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THE EFFECTIVENESS OF TREATMENT AT PAIN THRESHOLD VERSUS PAIN TOLERANCE USING ISCHAEMIC COMPRESSION

A dissertation submitted to the Faculty of Health Sciences, University of Johannesburg, as partial fulfilment of the requirements for the Masters Degree in Technology in Chiropractic by:

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DECLARATION

I declare that this dissertation is my own, unaided work. It is being submitted for the Masters Degree in Technology in the program Chiropractic at the University of Johannesburg. It has not been submitted before for any degree or examination in any other Tertiary Institute.

__________________________
Fatima Ismail

On this ____________________ day of ____________________ 2014
AFFIDAVIT

TO WHOM IT MAY CONCERN

This serves to confirm that I, Fatima Ismail, ID number 880422 0235 086, Student number 2005 839 12 enrolled for the Qualification M Tech Chiropractic.

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DEDICATION

In completion of this study I would like to thank Allah, the All Mighty, for blessing me with the knowledge, guidance and perseverance to complete the task at hand.

To my family, most notably my parents, I say thank you for allowing me the opportunity to fulfil my life with this degree. It was only with your unwavering support that I was able to complete this degree.

To Haroon, my pillar of strength, thank you for your ongoing love and encouragement throughout my years of study.
ACKNOWLEDGEMENTS

Dr Caroline Hay and Dr Christopher Yelverton, my supervisors, thank you for the infinite amount of work and time spent on guiding me through this process. Your invaluable input is much appreciated.

To all my friends and classmates, Jasantha, Sarah and Jaidan, thank you for the love, support and laughter throughout this journey that we have experienced together. There were too many stressful times but nothing ever dampened our spirits.

To all the participants that took part in this study, without your time this would not have been possible, thank you.
ABSTRACT

There is research to show that ischaemic compression is very effective in the treatment of myofascial trigger points (MFTP’s). It is less invasive when compared to other treatment methods such as dry needling however; according to Gulick (2010) there is a lack of randomised controlled studies with regard to standard ischaemic compression treatment protocols. This includes the appropriate amount of pressure, duration of compression or frequency of treatment (Gulick, 2010).

This study was conducted in order to determine whether ischaemic compression that is applied at pain threshold would have a similar effect when compared to ischaemic compression at pain tolerance in the treatment of active rhomboid major and minor myofascial trigger points, using a hand held algometer. This study was specifically undertaken to provide more information regarding the most effective method of ischaemic compression with regard to the amount of pressure that is most suitable during treatment. The results of this study could potentially improve patient comfort and reduce pain during treatment by showing that treatment at pain threshold may be as effective as conventional ischaemic compression at pain tolerance.

It was hypothesized that ischaemic compression applied at pain threshold may have a similar effect as application at pain tolerance by having a positive outcome on the subjective and objective findings in patients with active myofascial trigger points of the rhomboid major and minor muscles.

Participants were recruited into the study by word of mouth as well as with the use of advertisements that were placed around the University of Johannesburg Doornfontein campus and clinic. Thirty participants that conformed to the specified limitations and diagnostic criteria were accepted to partake in this study. The participants were then placed in a random and stratified manner into two groups of 15, based on age and gender. Group A received ischaemic compression of the rhomboid major and minor muscles at pain threshold while Group B received ischaemic compression of the same muscles at pain tolerance. Ischaemic compression was administered over a 30 second duration. Each participant received 2 treatments a week for 3 weeks while a 7th and final visit served only for measurement taking.
The subjective data was assessed using the Numerical Pain Rating Scale based on the referral pain felt by the patient. The objective data was obtained using the readings taken with the hand held algometer, measuring the amount pressure needed to first elicit pain (pain threshold) and then the pressure needed to cause the most pain that the participant was able to tolerate (pain tolerance).

The findings of this study show that Group A had statistically significant improvements with regard to the Numerical Pain Rating Scale, pain threshold and pain tolerance algometer readings, over time. Group B also showed statistically significant improvements with respect to the Numerical Pain Rating Scale, pain threshold and pain tolerance algometer readings, over time. Group A and Group B also both showed clinical improvements over time. The clinical improvement seen in Group B was greater, however it was not significant.

Based on the results obtained during this study, it could be ascertained that ischaemic compression at both pain threshold and at pain tolerance could be used to effectively treat active myofascial trigger points in the rhomboid major and minor muscles. Ischaemic compression at pain tolerance seemed to be more effective clinically however, it could not be statistically concluded whether one treatment was superior to the other as both treatments faired equally throughout the trial period of this study.
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CHAPTER ONE – INTRODUCTION

1.1 Problem and it’s Setting

Skeletal muscle is the largest organ in the human body accounting for 50 % of the body’s weight. Any one of these muscles can develop pain and dysfunction known as myofascial pain syndrome (Ching, 2007). Wear and tear accompanied with daily activity primarily has an effect on contractile muscle tissue yet; little attention is paid towards myofascial pain in medical texts (Travel, Simons and Simons, 1999). Currently, the leading diagnosis of pain sufferers amongst pain management physicians and general practitioners is that of myofascial pain (Harden, Breuhl, Gass, Niemiec, and Barbick, 2000).

A myofascial trigger point is a hyperirritable focal point within a taut band of skeletal muscle fibres. When these trigger points are compressed they refer pain in a characteristic pain referral pattern and evoke autonomic phenomena unique to the trigger point and the involved muscle (Travell et al., 1999). A study undertaken by Harden et al., (2000) found that myofascial pain syndrome is a legitimate diagnosis distinct from that of fibromyalgia.

There are numerous therapies available for the treatment of myofascial trigger points however, there is little consensus as to the best treatment protocol (Vernon and Schneider, 2009). Myofascial trigger point treatment aims to decrease pain and relieve tightness of hypertonic muscles (Han and Harrison, 1997). Travell and Simons (1983) describe ischaemic compression as a digital pressure which is firmly applied to the involved myofascial trigger point. Ischaemic compression has been proved to be a safe and effective technique and is known for successfully reducing myofascial trigger point tenderness (De Las Peñas, Alonso-Blanco, Fernandez-Carnero, and Miangolarra-Page, 2006).

Clinicians in many health care disciplines routinely identify and treat myofascial trigger points by non-invasive methods including that of ischaemic compression. However, there is a considerable discrepancy in the vast range of non-invasive treatment modalities proposed as effective for myofascial trigger points (Dommerholt and Huijbregts, 2010). This can be seen by the many ischaemic compression treatment protocols used in research trials. Hou, Tsai, Cheng, Chung and Hong (2002) propose a treatment protocol of
ischaemic compression at either a low pressure below that of pain threshold for prolonged periods or an increased pressure at pain tolerance for short periods of time. Bron, de Gast, Dommerholt, Stegenga, Wensing, and Oostendorp (2011) propose a gentle gradually increasing pressure that is applied until a definite increase in tissue resistance is felt. The pressure is then maintained until there is a relief of tension under the palpating finger or a decline in pain is felt. Following this, the procedure is repeated several times until pressure on the trigger point would provoke only a slight discomfort without pain.

Pain is described to be an unpleasant sensory and emotional experience that is associated with actual or potential damage to tissue. Pain threshold is the minimum intensity of a stimulus that is perceived as painful while pain tolerance is the maximum intensity of a pain producing stimulus that a subject is willing to accept in a given situation (Mersky and Bogduk, 1994). As pain is a major precursor of stress (Pasero, Paice and Mc Caffery, 1999) it would seem that reducing pain during treatment may be beneficial in the overall outcome of treatment.

1.2 Aim of the Study

The aim of this study was to determine whether a pressure that was applied at the first point of pain (pain threshold) during the ischaemic compression of a myofascial trigger point, would have a similar effect when compared to a pressure that was administered at the maximum level of pain a person was able to tolerate (pain tolerance).

1.3 Benefits of the Study

The potential outcome of this study could determine whether ischaemic compression administered at pain threshold produced the same result as with pain tolerance thereby allowing the Chiropractic profession to utilise the most effective method. If it was found that ischaemic compression was just as effective when applied at pain threshold then this would greatly increase patient comfort and reduce pain during treatment which could have a positive effect on the outcome of treatment. This study could also provide further knowledge on the treatment regimen of ischaemic compression in the Chiropractic profession as well as other professions that utilise ischaemic compression as a treatment method by giving clarity on the most effective protocol that could be used in practice.
CHAPTER TWO – LITERATURE REVIEW

2.1 Introduction

This chapter serves to give detail on relevant previously published literature and research thereby forming the necessary background literature for this specific study. Emphasis is placed on the anatomy of skeletal muscle specifically the rhomboid major and minor muscles, the development of myofascial trigger points (MFTP’s), ischaemic compression as a treatment, pressure mechanoreceptors and certain aspects of pain. For the purposes of this study, the rhomboid major and minor muscles will be referred to collectively as the rhomboid muscles.

2.2 Skeletal Muscle

2.2.1 Anatomy and physiology of muscle

Skeletal muscle is the largest organ in the human body accounting for 50% of the body's weight (Ching, 2007). As seen in Figure 2.1, each muscle is entirely surrounded by a layer of dense connective tissue called the epimysium that is continuous with a corresponding tendon. Numerous bundles of muscle fibers, termed fascicles, make up each muscle. These fascicles are separated from each other by a layer of connective tissue called the perimysium. The endomysium is the connective tissue layer that separates individual muscle fibers (Seidman, 2013).

Figure 2.1: Gross Anatomy of Muscle (Chavez-Murphey, 2012)
a. Histological features

Skeletal muscle cells contain the same organelles found in other cell types and can be seen in Figure 2.2. The following describes these organelles in detail (Forsman, 2013):

i. Nucleus: contains the genetic material of the muscle cell;
ii. Sarcolemma: this is the name given to the plasma membrane of the muscle cell;
iii. T-tubules: these are specialized invaginations of the sarcolemma that run transversely across the cell. The T-tubules are essential for carrying the depolarization brought to the cell by a motor nerve;
iv. Cytosol: this is the cytoplasm of the muscle cell;
v. Sarcoplasmic reticulum: this is the endoplasmic reticulum of the muscle cell;
vi. Terminal cisternae: these are sac-like regions of the sarcoplasmic reticulum that act as calcium storage sites. The calcium ions stored in the terminal cisternae are essential in muscle contraction. In skeletal muscle two terminal cisternae are associated with a single T-tubule to form a structure known as a triad;

vii. Mitochondria: these are sites of energy production namely adenosine triphosphate (ATP) synthesis within the muscle cell;

viii. Myofibril: these are cylindrical bundles of contractile proteins found within the muscle cell. There are several myofibrils within each muscle cell. Myofibrils are composed of individual contractile proteins called myofilaments. These myofilaments are generally divided into thick and thin myofilaments. The thin myofilaments are composed mainly of a protein known as actin. Actin filaments are anchored into the z-line of a sarcomere. The thick myofilaments are composed mainly of the protein myosin. It is the orderly overlapping of the actin and myosin filaments that give skeletal muscle their striated appearance (light and dark bands);

ix. The A-band: this is the dark band and corresponds to the length of a bundle of myosin filaments. Muscle contraction occurs as a result of the sliding of the myofilaments past each other;

x. The I-band (light band): the I-bands are composed mainly of actin filaments and are divided in half by a protein disc known as the Z-line. Actin filaments are
anchored into the Z-line. During muscle contraction the actin filaments slide over the myosin filaments which results in the shortening of the I-band;

xi. H-zone: this zone is found in the middle of the A-band and corresponds to the area where myosin is not overlapped by actin. During muscle contraction the actin sliding over the myosin encroaches into this area so that the H-zone shortens. In the middle of the H-zone a dark band known as the M-line is found. The M-line is comprised of protein fibers that function to anchor the myosin filaments;

xii. Sarcomere: this is the area between two Z-lines and is the functional or contractile unit of muscle.

