutaneous tissue on its own could have caused the inverse relationship between the muscle force and EMG RMS immediately post the SMT intervention, because the placebo interventions showed a much smaller decrease in the EMG RMS compared to the SMT intervention immediately post-interventions. The SMT intervention revealed a decrease in the biceps EMG RMS by 9.03 %, whereas the AAI 1 intervention showed a decrease in the biceps EMG RMS by 1.86 % and the placebo AAI 2 intervention showed a decrease in the biceps EMG RMS by 0.05 % immediately post-interventions.

5.4.2.1.4 INVOLUNTARY SPINAL REFLEX ARCS

It unlikely that activity of an arthrogenic reflex caused the inverse relationship between the muscle force and EMG RMS of the biceps brachii muscle immediately post the SMT intervention. When a joint capsule is stretched during joint movement; the joint mechanoreceptors will cause activation of the muscles which will reduce the joint capsular stretch and cause inhibition of the muscles which will increase the joint capsular stretch (Petty 2011: 16; Middleditch and Oliver 2005: 246). During the modified stretching of the biceps brachii muscle in the current study; the mechanoreceptors in the elbow joint tissue would have exerted powerful tonic excitatory influences on the α motor neurons innervating the elbow flexors and inhibited the elbow extensor muscles to reduce the capsular stretch of the extended elbow joint. The arthrokinetic reflex would therefore result in an increase in the biceps EMG RMS and not a decrease (Fitzgerald *et al.* 2012: 87; Mense and Gerwin 2010: 227, 229; Middleditch and Oliver 2005: 246; Merletti and Parker 2004: 6).

5.4.2.1.5 DESCENDING MOTOR PATHWAYS

5.4.2.1.5.1 PYRAMIDAL MOTOR PATHWAYS

Descending motor pathways projecting from the upper motor neurons in the motor cortex of the brain can be divided into pyramidal (corticospinal and corticobulbar) and extrapyramidal (Rea 2015: 196; Lundy-Ekman 2013: 201). The corticobulbar tract innervates the lower motor neurons of the cranial nerve (muscles of the face, head and neck) (Lundy-Ekman 2013: 201; Mendiza and Foundaz 2007: 11). The anterior and ipsilateral corticospinal tracts synapse on lower motor neurons innervating axial musculature in the spinal cord to function in posture and locomotion (Lundy-Ekman 2013: 201; Fitzgerald *et al.* 2012: 190). Activity of the corticobulbar tract, anterior corticospinal tract and ipsilateral corticospinal tract will, therefore, have no effect on the lower motor neurons innervating the biceps brachii muscle.

5.4.2.1.5.2 EXTRAPYRAMIDAL MOTOR PATHWAYS

There are several extrapyramidal motor pathways which arise from the reticular formation in the brainstem that modulate lower motor neurons and interneurons in the spinal cord by exerting an excitatory and / inhibitory influence on them, and can thereby function in posture, locomotion and spinal reflex arc activity. The extrapyramidal tracts function as part of the postural system to prepare the cortical projections to the spinal cord via the pyramidal motor pathways for fractionation. Fractionation is the ability of the LCST to selectively activate small groups of lower motor neurons to perform specific voluntary movements such as elbow flexion. All voluntary movements are initiated by the corticospinal tracts (Rea 2015: 169; Lundy-Ekman 2013: 201).

The lateral and medial reticulospinal tracts function as collateral modifications for the corticospinal tracts to provide coordination of muscle activity of the axial (trunk) and proximal muscles of all four limbs during locomotion, as well as function in anticipatory postural adjustments (Lundy-Ekman 2013: 199, 200; Fitzgerald *et al.* 2012: 192). The medial (pontine) reticulospinal tract facilitate cortical (voluntary) induce movements and increase muscle tone by exerting an excitatory influence on the lower motor neurons innervating the extensor muscle groups. The lateral (medullar) reticulospinal tract inhibit cortical induced movements and reduce muscle tone by exerting an inhibitory influence on the extensor muscle groups (Rea 2015: 171; Lundy-Ekman 2013: 199, 200; Fitzgerald *et al.* 2012: 192). The biceps brachii muscle is an elbow flexor (Diogo *et al.* 2013: 42; Muscolino 2008: 265). It is therefore unlikely that activity of the reticulospinal tracts caused the inverse relationship between muscle force and EMG RMS of the biceps brachii muscle immediately post the SMT intervention.

The tectospinal tract function in controlling postural changes of the head / trunk in response to visual and auditory stimuli by synapsing on lower motor neurons innervating the neck musculature (Rea 2015: 171; Lundy-Ekman 2013: 199, 200; Fitzgerald *et al.* 2012: 192). Activity of the tectospinal tract will therefore have no effect on the lower motor neurons innervating the biceps brachii muscle during the modified stretching.

Descending autonomic sympathetic fibers (hypothalamospinal tract) and parasympathetic fibers (hypothalamomedullary tract) also descend through the reticular information and supply dual motor and sensory innervation to viscera, glands, blood vessels as well as smooth muscles and cardiac muscles under automatic (unconscious) control (Freberg 2015: 33; Steward 2012: 214). The preganglionic cell bodies of the sympathetic nervous system are found in the spinal cord at all thoracic segmental levels as well as the first two/ three lumbar segments. The preganglionic cell bodies of the parasympathetic nervous system are found in the cranial nerves Ⅲ, Ⅶ, Ⅸ and Ⅹ and in the spinal cord at sacral segments of S2-S4 (Lundy-Ekman 2013: 168; Jacobson and Marcus 2011: 67). Visceral afferents can synapse on somatic efferents in the spinal cord which innervate skeletal muscles to produce muscle guarding in the setting of a pathological state; for example, appendicitis (inflammation of the appendix) will cause contraction of the abdominal musculature (Lundy-Ekman 2013: 169). Activity of descending autonomic fibers will therefore have no effect on the lower motor neurons innervating the biceps brachii muscle during the modified stretching.

The aminergic pathways namely the raphespinal and cureleospinal tracts exert facilitatory effects on lower motor neurons in the spinal cord during excessive limbic activity (emotional motor system). Their motor effects are not related to specific movement but produce a more generalized motor effect and can contribute to poorer motor performance and reduce the magnitude and rate of postural adjustments during high levels of stress or anxiety. The raphespinal and cureleospinal tracts also function to modulate sensory transmission between the first and second set of sensory neurons in the dorsal horn of the spinal cord regarding nociception (pain) (Lundy-Ekman 2013: 204; Fitzgerald *et al.* 2012: 194). High activity of these reticulospinal tracts may provide clarity for stress-related induced muscle spasms (Mense and Gerwin 2010: 230). It is therefore unlikely that activity of the raphespinal and cureleospinal tracts caused the inverse relationship between muscle force and EMG RMS of the biceps brachii muscle immediately post the SMT intervention.