---

**Figure 2.2: Microscopic Anatomy of Skeletal Muscle (Forsman, 2013)**

Muscles consist of extrafusal and intrafusal fibers. Intrafusal fibers or muscle spindles function to monitor muscle length and tone. They are sensitive to any rate of change in length. Muscle spindles are innervated by gamma fibers that set the length and tone of the muscle spindles. Muscle spindles are located throughout the muscle and provide a continuous feedback enabling the central nervous system to control muscle activity. Muscle stretching increases the rate of impulses sent to the central nervous system while muscle shortening decreases the rate of impulses to the central nervous system (Grubb, Hagedorn, Inoue, Leake, Lounsberry, Love, Matus, Morris, Stafford, Staton and Waters, 2010).
Extrafusal fibers are innervated by alpha motor neurons. Golgi tendon organs lie in the extrafusal fibers and are sensitive to muscle tension. Golgi tendon organs are located within the muscle tendons, thereby preventing muscles from developing excessive tension which could lead to muscle tears or an avulsion (Grubb et al., 2010).

b. Innervation

Muscles contract after receiving a motor impulse from a motor nerve. These nerve impulses serve only a limited number of muscle fibers. A motor unit consists of the muscle fibers that are served by a single motor neuron as seen in Figure 2.3. Motor units allow for selective contraction of muscle fibers so that there is control of the strength and extent of muscle contraction. Without motor units a nerve impulse to the muscle would result in the entire muscle contracting to its full extent (King, 2013).

Figure 2.3: Neuromuscular Junction of a Single Motor Neuron to a Muscle Fiber
(Forsman, 2013)
Muscle action begins at the motor end plate or the neuromuscular junction seen in Figure 2.4. This is where the motor axon terminal releases a transmitter called acetylcholine into the synaptic cleft between the axon and muscle membranes. Acetylcholine then binds to receptors localized in the muscle membrane at the motor end plate (King, 2013). This causes the sarcolemma to have an increased permeability to sodium ions thereby facilitating depolarisation (Hammer, 1999). The resulting local depolarization at the end plate normally exceeds threshold and triggers a depolarizing action potential. This muscle action potential moves toward both ends of the muscle fiber, activating the entire length of the fiber by a single axon terminal (King, 2013). The depolarisation is transferred deep into the muscle fibers by the transverse T-tubules. This results in the release of calcium ions from the sarcoplasmic reticulum leading to an eventual contraction (Hammer, 1999).

![Neuromuscular Junction](image)

**Figure 2.4: Neuromuscular Junction (Sturgis, 2013)**

c. **Blood supply**

Skeletal muscles receive approximately 20% of the cardiac output at rest. As the contraction process requires large amounts of energy in order to replenish the hydrolysed
ATP, skeletal muscles are well-supplied with blood (Klabunde, 2012). The vascular supply to skeletal muscle is provided by primary arteries. These primary arteries are distributed along the long axis of the muscle and give rise to feed arteries that course toward the epimysium of the muscle at right or oblique angles to the primary arteries. These terminal branches further give rise to capillaries that are embedded in the endomysium. Several capillaries surround each muscle fiber and are arranged variably around each fiber. This non-uniform distribution of capillaries around myofibrils and the difference in circumference of each muscle fiber indicates that oxygen is non-homogeneously distributed to skeletal muscles (Korthuis, 2011).

2.3 Relevant Anatomy of the Rhomboid Major and Minor Muscles

2.3.1 General anatomy

The rhomboid major and minor muscles form part of the superficial layer of the extrinsic back muscles, also called the deep posterior axio-appendicular or extrinsic shoulder muscles. The levator scapulae muscle is also included in this group. The superficial posterior axio-appendicular muscles are composed of the trapezius and latissimus dorsi muscles. Together these two groups of muscles serve to provide a direct attachment of the appendicular skeleton to the axial skeleton. This allows these muscles to produce and control upper limb motion. The rhomboid muscles lie deep to the trapezius muscle and form an oblique equilateral parallelogram with its fibers running in an inferolateral direction, as seen in Figure 2.5, from the vertebrae to the scapula (Moore and Dalley, 2006).

2.3.2 Origin and insertion sites of rhomboid major

The rhomboid major muscle is thinner, flatter and wider when compared to the superior rhomboid minor muscle. Rhomboid major originates from the spinous processes of T2-T5 vertebrae while inserting into the medial border of the scapula from the level of the spine to the inferior angle (Moore and Dalley, 2006).
2.3.3 Origin and insertion sites of rhomboid minor

Rhomboid minor originates from the nuchal ligament and spinous processes of C7 and T1 vertebrae while inserting into the smooth triangular area medial to the scapular spine (Moore and Dalley, 2006).

2.3.4 Muscle function

The rhomboid muscles function to retract and rotate the scapula causing the depression of the glenoid cavity. They hold the scapula against the thoracic wall by assisting the serratus anterior muscle (Moore and Dalley, 2006).

2.3.5 Innervation

The rhomboid muscles are innervated by the dorsal scapular nerve (C4, C5) (Moore and Dalley, 2006).
2.3.6 Blood supply

The rhomboid muscles receive their blood supply from the dorsal scapular artery, a deep branch of the transverse cervical artery (Moore and Dalley, 2006).

2.4 Myofascial Trigger Points

A myofascial trigger point is a hyperirritable focal point within a taut band of skeletal muscle fibres or its fascia as seen in Figure 2.6. Myofascial trigger points refer pain in a symptomatic pattern that may evoke autonomic phenomena that are not only specific to that muscle but to the trigger point as well (Travell et al., 1999). Myofascial trigger points refer pain at rest or during motion which can be mediated by a local twitch response (Hammer, 1999).

Hypersensitive trigger points found in muscle bellies, their tendinous junctions and attachments refer a pain that is a deep and intense muscular pain. Trigger points are palpable as hard indurated nodules within the muscle or facia that are both painful and symptomatic with direct pressure. Myofascial pain is referred in a constant manner with varying severity that does not conform to normal dermatomal, myotomal or sclerotomal patterns (Rachlin, 1994).

Trigger points may be found in various structures such as skeletal muscle, their fascia and their tendons, the capsules and ligaments of joints, the periosteum as well as the skin. Trigger points in the above mentioned tissues occur in much the same sites in everybody (Baldry, 1993). Autonomic phenomenon that is caused by myofascial trigger points include localised vasoconstriction, sweating and pilomotor activity that occur in the pain referral pattern of the involved trigger point (Travell et al., 1999).

Myofascial pain is a common dysfunction that has a lifetime prevalence affecting up to 85% of the general population (Fleckenstein, Ruger, Lehmeyer, Freiberg, Lang and Imich, 2010). MFTP’s are more likely to affect those that are in their mature years with maximum activity levels. MFTP’s are painful but are in no way life threatening however, they can and often alter ones quality of life (Travell et al., 1999).
2.4.1 The mechanism of myofascial trigger point development

The formation of myofascial trigger points can be explained by two separate theories. The energy crisis theory seen in Figure 2.7 postulates that any increased demand on a muscle as with an acute macrotrauma or a chronic recurrent microtrauma may lead to the disruption of the sarcoplasmic reticulum with the resultant release of stored calcium (Hong and Simmons, 1998). This disruption also renders the sarcoplasmic reticulum unable to remove calcium from the injured area (Baldry, 1993). Myofibrils will then initiate and maintain a sustained contraction as a result of the excess calcium in the presence of normal adenosine triphosphate (ATP) leading to fatigue of the muscle. The palpable taut band that is associated with myofascial trigger points occurs as a result of this sustained intramuscular contraction which creates a region of uncontrolled metabolism. In order for muscle fibers to return to their resting state, energy from the splitting of ATP is needed to
‘recck’ the contractile mechanism of the sarcomere. With the depletion of the ATP a progressive failure to relax occurs (Gatterman, 1990).

It is this sustained muscle contraction that reduces blood flow to the area. The vasoconstriction associated with the autonomic system also contributes to the decreased blood flow, which is activated by the trigger point sensory fiber input to the central nervous system (Gatterman, 1990). This decrease in local blood supply leads to the release of substances that sensitize muscle nociceptors thereby causing pain. These substances are histamine, released from mast cells, prostaglandins synthesised from endothelial cells and bradykinin from plasma cells (Kostopoulos and Rizopoulos, 2001). This region of increased metabolism and decreased circulation causes the muscle fibers to become shortened (Travell et al., 1999).

Figure 2.7: Schematic diagram illustrating the energy crisis hypothesis causing and perpetuating myofascial trigger points (Travell et al., 1999)

The motor end plate hypothesis is thought to also contribute to some degree of muscle shortening. The motor nerve synapses with a muscle cell at the motor endplate as seen in Figure 2.8. Needle EMG studies have found that each trigger point contains minute loci that produce characteristic electrical activity (Hubbard and Berkhoff, 1993). This activity is
thought to represent an increased rate of release of acetylcholine from the nerve terminal. This small amount of activity at the motor end plate is not enough to cause a muscle contraction however, it may be enough to activate a few contractile elements and therefore contribute to muscle shortening (Simons, 1996). These painful local muscle conditions are self-perpetuating and result in a decreased range of motion and general disability due to decreased flexibility and the resistance to stretch (Gatterman, 1990).

Figure 2.8: Diagram illustrating the integrated hypothesis causing the myofascial trigger point formation (Travell et al., 1999)

2.4.2 Classification of myofascial trigger points

Myofascial trigger points can be classified as being either active or latent based on their clinical characteristics. Active trigger points cause pain at rest, are tender to palpation, and refer a radiating pain to a site that is distant from the trigger point. Active trigger points prevent full lengthening of the involved muscle thereby restricting range of motion and causing pain and weakness (Travell et al., 1999). The stiffness associated with active trigger points is most marked following periods of inactivity (Gatterman, 1990). It is this referral of pain that distinguishes trigger points from tender spots that do not refer pain. A
latent trigger point does not cause spontaneous pain however; it may restrict muscle movement or produce weakness (Alvarez and Rockwell, 2002).

Trigger points can further be categorised as primary, secondary and satellite trigger points. A primary trigger point occurs as a result of an acute strain or injury to skeletal muscle or a more chronic overload resulting in repetitive microtrauma (Travell et al., 1999). Primary trigger points occur independently and are not activated by other trigger point activity in other muscles (Rachlin, 1994). Muscles with primary trigger points become hypersensitive, shortened and weak. As a result of this, antagonist and neighbouring muscles undergo a chronic over loading due to a protective spasm. This protective spasm is maintained to decrease strain on the first affected muscle. Secondary trigger points are found within these muscles. Satellite trigger points are found in muscles that lie in the pain referral area as other myofascial trigger points (Travell et al., 1999).

### 2.4.3 Trigger point examination

It is vital to rule out any psychosocial factors or red flags suggesting fractures and neurological deficits, malignancies or infections before diagnosing a patient with myofascial pain syndrome. Palpation of the muscle is the method of diagnosis. It is necessary for the muscle to be placed on a moderate stretch to cause some discomfort but no pain. The taut bands will feel much like tight cords compared to the soft slackened fibers of the rest of the muscle. Trigger points are palpated along these taut bands (Travell et al., 1999).

Electromyography identifies trigger points by recording a continuous low amplitude action potential that is interrupted by high voltage spikes of electromyographic activity. This is not the case at other non-tender sites (Hong and Simons, 1998). A study done by Bron, Franssen, Wensing and Oostendorp (2007) to determine the interrater reliability of palpation of myofascial trigger points in three shoulder muscles supports the idea that palpation is a reliable method in diagnosing trigger points. Therefore, the palpatory diagnosis was used for the purposes of this research.

#### a. Types of palpation:

i. Flat palpation is accomplished by moving the finger tips across the muscle at right angles to the muscle fibers seen in Figure 2.9 (Anderson, 1997). The mobility of
the subcutaneous tissue is used to slide across the muscle in order to detect any trigger points. This method is particularly used when the muscle can be pressed against underlying bone (Travell et al., 1999).

**Figure 2.9: Cross-sectional schematic drawing of flat palpation to localise the taut band (black oval) and fix the trigger point (red circle) (Travell et al., 1999)**

ii. Pincer palpation is achieved by grasping and rolling groups of muscle fibers, seen in Figure 2.10, in a back and forth motion between the thumb and the index finger in order to identify trigger points (Travell et al., 1999).

iii. Snapping palpation is similar to plucking a guitar string. The examiners fingertips slide vigorously across the muscle fibers in order to feel the cord like structures within the muscle (Travell et al., 1999).
Figure 2.10: Cross-sectional schematic drawing showing pincer palpation of a taut band (black ring) and trigger point (red circle) (Travell et al., 1999)

For the purposes of this study, flat palpation was used as the rhomboid muscles can be palpated over the underlying rib cage with relative ease.

b. Trigger point diagnosis

As described by Travell and Simons (1999) in order to diagnose an active myofascial trigger point, one looks for:

i. A history of a sudden onset during or shortly following acute overload stress, or a history of a gradual onset with chronic overload of the affected muscle;
ii. Characteristic patterns of pain that are referred from myofascial trigger points, patterns that are specific to individual muscles;

iii. Weakness and restriction in the stretch range of motion of the affected muscle;

iv. A taut, palpable band in the affected muscle;

v. Exquisite, focal tenderness to digital pressure in the band of taut muscle fibers;

vi. A local twitch response elicited through snapping palpation or needling of the tender spot;

vii. The reproduction of the patient's pain complaint by pressure on, or needling of, the tender spot;

viii. The elimination of symptoms by therapy directed specifically to the affected muscles.

Finding a site of local tenderness is essential to the diagnosis but non-specific. A local twitch response and pain reproduction, when present, are specific and strongly diagnostic of a myofascial trigger point. All individuals will not have all the criteria (Travell et al., 1999).

2.4.4 Treatment of myofascial trigger points

Treatment of myofascial trigger points often includes eliminating the cause of the trigger point activation as well as correcting any perpetuating factors. Treatment includes a wide range of non-invasive and invasive methods (Travell et al., 1999).

a. Non-invasive methods

For the purposes of this research other treatment methods similar to that of ischaemic compression will be noted. These include the NIMMO method, also known as the Receptor Tonus Technique (Laws, 2005), the Muscle Energy Technique and the Strain Counter-stain method (Nagrale, Glynn, Joshi and Ramteke, 2010).

i. The theory that muscular imbalances manifest from spinal misalignments and that spinal joints were ‘fixed’ or hypomobile rather than misaligned was founded by Nimmo in 1957 (Cohen and Gibbons, 1998). Therefore, hypomobile spinal joints
occurred as a result of hypertonic muscles that prevent the joints from moving freely in their normal ranges of motion. Nimmo states that it is more logical to treat the primary muscular dysfunction rather than the secondary misaligned or hypomobile spinal joints (Cohen and Schneider, 1990).

The Nimmo technique, also known as the Receptor Tonus Technique, is based on the inter-relationship of muscle tonus and the central nervous system as trigger points are viewed by Nimmo as an abnormal reflex arc or a segmental neuropathy. A normal reflex arc consists of weak afferent stimuli from muscle receptors. These afferent nerves synapse in the internuncial pool of the spinal cord. Following this they move along a pathway directly back to the muscle via a motor efferent nerve that alters the tonus of the involved muscle. If the muscle becomes irritated from trauma, a chronic contraction, histamine, serotonin, bradykinin or any other irritant the afferent nerve endings will be sensitized. The signal by the afferent input is intensified resulting in a more sustained output in the form of an increased muscle tonus. According to Nimmo, only the appropriate therapy can break this cycle of increased input and increased output hence the name Receptor Tonus Technique. Nimmo’s method involves manual pressure of the trigger point for 5 to 7 seconds with a quick release of the pressure. The pressure applied should be enough to reproduce the patient’s pain. Following this it is further pressed on 2 to 3 times while reassessing for any change in muscle tone (Cohen and Schneider, 1990).

ii. Muscle Energy Technique is a common conservative treatment in the form of a manual therapy for the release of restricted motion of the spine and extremities (Selkow, Grindstaff and Saliba, 2009). In 1948, Mitchell described that the early principles of Muscle Energy Technique could be incorporated into a manual medicine which could be applied to any body region or articulation. Muscle energy involves a voluntary contraction by the patient in a precisely controlled direction at varying levels of intensity. Muscle energy is used to lengthen a shortened, contractured or a spastic muscle. It also serves to strengthen a physiologically weakened muscle by reducing local oedema, mobilising restricted articulations and trigger points (Grubb et al., 2010).
Muscle Energy Technique is said to have an effect on range of motion by numerous mechanisms. Two most common mechanisms are autogenic inhibition and reciprocal inhibition. The method of Muscle Energy Technique involves a stretch of the muscle up to a point of a resistance barrier. This barrier occurs when both the patient and the practitioner can feel an increase in the resistance of the tissue to further elongate. The patient then contracts the muscle to about 25% of their own muscle force while the practitioner resists this contraction therefore, the body part remains in a static position resulting in an isometric contraction. After 10 seconds the patient is asked to relax. This is then followed by a further stretch in order to find a new barrier position. This process is repeated two to three times (Johnson, 2012).

Reciprocal inhibition occurs when there is a contraction of the agonist muscle with the simultaneous inhibition of the antagonist muscle resulting in a stretch of the antagonist muscle. This inhibition is accomplished by the excitation of the inhibitory interneuron in the spinal cord. Autogenic inhibition is thought to be a protective mechanism. When golgi tendon organs in a muscle become activated due to excessive tension build-up in the muscle there is a cessation of the muscle contraction as a result of the inhibition of the alpha motor neuron innervating that same muscle (Knierim, 1997).

iii. The Strain Counter-strain method is also known as positional release and is a gentle technique that serves to alleviate spinal or other joint pain by the passive shortening of the affected muscle. It is necessary for the practitioner to first palpate and identify any trigger points or tender points on the patient’s body. Once identified the practitioner places the affected muscle in a passively shortened position for about 90 seconds thereby returning the muscle to its original resting position and length (Knierim, 1997).

iv. Ischaemic compression is the treatment of choice for this study and is achieved by the application of a sustained pressure on an active trigger point for a period of 30
to 60 seconds. It is important for the muscle to be stretched to the point of discomfort. The pressure that is applied with the examiner’s thumb or finger temporarily occludes the blood supply to the area resulting in a reactive hyperaemia when the pressure is released. It is important to stretch the involved muscle following treatment. This sudden increase in blood flow serves to flush out inflammatory exudates and pain metabolites from the muscle. Ischaemic compression is useful in the breakdown of scar tissue and decreasing muscle tone. The increased flow of blood also nourishes the muscle and desensitizes nerve endings allowing greater movement of the involved muscle (Hunter, 1998).

There are three local effects following ischaemic compression which describe the mechanism of ischaemic compression. These include the stimulation of local metabolism, release of vasoactive substances and reflex vasodilation (Turchaninov, 2001).

- **Stimulation of local metabolism:**

  An active myofascial trigger point is the epicentre of a local muscle spasm rendering it a leading factor in the formation of a local ischaemia. This local ischaemia results in an insufficient arterial supply and an insufficient venous and lymph drainage. When compression is applied during treatment to the point of the patient’s pain threshold a mild trauma to the capillary bed is ensued in the trigger point area. As a result of this, micro haemorrhages occur and blood cells escape into the surrounding soft tissue. These free blood cells in the surrounding tissue elicit an immediate immune response in the form of macrophage and T cell release. These killer cells eliminate and dispose of these blood cells. As this process occurs, and as a result of this process there is a significant increase in the local metabolism of the trigger point area (Turchaninov, 2001).

- **Release of vasoactive substances:**

  The mild local trauma that occurs as a result of direct pressure leads to the release of vasoactive substances such as histamine from the involved tissue. Histamine is known to be a potent vasodilator and signals the release of fibroblasts. Fibroblasts function to repair damaged tissue by the production of collagen (Turchaninov, 2001).
• Reflex vasodilation:

The arterial perfusion to the trigger point area is significantly lower than the area around the trigger point. Minimal oxygenation is still maintained in the trigger point but blood supply is not sufficient enough to allow effective contraction. The blood flow around the area of the trigger point remains normally perfused. With ischaemic compression a condition of local hypoxia is created. As blood continues to be pumped by the heart there is a build-up of blood around the area of compression. Once the pressure is released the nervous system initiates a reflex vasodilation of the constricted capillaries in the area to restore normal oxygenation using this build-up of blood around the area (Turchaninov, 2001).

b. Invasive methods

Myofascial trigger points can be relieved with injection of a local anaesthetic or a saline solution. A similar relieving effect can also be achieved with myofascial dry needling which is considered to be a safer technique as the potential side effects associated with anaesthetics are avoided. These side effects range from anaphylactic shock to palpitations, fainting and cardiac arrest (Baldry, 1993).