The rubrospinal tract descends along the LCST to exert a facilitatory effect on lower motor neurons innervating flexor muscle groups at predominant cervical segmental levels. Activation of the corticospinal-rubrospinal system thereby functions in the fine control of movements in the upper extremity (Rea 2015: 171, 172; Siegel *et al*. 2010: 336). In addition, the overall size of the rubrospinal tract in humans is diminished. It is postulated that activation of the red nucleus in the

midbrain of the brainstem (origin of the rubrospinal tract) has a more potent influence on the cerebellum function (motor control and coordination of movements) via the rubro-olivary fibers, because of the increase in size of this descending projection from the red nucleus to the cerebellum via the inferior olivary nucleus in the medulla oblongata of the brainstem (Rea 2015: 171; Siegel *et al*. 2010: 336). Also, the existence of descending axons projecting from the inferior olivary nucleus to the spinal cord via the olivospinal tract is questioned in the latest medical literature (Rea 2015: 171). It is therefore unlikely that activity of the rubrospinal and olivospinal tracts caused the inverse relationship between muscle force and EMG RMS of the biceps brachii muscle immediately post the SMT intervention.

The vestibulospinal tracts function in the modulation of antigravity muscles to keep the eyes in the horizontal plane and the center of gravity between the feet. For example, when the head is tilted to more than one side the tone of the appropriate antigravity muscles will be automatically increased via the vestibulospinal pathway to center the head (Fitzgerald *et al.* 2012: 194). The medial vestibulospinal tract exert facilitatory effects on the lower motor neurons innervating the trapezius and sternocleidomastoid muscles (associated with cranial nerve XI) in response from sensory input received from the vestibular apparatus in the inner ear (Lundy-Ekman 2013: 200; Siegel *et al*. 2010: 336). The medial vestibulospinal tract will therefore have no effect on the lower motor neurons innervating the biceps brachii muscle during the modified stretching. The lateral vestibulospinal tract predominantly exerts powerful facilitatory influence on lower motor neurons innervating extensor muscle groups of the lower extremity and exerts inhibitory influence on lower motor neurons innervating flexor muscle groups of the lower extremity at lumbar segmental levels in response to sensory input from the vestibular apparatus in the inner ear as well as from the cerebellum (Siegel *et al*. 2010: 336). The lateral vestibulospinal tract function in response to the slightest destabilization in the center of gravity and is therefore continuously active during the upright position to maintain the center of gravity over the base of support (Lundy-Ekman 2013: 200). The lateral vestibulospinal tract will therefore have no effect on the lower motor neurons innervating the biceps brachii muscle during the modified stretching.

5.4.2.2 PROPOSED UNDERLYING NEUROPHYSIOLOGICAL MECHANISM

The Ib internuncials found in Rexed lamina VI and VII in the intermediate gray matter of the spinal cord were initially defined by their response to input from the GTOs. The more recent medical literature has demonstrated that Ib internuncials also receive sensory input from mechanoreceptors (type I-III nerve endings) in joint tissue. The variety of sensory input can reach the Ib internuncials through several independent spinal reflex arcs. Studies have shown that there is little specialization of Ib internuncial by type of their sensory input, as large Aβ-fiber afferents

from joint tissue can cause similar Ib inhibition on the homonymous α motor neuron (Greger and Windhorst 2013: 1001; Brushart 2011: 73). Facilitation of the facet joint Ib inhibitory di-synaptic reflex arc can cause Ib inhibition on the homonymous motor neuron pool (Greger and Windhorst 2013: 1001; Rice and Mcnair 2010: 256; Dunning and Rushton 2009: 512; Suter and McMorland 2002: 541). Iles *et al*. (1990: 489) infused uninjured human knee joints with saline and used the spatial facilitation technique to show that joint swelling can enhance Ib inhibition of the quadriceps H-reflex at rest and during voluntary muscle contraction. Iles *et al*. (1990: 489) concluded that raised joint mechanoreceptor Aβ-fiber afferents can therefore cause AMI by facilitating Ib inhibition.

The high-velocity low-amplitude thrust applied to the ipsilateral C5/C6 spinal segmental levels (SMT intervention) during the modified stretching of the biceps brachii muscle caused stimulation of the low threshold type I Ruffini end-organs and type II Pacinian corpuscle nerve endings as well as the high threshold type III Golgi nerve endings in the capsular tissue and synovium of the facet joints at the spinal segmental levels of C5/C6 (Olsen 2015: 85; Dunning and Rushton 2009: 509; Suter and McMorland 2002: 544; Pickar 2002: 363). The rapid high shear force produced on the facet joint capsular tissue by the spinal manipulation (Millan *et al*. 2012: 24; Nathan and Keller 1994: 431; Herzog *et al*. 1993: 448) will cause the mechanical opening of the transduction ion channels in the surface membrane of the sensory terminals of the mechanoreceptors Aβ-fiber afferent in the facet joint capsular tissue and synovium (Haines 2013: 329; Mileusnic and Loeb 2006: 1789, 1790; Merletti and Parker 2004: 13). Depolarization of the facet joint mechanoreceptors Aβ-fiber afferent endings will follow by allowing Na^+ ions and K^+ ions to diffuse across the surface membrane of the mechanoreceptors Aβ-fiber afferent endings (Lundy-Ekman 2013: 27; FitzGerald *et al*. 2012: 83, 84, 125, 126). The summation of positive electrode waves induced by the Na^+ ion influx will raise the receptor membrane potential of the facet joint mechanoreceptors Aβ-fiber afferent endings above threshold at their trigger zones, and cause the generation of action potentials in a sequential manner and thus cause firing of them (FitzGerald *et al*. 2012: 125, 126; Merletti and Parker 2004: 12). The stimulated facet joint mechanoreceptors Aβ-fiber afferents by spinal manipulation will discharge their excitatory nerve impulses in the form of generated action potentials to their first-order set of sensory neurons (Jacobson and Marcus 2011: 64) via the medial branch of the dorsal rami division of spinal nerves (Waldman 2009: 161; Manchikanti *et a*l. 2002: 243) before traveling thereafter along the spinal nerves in the distal intervertebral foramina between the pedicles of successive vertebrae (Fitzgerald *et al*. 2012: 38; Steward 2002: 25) at spinal segmental levels of C5/C6 (Biller 2012: 246; Muscolino 2008: 265). The Aβ-fibers of the facet joint mechanoreceptors afferent will then convoy their nerve impulses along the centripetal processes from their dorsal root ganglia to the entry zone of the spinal cord