The mechanism of dry needling includes the mechanical disruption of the muscle fibers and nerve endings resulting in an increase in extracellular potassium levels. This causes the depolarisation of nerve fibers and interrupts the positive feedback mechanism that perpetuates the cycle of pain. Nerve sensitizing substances are also removed by the resulting local haemorrhage and vasodilation (Yap, 2007). Endogenous opioids are released during dry needling and activate the descending pain inhibitory system resulting in pain relief. A decreased sensation of pain is also experienced as dry needling causes the closing of the pain gate-like mechanism as large diameter A-beta fibers are activated (Baldry, 2001).
2.4.5 Perpetuating factors

There are several factors that perpetuate the formation of myofascial trigger points. They include acute trauma, repetitive microtrauma, lack of exercise, prolonged poor posture, vitamin deficiencies, sleep disturbances as well as occupational or recreational activity that places any repetitive stress on a muscle (Alvares and Rockwell, 2002). Predisposing factors include degeneration of bones and joints, nerve root compression, emotional and psychological stress, endocrine and metabolic deficiencies, chronic infection and lastly chronic muscle imbalances (Ching, 2007).

2.4.6 Myofascial trigger points in the rhomboid major and minor muscles

Myofascial trigger points in the rhomboid muscles refer pain medially along the vertebral border of the scapula as well as the area between that border and the vertebral column as seen in Figure 2.11. The pain may also spread over the supraspinous portion. Trigger points may be located in the mid-belly of the muscles and along the vertebral border of the scapular. The patient will complain of a superficial aching pain occurring at rest that is unchanged by normal movement. On examination there is no restriction in range of motion of the arm (Travell et al., 1999).

2.4.7 Activating and perpetuating trigger points in rhomboid major and minor muscles

Myofascial trigger points of the rhomboid muscles become activated after prolonged periods with arm held out in abduction or flexion above 90°. Examples of this include overhead work or painting. Trigger points are further activated and perpetuated with leaning forward with a round shouldered position for long periods of time or by a sustained tension caused by a shortened pectoralis major muscle which occurs when working on one’s computer. A prolonged stretch due to the prominence of the scapula on the convex side in upper thoracic scoliosis due to idiopathic scoliosis, chest surgery, or a limb-length inequality can also activate and perpetuate rhomboid muscle trigger points (Travell et al., 1999).
Figure 2.11: Composite referred pain pattern caused by mid-muscle trigger points and trigger areas (enthesopathy) of the right rhomboid muscles (Travell et al., 1999).

2.4.8 Palpation of the rhomboid major and minor muscles

Myofascial trigger points in the rhomboid muscles are palpated using flat palpation with the patient in a seated or supine position. The patients’ arms should hang forward which allows for the relaxation of the rhomboid muscles as well as the abduction of the scapulae. This scapulae abduction causes the scapulae to spread away from the vertebral column. Myofascial trigger points in the rhomboid muscles are distinguished from those in the overlying trapezius muscle due to the direction of its fibers. The fibers run in an obliquely downward and lateral direction away from the vertebral column. Trigger points are palpated for in the mid-belly of the rhomboid muscle as well as along the medial and vertebral border of the scapulae. A local twitch response is not easily elicited in the rhomboid muscles (Travell et al., 1999).
2.5 Pain

Pain is a complex and unpleasant phenomenon that is composed of sensory experiences including time, space, intensity, emotion, cognition and motivation. It is an emotional experience that may originate from real or potentially damaged tissue and is uniquely experienced by each individual (Hanacek, 2004).

Pain production consists of the interaction of three systems (Hanacek, 2004):

- The Sensory-discriminative system processes information about the strength, intensity, quality and temporal and spatial aspects of pain;
- The Motivational-affective system determines the individual’s approach or avoidance behaviours to pain;
- The Cognitive-evaluative system includes the individual’s learned behaviour concerning the experience of pain thus blocking, modulating or enhancing the perception of pain.

2.5.1 Pain threshold and pain tolerance

Pain threshold is the point at which a stimulus is perceived as pain and remains relatively the same among healthy individuals or in the same person. Pain tolerance is defined as the amount of time that the intensity of the pain felt is willingly endured by the subject before initiating an overt pain response and is influenced by several conditions including physical and mental health, expectations, role behaviours and cultural background. Pain tolerance decreases with repeated exposure to pain, fatigue, anger, boredom, apprehension, sleep deprivation and is increased with alcohol consumption, medication, hypnosis, warmth, distraction or strong beliefs and faith and varies greatly among people as well as in the same person over time. Pain tolerance is decreased in the elderly possibly due to peripheral neuropathies and changes in skin thickness with aging (Hanacek, 2004) while males appear to be more tolerant to pain than females (Woodrow, Friedman, Siegelaub and Collen, 1972).
2.5.2 Neuroanatomy of pain

Sensation and perception of pain is accomplished by the combined activity of afferent pathways, the central nervous system and efferent pathways. Afferent pathways are composed of nociceptors also known as pain receptors, afferent nerves and the spinal cord. Afferent pathways terminate in the dorsal horn of the spinal cord (1st order afferent neuron). The spinal component of the afferent system is made up of 2nd order neurons. Pain signals are interpreted in the central nervous system specifically the limbic system, reticular formation, thalamus, hypothalamus and the cortex. Fibers connecting the reticular formation, midbrain and substantia gelatinosa are composed of the efferent pathway. These components are responsible for modulating pain sensation. The thalamus and cortex perceive, describe and localise pain. Dull long-lasting pain and diffuse pain are identified by parts of the thalamus, brainstem and reticular formation. Emotional and affective responses to pain are controlled by the reticular formation and the limbic system. Pain perception is associated with an autonomic response as the cortex; thalamus and brainstem are all interconnected with the hypothalamus and the autonomic nervous system (Hanacek, 2004).

Nociceptors are small unmyelinated or lightly myelinated afferent neurons that can be stimulated by chemical, mechanical or thermal noxae. They are located in the epidermis, subcutaneous tissue, visceral organs and muscle tendons and are not evenly distributed among these areas (Hanacek, 2004).

Afferent pathways travel from nociceptors to neurons in the dorsal horn of the spinal cord by small A-delta and C fibers. Spinothalamic tracts transmit input from the dorsal horn to the higher parts of the spinal cord and the central nervous system. Small unmyelinated C fibers transmit sensations of diffuse burning or aching pain while larger myelinated A-delta fibers carry well-localised sharp pain sensations (Hanacek, 2004).

2.5.3 Pain modulation

The pain gate theory developed by Melzack and Wall in 1967 as depicted in Figure 2.12, has profoundly influenced how the sensation of pain may be modulated by numerous physiochemical mechanisms in the central nervous system. This theory states that each
dorsal horn of the spinal cord possesses a gate-like mechanism that can either inhibit or facilitate the flow of afferent or incoming input into the spinal cord. The relative activity in large diameter A-beta and small diameter C and A-delta fibers determine the opening or closing of this gate. The activation of large diameter fibers (non-painful input) allows for the closure of the pain gate mechanism resulting in pain relief. The activation of small diameter fibers allows for the opening of the gate-like mechanism which then facilitates the sensation of pain (Baldry, 1993).

![Figure 2.12: Gate control theory of pain illustrating (a) unmodulated pain and (b) modulation of pain (Fitzakerley, 2013)](image)

Painful afferent impulses are further suppressed in the spinal cord by the excitation of inhibitory interneurons by collateral branches. These collateral branches are given off by large diameter afferent nerves from low threshold mechanoreceptors. The excitation of these collateral branches results in pain relief. Therefore, it can be noted that the
stimulation of low threshold mechanoreceptors in the area of pain can cause pain relief (Anderson, 1997).

Another aspect of the pain gate theory is the influence of the descending pain inhibitory system on the opening and closing of this gate mechanism. This analgesia system is a pain control system that is activated by the brain and can control the input of pain signals into the spinal cord. This system has three components: the peri-aquedectal grey matter located in the mesencephalon and the upper pons, the raphe magnus nuclei located in the lower pons and upper medulla and the dorsolateral columns. Nerves from the peri-aquedectal grey matter project onto the raphe magnus nuclei and are transmitted along the dorsolateral columns in the spinal cord. Pain can be blocked at this point thereby preventing it from reaching the brain (Guyton, 1991).

Enkephalins and serotonin are transmitter substances that are also involved in the endogenous system. Serotonin initiates the release of enkephalins from spinal cord neurons. Enkephalins are thought to cause pre-synaptic inhibition of both C and A-delta fibers at their synapsis in the dorsal horn. The descending analgesic system appears to have a long lasting pain relief effect (Guyton, 1991).

2.5.4 Importance of pain management during treatment

Pain that is inadequately managed during any form of treatment can have a number of adverse psychological and physical effects on individual patients. Psychological effects associated with poor management of pain include anxiety and depression, which could further predispose the patient to other health problems (Wells, Pasero and McCaffery, 2008).

Pain is a one of the major precursors of stress which has the ability to affect multiple systemic systems. The endocrine system reacts to the stress of pain by the excessive release of hormones resulting in the abnormal catabolism of carbohydrates, proteins and fats. The cardiovascular system responds to the stress of pain by activating the sympathetic nervous system which increases both heart rate and blood pressure. The immune system is also affected by the stress that occurs with pain by its suppression causing a decrease in the systems overall function. Ultimately the under-management of
pain has the ability to prolong the stress response which adversely affects the patient’s recovery (Pasero et al., 1999). It is for these reasons that reducing pain during treatment could possibly have a more positive outcome for the patient. Gordon, Dahl, Miaskowski, McCarberg, Todd, Paice, Lipman, Bookbinder, Sanders, Turk and Carr (2005) state that pain should be prevented and controlled to such a degree that facilitates the function and quality of life.

2.6 Pressure Mechanoreceptors

Mechanoreceptors are specialised cells that provide information to the central nervous system about touch, pressure, vibration and cutaneous tension. There are four major types of encapsulated mechanoreceptors that are located in the skin seen in Figure 2.13: Pacinian corpuscles, Merkel’s discs, Ruffini endings and Meissner’s corpuscles. These receptors are all referred to collectively as low-threshold or high-sensitivity as even a weak mechanical stimulation of the skin is able to elicit an action potential (Purves, Augustine, Fitzpatrick, Katz, La Mantia, Mc Namara and Williams, 2001). Mechanoreceptors such as Pacinian corpuscles and Merkel’s discs are innervated by large diameter nerve fibers (Brown, Bolton and Aminoff, 2002). For the purposes of this study, the Pacinian corpuscle and Merkel’s discs will be discussed further as these two receptors respond to pressure.

Figure 2.13: Skin receptors (Mason, 2014)
2.6.1 The Merkel’s disc

Merkel’s discs are mechanoreceptors found in the deepest layer of the epidermis. These receptors consist of two components which are seen in Figure 2.14: a specialised Merkel’s disc and a nerve ending (Holdcroft and Jagger, 2008). The Merkel’s disc is made from flattened, non-neural epithelial cells (Iheanacho and Olubiyi, 2007). The free nerve ending loses its myelination as it penetrates the epidermis layer (Holdcroft and Jagger, 2008) and innervates the Merkel’s disc via sensory terminals (Zelena, 1994). The Merkel's disc together with the free nerve ending, form a junction that is similar to that of a synapse (Holdcroft and Jagger, 2008). Merkel’s discs respond to light touch and superficial pressure (Iheanacho and Olubiyi, 2007).

With the application of pressure there is an initial burst of impulses released from the Merkel’s discs. This is followed by a short period of adaptation during which the frequency of impulses is decreased. A phase that is characterised by an irregular pattern of discharges occurs next with variable spikes in action potentials. This phase continues for as long as the pressure is maintained. It seems that the sensory terminals are responsible for the conduction of impulses in Merkel’s discs by the action of modulating dense core vesicles which contain a number of bioactive substances (Zelena, 1994).
2.6.2 The Pacinian corpuscle

The Pacinian corpuscle is a specialised mechanoreceptor that perceives deep pressure. Pacinian corpuscles are located in the subcutaneous tissue beneath the dermis of the skin. They are ovoid in shape and are encapsulated. Pacinian corpuscles consist of concentric layers of connective tissue and a nerve terminal that is located in the centre of the organelle seen in Figure 2.15 (Boron and Boulpaep, 2011).

![Cross-section of a Pacinian corpuscle](image)

**Figure 2.15: Cross-section of a Pacinian corpuscle (Stark, Carlstedt, Hallin, Risling, 1998)**

With the application of pressure to the overlying skin, the Pacinian corpuscle is compressed causing the transfer of energy to the nerve terminal. The compression also causes the deformation of the Pacinian corpuscle membrane leading to the opening of mechanosensitive channels. If a large enough depolarising receptor potential is generated from the current flowing through these channels, an action potential is fired by the axon (Boron and Boulpaep, 2011). The generated potential within the sensory neuron is a graded response therefore the greater the deformation, the greater the generator potential. Once the generator potential threshold is reached a flood of action potentials are triggered along the sensory neuron. Based on the above the more massive or rapid the deformation of the Pacinian corpuscle the higher the frequency of the nerve impulses generated in its' neuron (Kimball, 2011).
With the application of a steady pressure, the slick layers of the Pacinian corpuscle that contain a viscous fluid between them slip past one another. This slippage transfers the stimulus energy away so that the underlying axon terminal is no longer deformed and the receptor potential dissipates. The release of pressure causes the above mentioned events to be reversed and the depolarized state of the terminal is restored (Boron and Boulpaep, 2011).

2.6.3 Adaptation characteristics of pressure mechanoreceptors

The characteristics of the tissues surrounding the different types of cutaneous receptors in the body determines the sensitivity of the receptors to different patterns of pressure or changes in pressure (Sherwood, 2010). The surrounding tissues or capsules of mechanoreceptors also determine the adaptation characteristics of each type of mechanoreceptor (Kimball, 2011).

There are two types of cutaneous mechanoreceptors based on their speed of adaptation. These are tonic (slow adapting) and phasic (rapid adapting) receptors. Tonic receptors are slow to adapt to stimuli as the receptor continues to respond to the stimulus for as long as the stimulus is sustained. Merkel’s discs are examples of tonic receptors and continue to respond to light pressure for as long as the pressure is applied, within a reasonable time frame (Iheanacho and Olubiyi, 2007).

Phasic receptors are rapidly adapting receptors that do not respond to a sustained stimulus but do respond to changes in the stimulus. Pacinian corpuscles are examples of phasic receptors that do not respond to a steady deep pressure due to their ability to adapt to stimuli (Iheanacho and Olubiyi, 2007). Adaptation serves to prevent the nervous system from being subjected to an overload of information that is of an insignificant nature (Kimball, 2011).

2.7 Conclusion

This chapter served to review previous literature that was deemed pertinent to this study. The relevant anatomy of skeletal muscle specifically the rhomboid muscles, the
development of myofascial trigger points, ischaemic compression, pressure mechanoreceptors and aspects of pain were all emphasised upon.

As mentioned previously in 1.1, ischaemic compression has been proved to be a safe and effective technique and is known for successfully decreasing myofascial trigger point activity (De Las Peñas et al., 2006). However, there is little consensus to the best treatment protocol according to Vernon and Schneider (2009). Gulick (2010) states that there is a lack of randomised controlled studies with regard to standard ischaemic compression treatment protocols. With regard to treatment with ischaemic compression, current treatment protocols include the application of pressure that reaches the patient's maximum pain tolerance levels (Hains, Descarreaux, Lamy and Hains, 2010).

One could assume that treatment at the maximum level of pain tolerance would result in increased patient discomfort and pain. As discussed in 2.5.4, pain that occurs during treatment could cause adverse effects (Wells et al., 2008). Therefore, it is necessary to determine if treatment at pain threshold would yield the same effect as treatment at pain tolerance which could ultimately have a more positive effect on the outcome of treatment.

Chapter three will provide a detailed explanation of the methods in which this study was undertaken.
CHAPTER THREE – METHODOLOGY

3.1 Introduction

This chapter serves to elaborate on the construction of and procedures involved in carrying out this study.

3.2 Study Design

The design of this study was comparative in nature as the efficacy of two treatment regimens was compared. Random stratified sampling was used to allow for an equal proportion of random males and females with similar ages in each group.

3.3 Participant Recruitment

Any participant who presented with active rhomboid myofascial trigger points at the University of Johannesburg Chiropractic Day Clinic was considered as a potential candidate for the study. Thirty participants were recruited via word of mouth and posters placed around the University of Johannesburg Doornfontein campus and clinic (Appendix A). The posters invited possible participants with aching or burning pain between the shoulder blades that are between the ages of 18 and 40 years to participate in the study.

3.4 Sample Selection and Size

The study was explained to each participant and they were required to read the Information form (Appendix B) and sign the Consent form (Appendix C) specific to this study. A screening process was then conducted to determine whether each participant was eligible for this study by taking into consideration the Inclusion and Exclusion criteria. Participants had to present with active rhomboid myofascial trigger points.

3.4.1 Inclusion criteria

To be included in this study, participants needed to comply with the following criteria:

- Participants between the ages of 18 and 40 years old with active myofascial trigger points of the rhomboid muscles. The thirty participants aged between 18 and 40
years were divided randomly into two groups of fifteen each based on age and
gender as it has been shown that pain tolerance decreases with age and males
tolerate pain more than females (Woodrow et al., 1972). According to the Centers
for Disease Control and Prevention, spinal degeneration greatly increases after the
age of 40 therefore, no participant over the age of 40 was recruited (Centers for
Disease Control and Prevention, 2011).

- Diagnosis of active myofascial trigger points of the rhomboid muscles (Travell et
  al., 1999):
  1. Taut palpable band;
  2. Presence of spot tenderness of a nodule in a taut band;
  3. Presence of referred pain;
  4. Reproduction of patients’ symptomatic pain.

3.4.2 Exclusion criteria

Participants were excluded from the study if they presented with the following:

- Patients that were taking any form of medication that would influence the results of
  the study such as analgesics, muscle relaxants, NSAIDS or steroids (Poul, West,
  Buchanan and Grahame, 1993).
- Participants presenting with a muscle strain or a haematoma of the involved
  muscle (Perle, Schneider and Seaman, 1999).
- Participants using anti-coagulant medication (Perle et al., 1999).
- The following listed by Travell et al., 1999:
  1. Patients with vascular compromise, impingement, disease or
     aneurysm;
  2. Acute rheumatoid arthritis or any other systemic disease;
  3. Chronic diseases such as lupus or cystic fibrosis;
  4. Cancer and malignancy;
  5. Fractures, open wounds, trauma, sprain, strain and bruises.
  6. Any condition associated with fever, nausea, weight loss and
     neurologic problems;
  7. Severe acute or chronic pain or acute inflammation.
3.5 Random Group Allocation

The participants were randomly divided into two groups based on age and gender. Group A received pressure at pain threshold during treatment. Group B received pressure at pain tolerance during treatment. All participants were screened at the initial consult for the amount of pressure needed to elicit the very first point of pain. This pressure was then increased until the participant felt the maximum amount of pain that he or she was able to withstand. These measurements were taken with a hand held pressure algometer and documented.

3.6 Treatment Approach

3.6.1 Initial visit

The initial visit for both groups involved the following:

- The researcher conducted a Case History (Appendix D), a Full Physical Examination (Appendix E) and a Cervical Spine Regional Examination (Appendix F). Following this a Subjective Objective Assessment Plan (SOAP) Form was completed (Appendix G) for each participant.
- Each participant then completed subjective measurements in the form of a Numerical Pain Rating Scale (Appendix H) under the supervision of the researcher.
- Trigger point palpation was then applied on each participant in order to locate the most active myofascial trigger point of either the left or right rhomboid muscles. The participant was positioned in a supine position with their hand placed in the small of their backs. This allowed the scapula to be moved away from the rib cage which elevated the rhomboid muscles as well as placed the muscle in a slackened position. In order to maintain continuity in this research; all participants were positioned supine for the palpation process. Flat palpation was used to locate any spot tenderness and/or elicit a referral of pain (Travell et al., 1999). The most active myofascial trigger point found in either the left or right rhomboid muscles were chosen and used for the remainder of the study.
Objective measurements were taken using a hand held pressure algometer (Appendix I). The algometer tip was placed on the chosen trigger point and pressure was applied to determine the amount of pressure that caused the participants first point of pain (pain threshold) and then the amount of pressure that was most painful for the patient (pain tolerance). The two readings were recorded for all participants on a strip of paper that was placed in a sealed envelope after the 1st, 4th and 7th visits (Appendix I).

Treatment in the form of ischaemic compression was then administered by the application of a sustained pressure on an active trigger point for a period of 30 seconds with the muscle stretched to a point of discomfort (Turchaninov, 2001). The ischaemic compression during each treatment was administered with an algometer to make certain that the same and correct amount of pressure was delivered at all treatments. Participants in Group A received ischaemic compression only at pain threshold while Group B received treatment only at pain tolerance. All treatments were followed by stretching of the rhomboid muscles (Travell et al., 1999).
3.6.2 Follow-up visits

Follow-up visits included the following:

- Subjective measurements (Numerical Pain Rating Scale) were completed by each participant at the 1st, 4th and the 7th final visits (prior to treatment) under the supervision of the researcher. Pressure algometry of the same trigger point found in the 1st visit was undertaken at the 4th and 7th final visits to again determine the changes in pain threshold and pain tolerance of all participants in both groups.

- At each visit, participants were reassessed and a SOAP note was completed to update the current display of pain and symptoms of each participant.

- Also at each visit, Group A received treatment at pain threshold while group B received treatment at pain tolerance.

- Each participant was thus seen seven times in total and received ischaemic compression six times either at pain threshold or pain tolerance over a three week period with a 7th consultation been used only for the recording of measurements. A minimum of 48 hours was maintained between visits to aid in tissue recovery (Travell et al., 1999).
3.7 Subjective Data

3.7.1 Numerical Pain Rating Scale

The participants were subjectively evaluated by completing a Numerical Pain Rating Scale which served to record subjective estimates of pain intensity as well as pain improvement or deterioration. The scale consists of a 10cm horizontal line that is divided up into 10 blocks by short vertical lines. The left end of the line is marked with ‘no pain’ and the right end is marked with ‘severe pain’ (Appendix I). The participants were asked to mark an ‘X’ on the horizontal line indicating the severity of their pain (Maire, 2002).