via the dorsal nerve roots (FitzGerald *et al*. 2012: 177, 178), to enter the dorsal horn of the spinal cord at the posterolateral sulcus via the medial bundle fibers of the dorsal nerve roots (Fitzgerald *et al.* 2012: 38; Steward 2002: 25, 26). The Aβ-fibers of the facet joint mechanoreceptor afferents have projections via their collateral branches into laminae VI and VII in the intermediate gray matter of the spinal cord to form excitatory axodendritic and / or axosomatic glutamergic synapses on Ib internuncials (Greger and Windhorst 2013: 1001; Brushart 2011: 73). The generated impulses from the facet joint mechanoreceptor Aβ-fiber afferents by spinal manipulation will reach their synaptic terminals in the intermediate gray matter of the spinal cord to cause depolarization of their synaptic terminals. The released glutamate neurotransmitters from the depolarized synaptic terminals of the stimulated facet joint mechanoreceptor Aβ-fiber afferents will diffuse across the synaptic cleft to bind to their specific ionotropic receptors in the membrane of the Ib internuncials at their receiving zones, namely the AMPA and kainite receptors (Lundy-Ekman 2013: 49; Rastogi 2006: 448). The binding of the released glutamate neurotransmitters to the AMPA-K receptors in the receiving zones of the Ib interneurons will cause depolarization of the Ib interneurons. The summation will produce EPSPs that reach firing threshold at the trigger zone of the depolarized Ib internuncials, action potentials will be generated and cause firing of them (Lundy-Ekman 2013: 49; FitzGerald *et al.* 2012: 86, 87; Siegel *et al*. 2010: 100). The produced graded potentials in the Ib internuncials induced by the stimulated facet joint mechanoreceptor Aβ-fiber afferents, will be directly proportional to the quantity of stimulated mechanoreceptors in the facet joint capsular tissue and synovium by the spinal manipulation (Greger and Windhorst 2013: 1001; Brushart 2011: 73). The recruited Ib internuncials by the stimulatory effect of spinal manipulation on the facet joint mechanoreceptors, will cause Ib inhibition on the homonymous α motor neurons and possibly the y motor neurons innervating the biceps brachii and brachialis muscles, by forming in turn inhibitory axodendritic and / or axosomatic glycinergic synapses on the lower motor neurons in the anterior horn of the spinal cord (Greger and Windhorst 2013: 1001; Brushart 2011: 73). The released glycine neurotransmitters from the depolarized synaptic terminals of the recruited Ib internuncials will diffuse across the synaptic cleft to bind to their specific ionotropic glycine receptors in the membrane of the homonymous α motor neurons and possible the y motor neurons at their receiving zones (Lundy-Ekman 2013: 60, 62; Fitzgerald *et al.* 2012: 99, 100). The binding of the released glycine neurotransmitters to the GlyR in the receiving zones of the lower motor neurons will cause hyperpolarization of them. The summation will produce IPSPs that will drive the membrane potential at the axon hillock of the homonymous α motor neuron away from firing threshold to prevent the generation of action potentials, and thus inhibit firing of them (FitzGerald *et al.* 2012: 86, 87; Siegel *et al*. 2010: 100; Rastogi 2006: 488). The produced IPSPs in the homonymous α motor neurons induced by the stimulated facet joint mechanoreceptors Aβ-fiber afferent fibers, will also be directly proportional to the quantity of stimulated mechanoreceptors in

the facet joint capsular tissue and synovium by the spinal manipulation (Greger and Windhorst 2013: 1001; Brushart 2011: 73).

Facilitation of the facet joint Ib inhibitory di-synaptic reflex arc caused by the spinal manipulation of C5/C6 segmental levels will result in a decrease in the excitability of the homonymous α motor neurons innervating the biceps brachii muscle and thereby contribute to the decrease in EMG RMS of the biceps brachii muscle as seen in the inverse relationship between the muscle force and EMG RMS immediately post the SMT intervention (Mense and Gerwin 2010: 208). An inverse relationship between muscle force and EMG RMS during voluntary muscle contraction immediately post-spinal manipulation has not been reported in the chiropractic literature. This is due to EMG studies having only investigated the effects of spinal manipulation on muscle activity pre- and post-maximum voluntary muscle contraction and not during the muscle contraction. Also, these studies did not investigate the effect of spinal manipulation on muscle activity during facilitated GTO Ib inhibitory di-synaptic reflex as part of the convergent input on the homonymous motor neuron pool excitability (Olsen 2015: 86; Pickar 2002: 364).

The inverse relationship revealed between the muscle force and EMG RMS of the biceps brachii muscle immediately post-spinal manipulation of the C5/C6 segment, is due to the spatial summation of combined peak linear / non-linear GTO Ib afferent activity caused by the increase in voluntary effort by the participants during the modified stretching of the biceps brachii muscle and the transient increase in facet joint mechanoreceptor Aβ-fiber afferent activity caused by the spinal manipulation, which produced the transient summation of inhibition on the homonymous α motor neurons that are larger than the summation of excitation produced by the lateral corticospinal tract. As part of the convergent input on the α motor neurons innervating the biceps brachii muscle; the spinal manipulation caused transient facilitation of the facet joint Ib inhibitory di-synaptic reflex arc and thereby facilitated (reinforced) the Ib inhibition on the biceps brachii α motor neurons produced by the facilitated GTO Ib inhibitory di-synaptic spinal reflex arc during the modified stretching of the biceps brachii muscle, which resulted in sufficient IPSPs in the biceps brachii α motor neurons to drive the membrane potential at the axon hillock of each depolarized biceps brachii α motor neuron away from firing threshold and thereby prevented the lateral corticospinal tract from transiently generating sequential action potentials in the biceps brachii α motor neurons. A transient decrease in the EMG RMS during the increase in voluntary effort by the participants (increase in muscle force) of the biceps brachii muscle immediately post the SMT intervention will result.

When the stimulus is removed that caused the generation of EPSPs or IPSPs in the target neuron, the disturbance in the membrane potential of the target neuron will fade away and return to

baseline (Starr and McMilan 2015: 243; Rastogi 2006: 484). The high-velocity low-amplitude thrust delivered at the end range of motion of the facet joints during spinal manipulation is a rapid short-lived propulsive thrust that can produce a force between about 220 N to 550 N with a duration of between about 200 ms to 420 ms (Herzog *et al*. 1993: 448). The mechanoreceptors in the facet joint tissue will therefore be transiently activated by the spinal manipulation and the induced Ib inhibition on the homonymous motor neuron pool including the α motor neurons innervating the biceps brachii muscle via the facilitated facet joint Ib inhibitory di-synaptic spinal reflex arc will also occur transiently.

The transient Ib inhibition on the homonymous motor neuron pool caused by spinal manipulation is vindicated by several studies that observed an electromyographic response latency immediately post-spinal manipulation, as well as the immediately transient decrease in excitability of the homonymous motor neuron pool post-spinal manipulation. Herzog *et al.* (1999: 146) investigated the effect of spinal manipulation applied to the cervical spine, thoracic spine, lumbar spine and sacroiliac regions on the muscle activity of their associated paraspinal musculature in asymptomatic participants. Herzog *et al.* (1999: 146) reported an electromyographic response latency occurring within 50 ms to 200 ms immediately after the application of a high-velocity low-amplitude thrust. Colloca and Keller (2001: 489) confirmed these latter findings in symptomatic patients with low back pain. Colloca and Keller (2001: 489) reported an electromyographic response latency occurring within 2 ms to 3 ms immediately after the high velocity-low amplitude thrust applied via an Activator Adjusting Instrument. Dishman *et al.* (2002: 318) investigated the effects of lumbar spine manipulation on the excitability of the lumbar motor neuron pool in participants with low back pain. Dishman *et al.* (2002: 318) studied the effect of spinal manipulation on the excitability of the homonymous motor neuron pool by measuring and recording the amplitude of the tibial nerve H-reflex recorded from the gastrocnemius muscle, pre- and post- spinal manipulations. Dishman *et al.* (2002: 318) found in the majority of the participants a significant transient decrease in the homonymous motor neuron pool excitability immediately after the spinal manipulation.