In a study conducted, the validity and reliability of the Visual Analogue Scale, Numerical Pain Rating Scale, Verbal Rating Scale and the Faces Pain Scale-Revised, in detecting differences in painful stimulus intensity and differences between gender in response to experimentally induced pain, were compared. Subjects underwent various cold pressor trials and were then asked to rate their pain intensity on all four scales. The validity and reliability of the Numerical Pain Rating Scale was shown as it was the most responsive and able to detect gender-based differences in pain intensity in comparison to other more commonly used subjective pain measurement scales (Valente, Alexandra, Pais-Ribeiro and Jensen, 2011).

3.8 Objective Data

3.8.1 Pressure algometer

The objective data for this study consisted of measurements obtained by a hand held algometer. All myofascial trigger points have a pressure pain threshold which can be defined as the minimal amount of pressure or force needed to elicit pain in that area. The pressure algometer was used to measure this sensitivity (Rachlin, 2004). The algometer is a force gauge, spring operated plunger calibrated in kg/cm² fitted with a rubber disc of 1cm² surface. The algometer was placed at a 90 degree angle to the skin overlying the involved rhomboid muscle. Pressure was then administered until the participant indicated the first point of pain as well as point at which pain tolerance is reached. A reading was
taken in kg/cm² with the removal of the algometer and was recorded. Measurements were taken with the algometer at the 1st, 4th and 7th visits.

A study by Ylinen, Nykanen, Kautiainen and Hakkinen (2007) evaluated the intra-tester repeatability and validity of pressure algometry on the neck and shoulder area in women with neck pain. Measurements were taken on the levator scapulae and trapezius muscles as well as the sides of the sternum. It was concluded that at a group level, the reliability and validity of the measurements were good and the repeatability of measurements allow for pressure algometry to be useful in clinical trials.

![Pressure algometer instrument and usage procedure](Photograph by researcher, 2014)

**Figure 3.3: Photograph illustrating the pressure algometer instrument and usage procedure (Photograph by researcher, 2014)**

### 3.9 Data Analysis

Frequencies and Descriptives were the first tests done to ascertain an overall interpretation of all the data. The Crosstab test was then done to assess the average number of males and females in each group. The Shapiro-Wilk test was done to test for normality. As normality was not shown, the Non-parametric Mann-Whitney U test was done to compare between groups (intergroup). The Friedman test was done to compare any differences within the groups (intragroup) over time, specifically at the first, fourth and seventh consults. As differences were found, a further test was done to ascertain where these differences had occurred. This test is called the Wilcoxon Signed Ranks test.
3.10 Ethical Considerations

All participants that partook in this particular study read and signed the Information and Consent forms specific to this study. The Information and Consent forms outlined the names of the researcher, purpose of the study, participant assessment, treatment procedure and the benefits of partaking in the study. Any risks, benefits and discomforts that pertained to the treatments involved were explained and that the participant’s safety was ensured (prevention of harm). The Information and Consent forms also explained that the participant’s privacy was protected as only the doctor, participant and clinician were in the treatment room and that anonymity was ensured as the participant information was converted into data and therefore could not be traced back to the individual. The forms also stated that standard doctor/patient confidentiality would be adhered to at all times when compiling the research dissertation. The participants were informed that their participation was on a voluntary basis and that they were free to withdraw from the study at any stage. If the participant had any further questions, they were explained by the researcher; whose contact details were made available. The participants signed the Information and Consent forms, which signified that they understood all that was required of them for this particular study. Results of the study were made available on request.

With regards to this particular study, risks involved with ischaemic compression were minimal. A transient discomfort in the form of tenderness and slight possible bruising may have occurred. Benefits included the relief of pain and muscle spasm as well as local hyperaemia (Hou, Tsai, Cheng, Chung and Hong, 2002). All the necessary precautions were taken to ensure participant safety.

Participants were referred to the appropriate practitioners when necessary.

Results of this study would be made available on request.

3.11 Conclusion

A detailed explanation on the procedures involved in this study was provided in this chapter. The following chapter will indicate the results obtained during this study.
CHAPTER FOUR – RESULTS

4.1 Introduction

This chapter focuses on the presentation of the results obtained from the clinical trials of this study. A sample group of 30 participants all presenting with active rhomboid myofascial trigger points were used. The 30 participants were divided into two groups of 15 each. Group A received ischaemic compression for 30 seconds at pain threshold while Group B received ischaemic compression for 30 seconds at pain tolerance. Each participant received two treatments a week for three weeks while a seventh and final visit served only for measurement taking. No assumptions can be made with respect to the whole population as these results only represent a small group of subjects. The p-value for all tests done was set at 0.05 and represents the level of significance of the results.

Included in the analyses were the following:

- Demographic data analysis consisting of participants’ age distribution and gender.
- Subjective measurements consisting of the Numerical Pain Rating Scale.
- Objective measurements consisting of pressure algometry.

Pertaining to the figures, Group A that received treatment at pain threshold is represented by orange while Group B that received treatment at pain tolerance is represented by blue. This chapter compares the changes in the average results for the subjective and objective measurements for both individual Groups A and B over time. For this, non-parametric tests were performed including the Friedman test to compare results between visits 1 – 4 and visits 1 – 7 for each group. The Bonferonni adjustment was done following each Friedman test where a statistically significant improvement was found, in order to determine where exactly this improvement occurred over time. This was achieved by testing the smallest p-value of each variable against a significant level of 0.05/2 = 0.025. The Wilcoxon Signed Ranks test was used to compare visits 1 – 7 in each group. Non-parametric tests were also done to compare results for subjective and objective test results at each visit between the two groups. To do this, the Mann-Whitney U test was used to determine if there was a statistical significance between the results found in Groups A and B at the 1st, 4th and 7th visits.
### 4.2 Demographic Data Analysis

**Table 4.1: Demographic data of age and gender**

<table>
<thead>
<tr>
<th>Data</th>
<th>Group A</th>
<th>Group B</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age Distribution</td>
<td>22 - 29</td>
<td>22 - 35</td>
<td>22 - 35</td>
</tr>
<tr>
<td>(years)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Age</td>
<td>25.06</td>
<td>25.73</td>
<td>25.40</td>
</tr>
<tr>
<td>(years)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender Distribution</td>
<td>3 Males</td>
<td>4 Males</td>
<td>7 Males</td>
</tr>
<tr>
<td></td>
<td>12 Females</td>
<td>11 Females</td>
<td>23 Females</td>
</tr>
</tbody>
</table>

Candidates recruited for this study were aged between 22 and 35 years old which can be seen in Table 4.1. The combined sample group consisted of 7 male and 23 female participants. Participants allocated to Group A were aged between 22 and 29 years old with a mean age of 25.06 years. There were 3 male and 12 female candidates in Group A. Participants allocated to Group B were aged between 22 and 35 years old with a mean age of 25.73 years. There were 4 male and 11 female candidates in Group B.

The Fisher's Exact test was done for group gender cross-tabulation. It was found that there was an equal number of males and females when comparing both groups with \( p=1.000 \). The Shapiro-Wilks test was done to determine normality of the ages of participants within the two groups. Group A was found to have a non-statistically significant value of \( p=0.228 \) while Group B had a statistically significant value of \( p=0.012 \).
4.3 Subjective Data Analysis

4.3.1 Numerical Pain Rating Scale

Figure 4.1: Bar graph representing Numerical Pain Rating Scale values of both groups at each visit

Figure 4.1 compares the Numerical Pain Rating Scale (NPRS) values for both groups over time. The x-axis is represented by the NPRS results at the 1st, 4th and 7th visits. The y-axis is represented by the improvement seen on a scale of 0.00 to 10.00.

The NPRS scores show that at the 1st consultation, Group A had a mean NPRS value of 5.46 while Group B had a mean NPRS value of 5.86. At the 4th consultation, Group A had a mean NPRS value of 3.46 and Group B had a mean NPRS value of 3.66. At the 7th consultation, Group A had a mean NPRS value of 2.06 while Group B had a mean NPRS value of 1.66.

Based on the above mean NPRS values that were obtained for both groups, a percentage was calculated in order to determine whether an overall improvement or lack thereof had occurred. Group A showed a clinical improvement of 62.27 % and Group B showed a clinical improvement of 71.67 %.
Intragroup analysis

The Friedman test was used to form a comparison, of each group individually, over time. With regards to the NPRS, Group A had a p-value of 0.000 and Group B also had a p-value of 0.000. This indicates that both groups had significant differences in the NPRS scores over time.

The Wilcoxon Signed Ranks test was used to specifically detect where exactly any differences occurred over time within each of the two groups. As a result of this, the test was able to detect differences between the 1st and 4th visits as well as the 1st and 7th visits.

With regards to the NPRS the values demonstrated a statistically significant improvement for Group A for the intervals between the 1st and 4th visits (p=0.001) and between the 1st and 7th visits (p=0.001). The values also demonstrate a statistically significant improvement for Group B for the intervals between the 1st and 4th visits (p=0.001) and for the interval between the 1st and 7th visits (p=0.001). The p-values for both groups indicate that both groups had significant differences which had occurred over both time intervals.

Intergroup analysis

The Mann-Whitney U test was used to form a comparison between the two groups at specific intervals, that is, between Group A and Group B at the 1st, 4th and 7th visits. With regards to the NPRS, the 1st visit had a p-value of 0.421, the 4th visit had a p-value of 0.626 and the 7th visit had a p-value of 0.227. Therefore, no significant differences occurred between the two groups at the specific time intervals.
4.4 Objective Data Analysis

4.4.1 Pressure algometer

a. Pain threshold

Figure 4.2: Bar graph representing pressure algometer values at pain threshold of both groups at each visit

Figure 4.2 compares the pressure algometer readings taken at pain threshold, which was the very first point that the patient indicated they felt pain, for both groups over time. The x-axis is represented by the pressure algometer value at the 1st, 4th, and 7th visits. The y-axis is represented by the percentage improvement on a scale of 0.00 kg/cm² to 10.00 kg/cm².

The pressure algometer results taken at pain threshold showed that at the 1st visit, Group A had a mean algometer value of 2.57 kg/cm² whilst Group B had a mean algometer value of 2.55 kg/cm². At the 4th visit, Group A presented with a mean algometer value of 3.84 kg/cm² where Group B had a mean algometer value of 4.36 kg/cm². Group A had a mean algometer value of 5.32 kg/cm² while Group B had a mean algometer value of 5.82 kg/cm² at the 7th visit.
Based on the mean algometer readings shown above, a percentage was calculated to
determine whether an overall improvement or lack thereof had occurred. Group A showed
a clinical improvement of 107.03 %, whereas Group B showed a clinical improvement of
127.96 %.

**Intragroup analysis**

The Friedman test was used to form a comparison, of each group individually, over time.
With regards to the algometer readings taken at the point of pain threshold between the 1st
and 7th visits, Group A had a p-value of 0.000 and Group B had a p-value of 0.000. This
indicates that both groups had significant differences in the algometer readings over time.

The Wilcoxon Signed Ranks test was used to specifically detect where any differences
occurred over time within each of the two groups. As a result of this, the test was able to
detect differences between the 1st and 4th visits as well as the 1st and 7th visits.