Spinal manipulation may induce neuroplasticity in the form of habituation and result in a decrease in abnormal arthrokinetic reflex arcs. By gradually or repeatedly inducing an abnormal stimuli or abnormal response over time, habituation can be achieved and result in a reduction of the abnormal stimuli or abnormal response (Lundy-Ekman 2013: 67, 69; Sweetow and Sabes 2010: 461). By inducing Ib inhibition repetitively over time on a motor neuron pool which is already subjected to Ib inhibition caused by arthogenic muscle inhibition, habituation can be achieved to the facilitated joint Ib inhibitory di-synaptic reflex arc and thereby cause a decrease in muscle weakness and an increase in strength of the homonymous musculature. Spinal manipulation can

cause transient facilitation of the facet joint Ib inhibitory di-synaptic reflex arc and thereby achieve habituation over time to a facilitated Ib inhibitory di-synaptic spinal reflex arc caused by joint dysfunction which has shared innervation with the homonymous motor neuron pool. Du Plessis (2014) investigated the effect of spinal manipulation of the C5/C6 segment on the activity and strength of the biceps brachii muscle in participants with chronic neck pain over a three week period. Three measurements were recorded spanning the three weeks. Immediately before and after the C5/C6 spinal manipulation; the strength of the biceps brachii muscle was measured during maximal biceps contraction against a Baseline Hydraulic Push-Pull dynamometer, as well as the activity of the biceps brachii muscles by using a surface EMG. The mean dynamometry increased from the first reading recorded of 20.67 kg to the second reading recorded of 21.49 kg, and increased from the second to the third reading recorded of 22.99 kg, with a significant increase in the biceps brachii muscle strength over the three weeks ($p = 0.005$). The mean EMG amplitude increased from the first reading recorded as 150.76 mV to the second reading recorded as 151.66 mV, and increased from the second to the third reading recorded as 152.02 mV, with a significant increase in the biceps brachii muscle activity over the three weeks ($p = 0.000$). Du Plessis (2014) concluded that the underlying neurophysiological mechanism for the significant ($p < 0.05$) increase in the muscle force and activity post-spinal manipulation is unclear. The incremental increase in muscle force and muscle activity over the three weeks duration is possibly due to the spinal manipulation achieving habituation to altered arthrokinetic reflex arcs present.

5.4.2.3 DISINHIBITION

The notion of transient disinhibition of the homonymous α motor neurons following the transient facilitation of the facet joint Ib inhibitory di-synaptic reflex arc caused by spinal manipulation is vindicated by several EMG studies that have demonstrated a transient increase in EMG (spike) following the electromyographic response latency (Olsen 2015: 86; Pickar 2002: 364) as well as an increase in muscle strength and / or decrease in AMI immediately post-spinal manipulation (Dunning and Rushton 2009: 512; Picker 2002: 358, 364, 365; Suter and McMorland 2002: 541). DeVocht *et al.* (2005: 465) investigated the effect of spinal manipulation of the lumbar spine on EMG activity of localized tight muscle bundles in the segmental paraspinal muscles. They found that in some participants there was an immediate transient decrease in EMG activity after spinal manipulation, but also found in other participants a short lived spike in EMG activity post-spinal manipulation following the electromyographic response latency before returning to baseline. Herzog *et al*. (1999: 146) also demonstrated in their study that spinal manipulation applied to the cervical spine, thoracic spine, lumbar spine and sacroiliac regions increased the associated paraspinal EMG activity transiently in some participants following the electromyographic response latencies observed. Colloca and Keller (2001: 489) also reported in their study following the

electromyographic response latency observed, that the EMG amplitude increased to reach peak amplitude within 50 ms to 100 ms post-spinal manipulation before returning to baseline. Keller and Colloca (2000: 585) investigated the effect of spinal manipulation on segmental paraspinal muscle strength in participants with low back pain, by measuring the EMG activity during paraspinal extension isometric maximal voluntary contraction, before and after spinal manipulation. They found a significant transient increase in paraspinal EMG activity post spinal manipulation, compared to a placebo spinal manipulation which showed no significant change. Suter and McMorland (2002: 541) applied spinal manipulation to C5/C6/C7 segments with evidence of motor inhibition of the biceps brachii muscle using an interpolated twitch technique and EMG. Their results showed a significant reduction in biceps brachii muscle inhibition and an increase in biceps brachii muscle force post spinal manipulation. Dunning and Rushton (2009: 508) found a significant increase in biceps brachii muscle activity post-spinal manipulation of the ipsilateral C5/C6 segment in asymptomatic patients for neck pain and bilateral upper extremity pain, and based the underlying neural mechanism on an excitatory effect of spinal manipulation on the motor activity. Suter *et al.* (2000: 385) found a significant decrease in inhibition of the knee extensor muscles post manipulation of the ipsilateral sacroiliac joint in symptomatic patients with sacroiliac syndrome, anterior knee pain and EMG evidence of motor inhibition of the knee extensor muscles, and based the underlying neural mechanism on disinhibition of the involved neural pathways.

Spinal manipulation causes a transient increase in EMG following the electromyographic response latency and improves the functional capacity of the homonymous musculature (Olsen 2015: 86; Pickar 2002: 364) theoretically by reducing Ib inhibition on the homonymous motor neurons pool caused by alterations in the normal anatomical, physiological and / or biomechanical dynamics of the vertebral segment and thereby causing disinhibition of the homonymous α motor neurons (Dunning and Rushton 2009: 511, 512; Pickar 2002: 359, 360; Suter and McMorland 2002: 543, 544). By reducing or normalizing the alterations in the vertebral segment to its pre-injury / normal state, spinal manipulation can reduce the efferent manifestations induced by the altered arthrokinetic reflex of the facet joints (Olsen 2015: 85; Vernon 2010: 28; Gutterman 2005: 281; Pickar 2002: 359) and thereby reduce AMI of the homonymous muscle group (Middleditch and Oliver 2005: 247; Hendrickson 2002: 291).

5.5 LIMITATIONS

There are several limitations to this current study that need to be acknowledged. No verifications exist to ensure that the wanted spinal levels of manipulation will be indeed the specific C5/C6 level (Dunning and Rushton 2009: 502; Beffa and Mathews 2004: 118). In contrast, specific palpation of the vertebral bodies can lead to correct spinal level identification. In this current study specific

palpation was used to correctly identify the C5/C6 spinal levels (Benzel 2012: 958; Magee 2008: 180). Although the same person (the researcher) applied the C5/C6 spinal manipulation to all participants, the magnitude of the thrusting force of the C5/C6 spinal manipulation applied cannot be standardized between all of the participants. The exact replication of surface EMG electrode placement between all participants cannot be standardized and verified (Dunning and Rushton 2009: 502).

The exact replication of a low force isometric contraction of the biceps brachii muscle during the modified stretching between all the participants cannot be standardized. Each participant contracted their biceps brachii muscle at their own intensity by their free will which resulted in a large standard deviation in relevance to the differences between the interventions, because the generated biceps brachii muscle force and EMG RMS variables are entirely dependent on the voluntary effort of the participant (Lundy-Ekman 2013: 192; Merletti and Parker 2004: 6, 7).

CHAPTER 6 CONCLUSION AND RECOMMODATIONS

6.1 INTRODUCTION

This chapter provides the final conclusion of the research study conducted, as well as the final opinion of the researcher. It also provides recommendations based on the limitations of the study and new insights.

6.2 CONCLUSION

The aim of the study was to determine the effect of spinal manipulation of the C5/C6 segment during three sets of modified stretching of the biceps brachii on the muscle's electromyograph to investigate whether the electromyographic response post-spinal manipulation was affected by a facilitated GTO Ib inhibitory di-synaptic spinal reflex as part of the convergent input on the homonymous motor neuron pool excitability. The noteworthy scientific finding of the inverse relationship between muscle force and EMG RMS observed immediately post the spinal manipulation of the C5/C6 intervention provides the scientific evidence that spinal manipulation had a transient facilitatory effect on the GTO Ib inhibitory di-synaptic reflex arc as part of the convergent input on the homonymous motor neuron pool by causing facilitation of the facet joint Ib inhibitory di-synaptic reflex arc.

The electromyographic response post-spinal manipulation may provide the exact underlying neurophysiological mechanism of spinal manipulation on motor activity. The electromyographic response post-spinal manipulation consists of an initial latent period consisting of a few milliseconds in duration followed by a transient increase in EMG amplitude, or, solely, a transient decrease in the EMG amplitude or decrease in the excitability of the homonymous motor neuron pool immediately post-spinal manipulation (Olsen 2015: 86; Pickar 2002: 364). The underlying neurophysiological mechanism for the electromyographic response latency occurring immediately post-spinal manipulation is largely unknown (Pickar 2002: 364). Several EMG studies in the chiropractic literature have postulated that the underlying neurophysiological mechanism for the transient increase in EMG amplitude immediately post-spinal manipulation as well as the improvement of muscle functioning entails either facilitation or disinhibition of the involved neural pathways (Pickar 2002: 359, 360; Dunning and Rushton 2009: 511, 512; Suter and McMorland 2002: 543, 544).

The finding of spinal manipulation causing transient facilitation of the facet joint Ib inhibitory di-synaptic spinal reflex arc possibly provides the scientific evidence of the exact underlying neurophysiological mechanism of spinal manipulation on motor activity as well as for the electromyographic response latency occurring immediately post-spinal manipulation. The stimulatory effect of spinal manipulation on type I-III nerve endings in the facet joint capsular tissue and synovium (Olsen 2015: 82; Millan *et al.* 2012: 24; Vernon 2010: 24; Pickar 2002: 358, 359) cause transient facilitation of Ib inhibition on the homonymous motor neuron pool via the facet joint Ib inhibitory di-synaptic reflex arc as vindicated by several EMG studies that demonstrated the electromyographic response latency, transient decrease in the EMG amplitude as well as transient decrease in homonymous motor neuron pool excitability immediately post-spinal manipulation (Olsen 2015: 86; Dishman *et al*. 2002: 318; Pickar 2002: 364; Colloca and Keller 2001: 489). The notion that spinal manipulation causes disinhibition of the homonymous α motor neurons immediately after the electromyographic response latency post-spinal manipulation is highly plausible as vindicated by several EMG studies that demonstrated a transient increase in EMG following the electromyographic response latency and an improvement in the functional capacity of the spinal segmentally innervated musculature post-spinal manipulation (Pickar 2002: 359, 360; Dunning and Rushton 2009: 511, 512; Suter and McMorland 2002: 543, 544). Spinal manipulation can cause disinhibition of the homonymous α motor neurons by normalizing the mechanoreceptors Aβ-fiber afferent discharge in the facet joint capsular tissue and synovium (Olsen 2015: 82; Millan *et al*. 2012: 24; Vernon 2010: 24; Pickar 2002: 358, 359) by reducing or normalizing the alterations in the normal anatomical, physiological and / or biomechanical dynamics of the individual vertebral segments to its pre-injury / normal state and in doing so reduce the efferent manifestation of Ib inhibition on the homonymous motor neuron pool induced by the altered arthrokinetic reflex of AMI (Olsen 2015: 85; Rice *et al.* 2014: 503; Petty 2011: 16; Pickar 2002: 359).

The finding of spinal manipulation causing transient Ib inhibition on the homonymous motor neuron pool also has clinical implications for rehabilitation practitioners and physical therapists. Spinal manipulation may achieve habituation to altered arthrokinetic reflex arcs of AMI in nature and thereby cause a decrease in muscle weakness and an increase in strength of the homonymous musculature as vindicated by several EMG studies that showed an incremental increase in muscle force and muscle activity over time and an increase in muscle strength post-spinal manipulation (Olsen 2015: 86; Du Plessis 2014; Pickar 2002: 365; Dishmen *et al*. 2002: 1). This finding may also provide the scientific evidenced based support to the suggestion that for optimal management of patients with muscle weakness suspected to be of arthrogenic nature, the application of spinal manipulation to the segmentally innervated facet joints may be a beneficial approach before traditional strength training is initiated (Dunning and Rushton 2009: 512).

6.3 RECOMMENDATIONS

This current study raises areas for further research. Further research is warranted to add statistical significance to the noteworthy finding of the inverse relationship between muscle force and EMG RMS observed immediately post-spinal manipulation. It would be useful to investigate the effect of spinal manipulation on the facilitated GTO Ib inhibitory di-synaptic reflex arc when eliminating the effect of voluntary effort by the participants on the homonymous motor neuron pool excitability. The insignificant results between the placebo AAI interventions and SMT intervention immediate pre- and post-intervention for all variables (biceps brachii muscle force and EMG RMS) was possible caused by the large standard deviation in relevance to the differences between the interventions. By using a muscle stimulator at the same low-frequency for all participants to induce non-linear GTO Ib afferent activity during passive stretching of the biceps brachii muscle; the exact replication of intensity of biceps brachii muscle contraction between all the participants can be standardized, the corticospinal influence on the homonymous motor neuron pool excitability can be excluded (Dishman *et al.* 2002: 318), the large standard deviation can be reduced, and thereby statistical significance may be added to the noteworthy scientific finding of the inverse relationship between muscle force and EMG amplitude observed immediately post-spinal manipulation.

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APPENDIX A

LETTER OF INFORMATION

Title of the Research Study: The effect of spinal manipulation on biceps brachii muscle activity

Principal Investigator/s/researcher: Aldo Victor, BTech: Chiropractic.