With regards to the algometer readings taken at the point of pain threshold, Group A had a
p-value of 0.003 for the interval between the 1st and 4th visits and a p-value of 0.001 for
the interval between the 1st and 7th visits. Thus both p-values indicate a significant
difference had occurred over both time intervals. Group B had a p-value of 0.001 for the
interval between the 1st and 4th visits and a p-value of 0.001 for the interval between the
1st and 7th visits. Thus both p-values indicate a significant difference had occurred over
both time intervals.

**Intergroup analysis**

The Mann-Whitney U test was used to form a comparison between the two groups at
specific intervals, that is, between Group A and Group B at the 1st, 4th and 7th visits. With
regards to the algometer readings taken at the point of pain threshold, the 1st visit showed
a p-value of 0.421, the 4th visit showed a p-value of 0.626 and 7th visit showed a p-value
of 0.227. Therefore, no significant differences had occurred between the groups at specific
time intervals.
b. Pain tolerance

![Bar graph representing pressure algometer values at pain tolerance of both groups at each visit]

Figure 4.3: Bar graph representing pressure algometer values at pain tolerance of both groups at each visit

Figure 4.3 compares the pressure algometer readings taken at pain tolerance (which occurs when the patient is no longer willing to tolerate an increased pain stimulus) for both groups over time. The x-axis is represented by the pressure algometer value at the 1st, 4th and 7th visits. The y-axis is represented by the percentage improvement on a scale of 0.00 kg/cm$^2$ to 10.00 kg/cm$^2$.

The pressure algometer results show that at the 1st visit, Group A had a mean algometer value of 5.72 kg/cm$^2$ whilst Group B had a mean algometer value of 6.02 kg/cm$^2$. At the 4th visit, Group A had a mean algometer value of 7.54 kg/cm$^2$ whereas Group B had a mean algometer value of 7.52 kg/cm$^2$. At the 7th visit, Group A had a mean algometer value of 8.52 kg/cm$^2$ and Group B had a mean algometer value of 9.79 kg/cm$^2$.

Based on the mean algometer readings shown above, a percentage was calculated to determine whether an overall improvement or lack thereof had occurred. Group A showed a clinical improvement of 48.95 %, whereas Group B showed a clinical improvement of 62.62 %. 
Intragroup analysis

The Friedman test was used to form a comparison, of each group individually, over time. With regards to the algometer readings taken at the point of pain tolerance between the 1st and 7th visits Group A had a p-value of 0.000 and Group B had a p-value of 0.000. This indicates that both groups had significant differences in the algometer readings over time.

The Wilcoxon Signed Ranks test was used to specifically detect where exactly any differences occurred over time within each of the two groups. As a result of this, the test was able to detect differences between the 1st and 4th visits as well as the 1st and 7th visits.

With regards to the algometer readings taken at the point of pain tolerance, Group A had a p-value of 0.008 for the interval between the 1st and 4th visits and a p-value of 0.001 for the interval between the 1st and 7th visits. Thus both p-values indicate a significant difference occurred over both time intervals. Group B had a p-value of 0.001 for the interval between the 1st and 4th visits and a p-value of 0.001 for the interval between the 1st and 7th visits. Thus both p-values indicate a significant difference occurred over both time intervals.

Intergroup analysis

The Mann-Whitney U test was used to form a comparison between the two groups at specific intervals, that is, between Group A and Group B at the 1st, 4th and 7th visits. With regards to the algometer readings taken at the point of pain tolerance, the 1st visit showed a p-value of 0.648, the 4th visit showed a p-value of 0.787 and 7th visit showed a p-value of 0.068. Therefore, no significant differences occurred between the groups at specific time intervals.
c. Treatment pressure

Figure 4.4: Bar graph comparing pain tolerance and pain threshold values used for both groups at each visit

Figure 4.4 compares the amount of pressure used for treatment during ischaemic compression in kg/cm², which was administered with a hand-held algometer over time. The x-axis represents the average pressure used during treatment at the 1st and 4th visits. The y-axis represents the average percentage increase of pressure used during treatment on a scale of 0.00 kg/cm² to 10.00 kg/cm². The same treatment value used at the 1st visit was used at the 2nd and 3rd visits while the new readings taken at the 4th visit were used at the 5th and 6th visits. No treatment was administered at the 7th visit therefore; Figure 4.4 does not represent the 7th visit.

The treatment pressure used showed that at the 1st visit, Group A had a mean treatment pressure value of 2.57, whilst Group B had a mean treatment pressure value of 6.02. At the 4th visit, Group A had a mean treatment pressure value of 3.84 and Group B had a mean treatment pressure value of 7.52.

Based on the mean treatment pressure value shown above, a percentage was calculated to determine whether an overall improvement or lack thereof had occurred. Group A
showed a clinical improvement of 49.41%, whereas Group B showed a clinical improvement of 24.91%.

**Intragroup analysis**

The Friedman test was not performed for this variable as it only consists of two time periods.

The Wilcoxon Signed Ranks test was used to specifically detect where exactly any differences occurred over time within each of the two groups. As a result of this, the test was able to detect differences between the 1st and 4th visits.

With regards to the treatment pressures that were administered during treatment, Group A had a p-value of 0.003 for the interval between the 1st and 4th visits. This p-value indicates a significant difference occurred over both time intervals. Group B had a p-value of 0.001 for the interval between the 1st and 4th visits. This p-value also indicates a significant difference occurred over both time intervals. These significant differences show that at each subsequent visit, the treatment pressure that was administered was significantly higher than the treatment pressure used at the previous visit.

**Intergroup analysis**

The Mann-Whitney U test was used to form a comparison between the two groups at specific intervals, that is, between Group A and Group B at the 1st and 4th visits. With regards to the treatment pressures that were administered during treatment, the 1st visit showed a p-value of 0.000 and the 4th visit showed a p-value of 0.000. Therefore, significant differences occurred between the groups at specific time intervals. These p-values show that over time, both Group A and Group B were able to tolerate increasing amounts of pressure during ischaemic compression.

**4.5 Conclusion**

The purpose of this chapter was to indicate which statistical analyses were conducted on the research data. The next chapter will give insight on the findings thereof.
CHAPTER 5 – DISCUSSION

5.1 Introduction

The previous chapter served to present the results obtained during the clinical trial of this study. This chapter will propose possible reasons why the results were found as stated and give explanations of the findings with reference to pertinent literature as well as previous studies.

5.2 Demographic Data

A total number of 30 participants were used in this study. The participants were divided into two groups of 15 each. These were named Group A and Group B.

With reference to the age of participants, the mean age for all 30 participants was 25.40 years with a minimum age of 22 years and a maximum age of 35 years. According to Vecchiet (2002), active myofascial trigger points are most commonly found in persons under the age of 50 years. This is due to the fact that these years are the most active in which muscles are more likely to be overused with resulting micro-trauma. The mean age for this study falls well inside this age bracket.

Gender distribution of this study showed that Group A had 3 males and 12 females while Group B had 4 males with 11 females resulting in a total of 7 males and 23 females in the two groups combined. It was found that there was an equal distribution of males and females with $p=1.000$, thus there was a comparable number of male and female participants in this study.

Participants were selected in a random stratified manner with the aim of having equal numbers of male and female participants in the two groups. Epidemiological studies show that the prevalence of myofascial trigger points is slightly higher in the female population (Rollman and Lautenbacher, 2001). Therefore the gender distribution in this study falls in line with these epidemiological studies.
5.3 Subjective Data

5.3.1 Numerical Pain Rating Scale

Clinical interpretation

Based on the mean values obtained and noted in Chapter four, Group A and Group B both showed a clinical improvement over time. Group B showed a greater clinical improvement over time however, this improvement was not significant.

Intragroup analysis

Group A and Group B both showed significant differences with regards to the NPRS values obtained over time. Therefore, Group A and Group B demonstrated clinical improvements that were statistically significant. These statistically significant changes were consistent throughout the treatment period, as measured at the 4th and 7th visits, for both groups.

Intergroup analysis

The results from the NPRS statistical analysis revealed that there was no statistical significance between both Group A and Group B over time. This shows that neither Group A nor Group B showed a significant improvement above the other in their individual pain perception of myofascial trigger points at the specific time intervals.

5.3.2 Subjective data discussion

Ischaemic compression is a treatment method that causes the intentional and temporary decrease in blood supply within a myofascial trigger point in order to increase the local blood flow. This process leads to the elimination of waste products thereby increasing the local oxygen supply. This also allows for the affected tissues to begin the process of healing (Arnau-Masanet, Barrios-Pitarque, Bosch-Morell, Montanez-Aguilera, Pecos-Martin and Valtuena-Gimeno, 2010). Ischaemic compression has the ability to increase local microcirculation and increase the oxygen supply to hypoxic cells (Hakguder, Birtane, Gurcan, Kokino, and Turan, 2003). This therefore accounts for the statistical improvement found over time for both the groups (intragroup analysis).
De Las Peñas et al., (2006) carried out a study to compare the effects of a single treatment of the ischemic compression technique with transverse friction massage for myofascial trigger point tenderness, the results showed a significant improvement in the pain pressure threshold and the significant decrease in the visual analogue scores. Hou (2002) investigated the immediate effect of physical therapeutic modalities on myofascial pain in the upper trapezius muscle and found that ischaemic compression therapy provides immediate pain relief and the suppression of myofascial trigger point sensitivity. In another randomized controlled trial by Gemmell, Miller and Nordstrom (2008), it was shown that ischaemic compression was superior to sham ultrasound in immediately reducing pain in patients with active trigger points.

Pain receptors (free nerve endings) are found within human body tissues and function to generate and transmit pain impulses to the pain centre of the brain, the thalamus. Pain impulses are transmitted via A-delta and C-fibers (peripheral nerves) as well as via lateral spinothalamic tracts found within the spinal cord (Melzack and Wall, 1965).

Three spinal cord systems receive nerve impulses transmitted by the stimulation of skin receptors. These are the substantia gelatinosa cells found in the dorsal horn of the spinal cord, the dorsal-column fibers that transmit impulses to the brain and lastly the initial transmission (T) cells that are found in the dorsal horn of the spinal cord. It is the interaction of these three systems that give rise to the pain gate control theory. The substantia gelatinosa cells function to moderate the afferent impulses before they reach the T-cells. The dorsal-column cells activate selective processing in the brain and therefore function to have an impact on the gate control system. The T-cells function to activate neural action mechanisms (Melzack and Wall, 1965).

According to the pain gate theory, it is this stimulation of the relevant brain centers that cause the activation of the descending afferent fibers which have the ability to influence the afferent fibers at the initial synaptic level (Melzack and Wall, 1965).

Pressure receptors are more thickly myelinated as well as notably longer when compared to that of pain receptors and pain fibers. This allows pressure receptors to possess the ability to transmit pressure stimuli quicker than that of pain receptors (Tsau, 2007).
Pressure stimuli also increase the activity in large diameter fibers (Baldry, 1993) which facilitates the closure of the gate to pain stimuli (Tsau, 2007).