Co-Investigator/s/supervisor/s: Dr Grant Matkovich, MTech: Chiropractic

Brief Introduction and Purpose of the Study:

This pre-post-test study will recruit 20 participants (Esterhuizen, 2015) of any gender or race residing within the Ethekweni municipality between the ages of 18 and 40. The participants must be asymptomatic for neck pain and bilateral upper extremity pain. Three sets of modified stretching (autogenic inhibition) of the biceps muscle with two minute rest interval between the sets will be performed per participant. The first and second sets of modified stretching will include an intervention with an Activator II Adjusting instrument (AAI). The third set of modified stretching will include spinal manipulation of the C5/C6 segment. Surface electromyography readings and dynamometer readings will be recorded concurrently before, during and immediately after the interventions. A Biopac - Bionomadix complete wireless research system with 4 channel EMG recording with a TSD121C Hand Dynamometer 100kg will be used.

The study will investigate the effect of spinal manipulation of the C5/C6 segment during three sets of modified stretching (autogenic inhibition) of the biceps muscle on the muscle's electromyograph.

Outline of the Procedures:

Dear participant,

Thank you for your participation in my research study. Your time and help are greatly appreciated.

During the first consultation at the Chiropractic Day Clinic you will read this information sheet and ask any questions about the research. If you agree to take part in this research, you will be obliged to sign an informed consent form. An hour long appointment will follow. Please do not take any form of medication or partake in any activity that can put you at risk for injury to your neck or biceps muscle in the duration of the research.

You will meet the researcher at the DUT Chiropractic Day Clinic for your initial appointment. The researcher will take a case history and perform a physical examination and a cervical spine regional examination, in order to see if you meet the inclusion and exclusion criteria. Once you have met the inclusion and exclusion criteria, you will be accepted into this study and a follow up appointment will be scheduled for you at the Chiropractic Day Clinic within two weeks of your initial appointment.

You will meet the researcher at the Chiropractic Day Clinic for your follow up appointment. You will perform three sets of modified stretching of your biceps muscle with two minute rest interval between the sets, and during each set of modified stretching you will receive an intervention. The first and second interventions will include an Activator II Adjusting Instrument. The third intervention will include spinal manipulation of the C5/C6 segment. A full description of the procedure will be explained verbally to you and time will be given for any queries or questions regarding the procedure of this study.

You will be asked to remove any clothing covering the area around the biceps muscle of interest and appropriate clothes (clinic gown for females) will be provided if necessary. You will be instructed by the researcher to be seated on a chair in front of a small bed or table. You will be further instructed to place your arm on the table. Your arm will be placed on the table so that your elbow will be horizontal to your shoulder. Your elbow will be extended and your

forearm supinated passively by the researcher. Your extended elbow will remain in contact with the table and your distal supinated forearm will hang off the table. Proper skin preparations will be applied to your biceps muscle at the sites of the Surface Electromyography electrodes placement. The researcher will ensure that your skin is clean from dirt and dry by using a towel. Your skin will be swept gently with fine sand paper abrasion tape to remove any dead skin cells that could impede with the EMG signal, prior to wiping the sites with a cleansing wipe and allowing it to air dry. Surface Electromyography (SEMG) electrodes will then be safely and correctly placed on your biceps muscle of interest.

Before the modified stretching of your biceps muscle commences and the intervention are applied, the researcher will identify C5/C6 spinal levels on you by using specific palpation technique. The researcher will stand behind you. The researcher will then palpate the occiput of your skull. The researcher will descend from the occiput in the posterior midline of your neck, the C2 spinous process will be palpated as the first bump. The next spinous process which are most palpable are C6 and C7. The researcher will then differentiate between C5, C6 and C7 spinous process by passively flexing and extending your neck. With the flexion and extension movements the C5 and C6 spinous process will move in and out and the C7 spinous process will remain stationary. The C5/C6 spinal levels will be marked on your skin by using a non-permanent marker pen. Horizontal lines will then be drawn on your skin through C5 and C6 spinous process.

Following the placement of the SEMG electrodes on your biceps muscle, a 10mm by 2m tie down strap will be strapped to your distal forearm by the researcher. The pull side of a Biopac - TSD121C Hand Dynamometer 100kg will be attached to the tie down strap. Another 10mm by 2m tie down strap will be used to attach the push side of the dynamometer to a 1kg plate, which will hang below your distal forearm. The weight of the 1kg plate will fully extend your elbow and passively stretch your biceps muscle to elicit the first phase of the modified stretching (autogenic excitation). This will be done during all the three sets of modified stretching of your biceps muscle. You will then be instructed by the researcher to perform a light isometric contraction against the weight of the 1kg plate for ten seconds by contracting your biceps muscle. The light isometric muscle contraction against the weight of the 1kg plate will elicit the second phase of the modified stretching (the autogenic inhibition phase). This will also be done during all the three sets of modified stretching of your biceps muscle. Before you are asked to perform the light isometric contraction, you will be instructed to maintain the same biceps muscle contraction for the whole duration of the ten seconds of light isometric contraction against the weight of the 1kg plate, in order to rule out the most unwanted voluntary effort by your free will. You will also be instructed to only contract your biceps muscle while you perform the light isometric contraction and not to flex your elbow. Your elbow must remain stationary to hold your biceps muscle in a passively stretched position. You will also be notified beforehand, that by the fourth second while you are performing the light isometric contraction against the weight of the 1kg plate for ten seconds in all the three sets of modified stretching of your biceps muscle, you will receive an intervention. A stop watch will be placed in front of you.

You will receive the first intervention with an Activator ii Adjustment instrument (AAI) during the first set of modified stretching of your biceps muscle. The researcher will stand behind you. By the fourth second while you are performing the light isometric contraction against the weight of the 1kg plate for ten seconds, the researcher will place an AAI against the back of your neck. The researcher will then pull the trigger of the AAI to deliver the first intervention. The researcher will remove the contact of the AAI from your neck immediately after the first intervention.

After you performed the light isometric contraction against the weight of the 1kg plate for ten seconds during the first set of modified stretching of your biceps muscle, the tie down strap with the attached dynamometer and 1kg plate will be unstrapped from your distal forearm by the researcher. You will then be instructed to rest for two minutes. You will also be instructed to stay in the seated position for the full two minutes duration of rest. The SEMG electrodes will not be removed from your biceps muscle during the rest period.

After the two minutes of rest, the researcher will induce the first phase of the modified stretching again by passively extending and supinating your arm and slowly strapping the tie down strap with the attached dynamometer and 1kg plate to your distal forearm. You will then receive the second intervention with an Activator ii Adjustment instrument during the second set of modified stretching of your biceps muscle. The researcher will stand behind you. By the fourth second while you are performing the light isometric contraction against the weight of the 1kg plate for ten seconds, the researcher will place an AAI against the back of your neck. The researcher will then pull the trigger of the AAI to deliver the second intervention. The researcher will remove the contact of the AAI from your neck immediately after the second intervention.

After you performed the light isometric contraction against the weight of the 1kg plate for ten seconds during the second set of modified stretching of your biceps muscle, the tie down strap with the attached dynamometer and 1kg plate will be unstrapped from your distal forearm by the researcher. You will then be instructed to rest for two minutes again. You will also be instructed to stay in the seated position for the full two minutes duration of rest. The SEMG electrodes will not be removed from your biceps muscle during the rest period.

After the two minutes of rest the researcher will induce the first phase of the modified stretching again by passively extending and supinating your arm and slowly strapping the tie down strap with the attached dynamometer and 1kg plate to your distal forearm. You will then receive the third intervention during the third set of modified stretching of your biceps muscle. The researcher will stand behind you. By the fourth second while you are performing the light

isometric contraction against the weight of the 1kg plate for ten seconds, the researcher will apply a high velocity- low amplitude thrust in an anterior rotational vector along the C5/C6 facet joint planes to deliver the C5/C6 spinal manipulation. The researcher will then re-position your head to the starting position and remove any contact from you immediately after the third intervention.

After the third set of modified stretching of your biceps muscle, the researcher will unstrap the tie down strap with the attached dynamometer and 1kg plate from your distal forearm. The SEMG electrodes will then be safely and correctly removed from your biceps muscle and you will be thanked for your participation in this study.

The SEMG readings and dynamometer readings will be recorded concurrently for all the three sets of modified stretching of your biceps muscle; before, during and immediate after all the interventions.

Risks or Discomforts to the Participant:

All objective measurements, interventions and procedures utilized in this study are non-invasive and will be applied within the safety procedure parameters. The modified stretching performed may lead to transient delayed-onset muscle soreness of your biceps muscle. This is a normal response to the modified stretching and usually abates after 48-72 hours. Side effects of spinal manipulation may occur and include transient local soreness, discomfort, stiffness, headaches and nausea. Side effects of spinal manipulation are uncommon and usually abate 48-72 hours after the spinal manipulation.

Benefits:

The research may benefit you directly by reducing the arthrogenic muscle inhibition (muscle weakness) of your biceps muscle and improving the function of your biceps muscle.

The results may indicate that spinal manipulation of a subluxated vertebral segment can cause reduced inhibition of the Ib inhibitory spinal pathway and thus reduces AMI and improve muscle functioning of the muscle(S) innervated by those dysfunctional spinal levels. These findings could give chiropractors a better understanding of the neurophysiological effect of spinal manipulation.

Reason/s why the Participant May Be Withdrawn from the Study:

You will not be forced to participate in the study and will do so by free will. You are free to withdraw from this study at any stage without any negative repercussions.

Remuneration:

You will not be offered any form of remuneration for taking part in the study.

Costs of the Study:

The initial and follow up consultation are free of charge.

Confidentiality:

You will be given a code and your name will not be used on the data sheets. Only the clinic reception and researcher will have access to the names and the codes.

The SEMG readings and dynamometer readings will be recorded and stored on the hard drive directly and processed to obtain the digital values for the identified variables via the Biopac - MP 150 Data Acquisition system and AcqKnowledge software. All of the electronic data obtained will be burned on a CD. The electronic data obtained will be completely removed from the hard drive, after the electronic data has been burned on the CD.

The research data with the electronic data obtained in the form of a CD will be kept in a safe place at the Chiropractic Day Clinic for a period of 5 years. After the period of 5 years the research data will be shredded and the CD will be destroyed. Your name will not appear on any of the data sheets or thesis.

Please feel free to ask questions on any aspect of this study. Should you have any complaints or queries, please contact my research supervisor or the Constitutional Research Ethics Committee Administration: 031 373 2900.

Yours sincerely,

Aldo Victor.

Research-related Injury:

There will be no compensation in the event of an injury.

Should you develop pain or any adverse effect; you will be withdrawn from the study immediately, the DUT Head of Department of Chiropractic will be contacted and the adverse event will be reported to IREC. You will be allowed to contact your personal general medical practitioner, alternatively you will be referred to the closest local hospital with a referral letter.

King Dinuzulu Hospital:

Telephone: +27 31 2426000

Physical Address: Corner of Dr R D Naidoo drive and Viola Road, Sydenham Durban.

Directions from the DUT Chiropractic Day Clinic to King Dinuzulu Hospital: Head west on Steve Biko road toward Heswall road (180m), turn right onto Botanic Gardens road (550m), continue onto Edith Benson Cres (450m), turn left onto John Zikhali road (950m), turn right onto Vause road (140m), slight left onto Overport drive (170m), Slight right onto Peter Mokabe Ridge (600m), turn left onto Crescent St (900m), turn right onto Felix Dlamini road / M10 (350m) and turn left onto Rd Naidu Dr (1,0km) to reach King Dinuzulu Hospital on your right.

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

DUT Chiropractic Head of Department: Dr. A. Docrat	**Contact number:** 031 373 2589
Supervisor: Dr Grant Matkovich	**Contact number:** 0825683986
Co-Supervisor: Professor Threethambal Puckree	**Contact number:** 031 373 2967

Institutional Research Ethics administrator can be contacted on 031 373 2900 and complaints can be reported to the DVC: TIP, Prof F. Otieno on 031 373 2382 or dvctip@dut.ac.za.

CONSENT

Statement of Agreement to Participate in the Research Study:

- I hereby confirm that I have been informed by the researcher, _____ (name of researcher), about the nature, conduct, benefits and risks of this study - Research Ethics Clearance Number: _____,
- I have also received, read and understood the above written information (Participant Letter of Information) regarding the study.
- I am aware that the results of the study, including personal details regarding my sex, age, date of birth, initials and diagnosis will be anonymously processed into a study report.
- In view of the requirements of research, I agree that the data collected during this study can be processed in a computerised system by the researcher.
- I may, at any stage, without prejudice, withdraw my consent and participation in the study. I
- have had sufficient opportunity to ask questions and (of my own free will) declare myself prepared to participate in the study.
- I understand that significant new findings developed during the course of this research which may relate to my participation will be made available to me.

_____ _____ _____

Full Name of Researcher **Date** **Signature**

_____ _____ _____

Full Name of Witness (If applicable) **Date** **Signature**

_____ _____ _____

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

_____ _____ _____ _____

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

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

APPENDIX B

CHIROPRACTIC PROGRAMME

CHIROPRACTIC DAY CLINIC

CASE HISTORY

Patient:_____ Date:_____

File # _____ Age:_____

Sex: _____ Occupation:_____

Student:_____ Signature:_____

FOR CLINICIANS USE ONLY:

Initial visit

Clinician: Signature:

Case History:

Examination:

Previous: Current:

X-Ray Studies:

Previous: Current:

Clinical Path. lab:

Previous: Current:

CASE **STATUS:**

PTT:	Signature:	Date:

CONDITIONAL:
Reason for Conditional:

Signature: Date:

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

Case Summary signed off: Date:

Student's Case History:

1. Source of History:

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

3. Present Illness:

	Complaint 1(principle complaint)	**Complaint 2 (additional or secondary complaint)**

Location Onset : Initial: Recent: Cause: Duration Frequency Pain (Character) Progression Aggravating Factors Relieving Factors Associated S & S Previous Occurrences Past Treatment Outcome:		

4. Other Complaints:

5. Past Medical History:

General Health Status

Childhood Illnesses

Adult Illnesses

Psychiatric Illnesses

Accidents/Injuries

Surgery

Hospitalizations

6. Current health status and life-style:

Allergies

Immunizations

Screening Tests incl. x-rays

Environmental Hazards (Home, School, Work)

Exercise and Leisure

Sleep Patterns

Diet

Current Medication

Analgesics/week:

Other (please list):

Tobacco

Alcohol

Social Drugs

7. Immediate Family Medical History:

Age of all family members

Health of all family members

Cause of Death of any family members

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

8. Psychosocial history:

Home Situation and daily life

Important experiences

Religious Beliefs

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

General

Skin

Head

Eyes

Ears

Nose/Sinuses

Mouth/Throat

Neck

Breasts

Respiratory

Cardiac

Gastro-intestinal

Urinary

Genital

Vascular

Musculoskeletal

Neurologic

Haematological

Endocrine

Psychiatric

APPENDIX C

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		

6.3.1.1.1.1 SYSTEM SPECIFIC EXAMINATION:

CARDIOVASCULAR EXAMINATION

RESPIRATORY EXAMINATION

6.4 *ABDOMINAL EXAMINATION*

6.5 *NEUROLOGICAL EXAMINATION*

6.5.1 COMMENTS

Clinician: **Signature:**

APPENDIX D

CHIROPRACTIC PROGRAMME

REGIONAL EXAMINATION – CERVICAL SPINE

Patient: File No:

Date: Student:

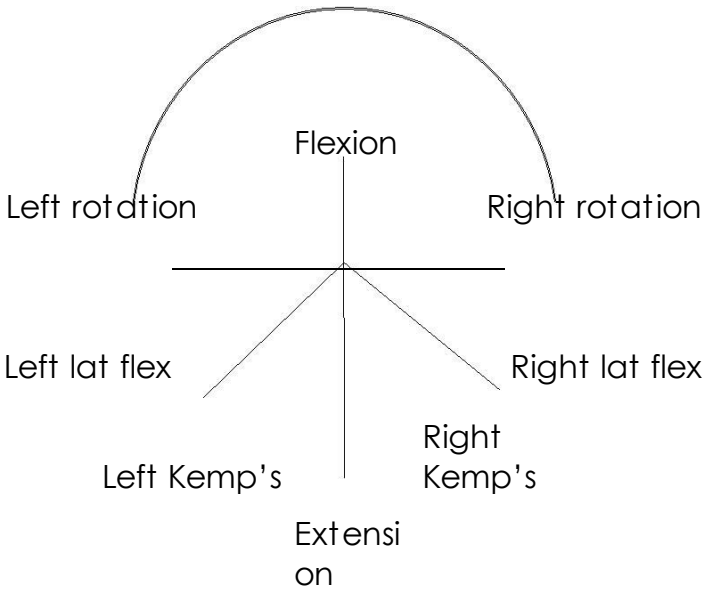
Clinician: Sign:

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					

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:

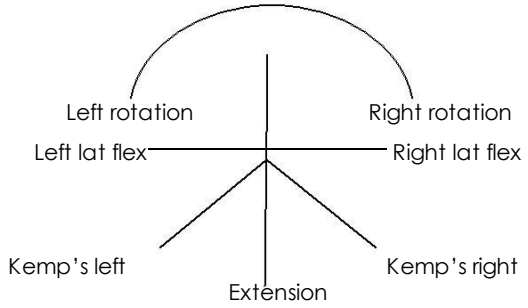
BASIC EXAM: THORACIC SPINE:

Case History:

ROM:

Flexion

Passive:
RIM:
Orthopaedic:
Neuro:
Vascular:



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

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APPENDIX E

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Patient Name: ***File #:***

Page:

Date: ***Visit:*** ***Intern:***

Attending Clinician: ***Signature:***

S: *Numerical Pain Rating Scale (Patient)* *Intern Rating* ***A:***

Least ***0 1 2 3 4 5 6 7 8 9 10*** *Worst*

0: ***P:***

E:

Special attention to: ***Next appointment:***

APPENDIX F

Data collection sheet

	Baseline (500 ms pre-intervention)	Post-intervention (first 500 ms)	Percentage difference (%)	
RMS (µV)				
Placebo AAI 1				
Placebo AAI 2				
SMT				
Force (kg)				
Placebo AAI 1				
Placebo AAI 2				
SMT				

APPENDIX G

Research is currently being conducted at the Durban University of Technology Chiropractic Day Clinic on

MUSCLE WEAKNESS

Is your BICEPS MUSCLE WEAKER THAN NORMAL?

Are you between the ages of 18 and 40?

TREATMENT MAY BE PROVIDED

To those who qualify to take part in this study

Contact Aldo Victor on 072 215 6875

Or the Chiropractic Day Clinic on 031 373 2205

to see if you qualify for this study.

APPENDIX H

The prospective participants will be asked the following questions during the telephonic conversation to see if they qualify:

QUESTIONS ASKED	EXPECTED ANSWERS
"Would you mind answering a few questions to see if you qualify to participate in the study?"	**Yes**
"How old are you?"	**18-40 yoa**
"Do you have any neck pain?"	**NO**
"Have you had any neck pain in the past two weeks?"	**NO**
"Have you had any surgery to your neck?"	**NO**
"Have you had any injury to your neck in the past two months?"	**NO**
"Do you have any pain in your right or left arm? Including any shoulder pain or elbow pain"	**NO**
"Have you had any surgery to your right or left arm?"	**NO**
"Have you had any injury to your right or left arm in the past two months? Including any injury to your biceps muscle, shoulder or elbow"	**NO**

MEMORANDUM

To : Prof Puckree
Chair : RHDC

Prof Adam
Chair : IREC

From : Dr Charmaine Korporaal
Clinic Director : FoHS Clinic

Date : 24.02.2015

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

---

Permission is hereby granted to :

Mr Aldo Victor (Student Number: 21011494)

Research title : "The effect of spinal manipulation on biceps brachii arthrogenic muscle**".**

Mr Victor, is requested to submit a copy of his RHDC / IREC approved proposal along with proof of his MTech: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 him seeing patients.

Thank you for your time.

[redacted]

Dr Charmaine Korporaal
Clinic Director : FoHS Clinic

Cc: Chiropractic Day Clinic : Mrs Pat van den Berg
Supervisor : Dr Grant Matkovich

Appendix J

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

P O Box 1334, Durban, South Africa,
4001 Tel:031 3732900 Fax: 031 3732407 Email bwshad dutacza
http://www.dut.ac.za/research/institutional_research_ethics
www.dut.ac.za

10 September 2015

IREC Reference Number: REC 111/15

Mr A A Victor The Shades
Flat number 7l
23 Weaver Crescent
Umhlanga Rocks
4319

Dear Mr Victor

The effect of spinal manipulation on biceps brachii muscle activity

I am pleased to inform you that Full Approval has been granted to your proposal REC 111/15.

The Proposal has been allocated the following Ethical Clearance number IREC 112/15. Please use this number in all communication with this office.

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

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

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

Yours Sincerely

[redacted]

Professor M N Sibiya
Deputy Chairperson: IREC

DUT DURBAN UNIVERSITY OF TECHNOLOGY
-09- 10
INSTITUTIONAL RESEARCH ETHICS COMMITTEE
P O BOX 1334 DURBAN 4000 SOUTH AFRICA