The NPRS was used to measure the participants’ individual perception of pain. Clinically, both groups showed an improvement in pain relief over time based on the subjective data collected. Ischaemic compression creates a mechanical disruption of the actin-myosin myofibril cross links that are locked, within the trigger point (Perle et al., 1999). This causes a decrease in the sensory afferent input of the noxious stimuli to the brain via the pain gate theory (Martin, Wilcox and Moodley, 2008). Pain relief associated with ischaemic compression is also linked with alternating spinal reflex mechanisms that cause a decrease in muscle spasm (Ingbur, Kostopoulos, Larkin and Nelson, 2008).

As both groups showed a clinical pain improvement it could be said that the application of pressure, whether at pain threshold or at pain tolerance, is able to close the pain gate to painful stimuli. As mentioned in Chapter two, Merkel’s discs respond to a more superficial and lighter pressure (Iheanacho and Olubiyi, 2007) and would therefore respond to the pressure at pain threshold. Pacinian corpuscles respond to deep pressure (Boron and Boulpaep, 2011) and would therefore respond to pressure at pain tolerance. Based on the pain gate theory explanation, the activation of both of these receptors was able to close the pain gate to painful stimuli (Baldry, 1993) as mentioned above. Therefore, it can be deduced that it could be possible for treatment at pain threshold to have a similar effect in pain relief when compared to treatment at pain tolerance which would substantiate the statistically significant improvement seen in both groups over time.

5.4 Objective Data

5.4.1 Pressure algometer

a. Pain threshold

Clinical interpretation

In terms of clinical significance and the mean values obtained and seen in Chapter four, both Group A and Group B demonstrated clinical improvements over time and again Group B showed a greater improvement in the objective algometry readings for pressure pain
threshold and myofascial trigger point sensitivity. However, this improvement was not significant.

Intragroup analysis

Taking into consideration the intragroup analysis the results from the pressure algometer statistical analysis revealed that both Group A and Group B showed significant differences in the algometer readings over time. Thus, participants in both groups had a significant improvement in their threshold of pain in the rhomboid muscle with the application of pressure by the algometer from the 1st to the 7th visits.

Intergroup analysis

In terms of the intergroup analysis, the statistical results for the pressure algometer showed that there was no statistical significance when comparing Group A to Group B at either the 1st, 4th or 7th visits. This indicates that before treatment had begun there was no discrepancy in the average pressure algometer readings between the two groups. At the end of the course of treatment, neither Group A nor Group B showed a significant improvement above the other at the specific time intervals.

b. Pain tolerance

Clinical interpretation

A clinical interpretation could be made based on comparing Group A and Group B. According to the mean values obtained and noted in Chapter four, both groups showed a clinical improvement over time. Group B showed a greater improvement than Group A with regard to objective algometer readings for pressure pain tolerance and myofascial trigger point sensitivity over time however, this improvement was not significant.

Intragroup analysis

In terms of intragroup analysis, the results from the pressure algometer statistical analysis revealed a significant difference for both groups over time. Thus, participants from both
groups showed a statistical improvement in the pain tolerance of pain in the rhomboid muscles with the application of pressure by the algometer from the 1st to the 7th visits.

**Intergroup analysis**

With regard to intergroup analysis the statistical results show that there was no statistical significance when comparing Group A to Group B at either the 1st, 4th and 7th visits. This indicates that before the initiation of treatment there was no discrepancy in the average pressure algometer readings between the two groups. Neither Group A nor Group B showed a significant improvement above the other at the specific time intervals.

c. **Treatment pressure**

**Clinical interpretation**

In terms of clinical significance, Group A showed a greater clinical improvement over time when compared to Group B as seen in Chapter four. Group A thus showed a greater improvement than Group B with regard to the amount of increasing pressure that could be used during treatment.

**Intragroup analysis**

Taking into consideration the intragroup analysis, the statistical analysis revealed that both groups demonstrated significant differences in with standing higher treatment pressures over time. Therefore, participants in both groups were shown to be increasingly able to tolerate higher treatment pressures over time.

**Intergroup analysis**

With regard to intergroup analysis the statistical results show that there was a clinical significant difference when comparing Group A to Group B at the 1st and 4th visits. This indicates that both groups were increasingly able to tolerate higher treatment pressures at the specific time intervals.
5.4.2 Objective data discussion

An algometer is used to quantitatively assess the pressure pain threshold of a myofascial
trigger point (De Las Peñas, Campo, Carnero, and Miangolarra-Page, 2005). The pressure
pain threshold is defined by the minimum amount of pressure that causes pain (Ylinen et
al., 2007). Based on the objective data obtained during this study, both Group A and Group
B were proven to be effective with regard to pain relief.

A study done by Dearing and Hamilton (2008) found that ischaemic compression was more
effective than Muscle Energy Technique in reducing pressure pain threshold at trigger
points in asymptomatic patients.

According to the results obtained during this study, both Group A and Group B treatment
protocols proved to be effective for active rhomboid myofascial trigger points and neither
Group A nor Group B showed to be superior to the other. A possible explanation for the
improvement seen in both groups could be based on the pressure mechanoreceptors, the
Pacinian corpuscle and Merkel’s discs.

As discussed in 2.6.2, one would assume that Group B that received pressure at pain
tolerance would have a superior effect when compared to Group A. This assumption could
be based on the fact that the increased mechanical deformation of the Pacinian corpuscles
that occurs with deeper pressure application would have a higher frequency of nerve
impulses (Kimball, 2011). This increased mechanical deformation of the Pacinian
corpuscles may not occur in Group A and specifically with pressure at pain threshold as
perhaps the applied pressure is not deep enough to stimulate the Pacinian corpuscles.
However, as mentioned in 2.6.3, Pacinian corpuscles are phasic (rapid adapting) receptors
and therefore cease firing generator potentials after a short period of time even if the deep
pressure stimulus is maintained. Therefore, the application of a steady deep pressure
results in the dissipation of the receptor potential due to their ability to adapt to stimuli
(Iheanacho and Olubiyi, 2007). This would possibly explain why only a short period of
treatment, specifically 30 seconds, was sufficient enough to exert an effect.
It could also be said that Group A improved as according to Zelena (1994) Merkel’s discs generate impulses for as long as the superficial pressure stimulus is maintained as they are tonic receptors.

Therefore, it can be deduced that both groups improved due to different mechanisms of mechanoreceptor activity and characteristics. Group A showed an improvement as a result of the sustained action of the Merkel’s discs for the full duration of the treatment while Group B’s improvement could possibly be attributed to the rapid and intense response of the Pacinian corpuscles over the beginning of the treatment. Although the improvements that were seen may have occurred due to two separate mechanoreceptor mechanisms, ultimately both processes lead to the stimulation of large diameter nerve fibers which was the desired effect as pain was relieved.

Taking into consideration the above literature it can be deduced that Group A possibly showed an improvement over time due to the sustained action of the Merkel’s discs that were sensitive to the lighter pressure administered in Group A. According to the above literature, it could also possibly be said that Group B showed an improvement over time due to the increased amount of mechanical deformation to the Pacinian corpuscle that occurred with the deeper pressure administered in Group B.

5.5 Conclusion

In conclusion, both Group A and Group B showed both clinical and statistical significant improvements over time with regard to the subjective and objective data results collected.

Group A specifically showed statistical significant improvements over time for the NPRS and pain threshold and tolerance algometer readings. Group A also showed a clinical improvement for the NPRS and pain threshold and tolerance algometer readings.

Group B specifically showed a statistical significant improvement over time for the NPRS and pain threshold and tolerance algometer readings. Group B also showed a clinical improvement for the NPRS and pain tolerance and threshold algometer readings.

Further review of literature highlights the mechanism of ischaemic compression and the appropriate pressures used. Ischaemic compression creates a mechanical disruption
(Perle et al., 1999) and as previously mentioned it is the actual amount of mechanical disruption to the mechanoreceptors (Kimbal, 2011) that renders ischaemic compression effective in treating active myofascial trigger points via the pain gate theory. Therefore, one would assume that Group B (pain tolerance) would have a superior treatment effect when compared to Group A (pain threshold) however, the sustained action of the Merkel’s discs allowed Group A to fair just as well as Group B. The various clinical and statistical improvements show that both methods of ischaemic compression faired equally against each other, each via their own different mechanisms, and therefore can both be used to effectively treat active rhomboid myofascial trigger points.
CHAPTER SIX – CONCLUSION AND RECOMMENDATIONS

6.1 Conclusion

The aim of this study was to determine whether ischaemic compression that is applied at pain threshold would have a similar effect when compared to that of pain tolerance in the treatment of active rhomboid myofascial trigger points, with regard to changes in pain threshold and tolerance as well as pain perception.

A total number of 30 participants were used in this study, 15 of those were placed in the pain threshold group while the other 15 were placed in the pain tolerance group. Participants were treated for a total of 6 treatments receiving ischaemic compression, of 30 seconds in duration, at either pain threshold or pain tolerance. Subjective and objective measurements were taken at the 1st, 4th and 7th visits and consisted of the Numerical Pain Rating Scale and pressure algometer readings.

Based on the above results, it can be concluded that ischaemic compression at both pain threshold and tolerance can be used to effectively treat active myofascial trigger points in the rhomboid muscle, as both groups showed a significant clinical and statistical improvement in both the subjective and objective perceptions of pain. It was not statistically concluded whether the one treatment was superior to the other as both groups performed equally throughout the trial period.

The outcome of this study will have a positive effect on the Chiropractic profession by contributing further knowledge on the treatment protocol of ischaemic compression. The knowledge that ischaemic compression is as effective at pain threshold will allow greater patient comfort and a decreased pain experience during treatment. This may have an improved treatment outcome. This study also sheds light on the amount of pressure and length of pressure application with regard to ischaemic compression which will assist professionals during the use of ischaemic compression.

6.2 Recommendations

The following recommendations can be used in the design of future studies involving ischemic compression as a treatment protocol in myofascial pain and dysfunction:
a. A larger sample group could be used allowing for a more accurate representation of the population;

b. A sample containing either males or females could be included in order to exclude any gender variables in pain perception. According to Kroner-Herwig, Gabmann, Tromsdorf and Zahrend (2012) in a study done to determine the effects of sex and gender role on responses to pressure pain, it was found that females are more sensitive to mechanical pain than males. Females were also found to have lower pain thresholds than males;

c. EMG readings could be included in the study in order to measure the level of muscle activity within the rhomboid muscle before and after treatments;

d. A third group could be included that could receive treatment at a pressure between pain threshold and pain tolerance;

e. A study could be done comparing ischaemic compression at pain tolerance versus ischaemic compression at pain tolerance coupled with chiropractic adjustment to the spinal segments innervating the rhomboid muscles (C4, C5) in order to determine the efficacy of a comprehensive treatment of the involved muscle;

f. A one month follow up could be included in the study after the completion of the 6 treatments in order to determine the long term effects of the treatment;

g. Different durations of ischaemic compression treatment could be compared. As mentioned previously, there is a lack of research to indicate the most effective duration of ischaemic compression (Gulick, 2010). Hunter (1998) states that pressure should be applied for a period of 30 to 60 seconds. Turchaninov (2001) suggests that pressure should be applied for 30 seconds while Hanten, Olsen, Butts and Nowicki (2000) found that it is best to maintain pressure until the tenderness is no longer present.
REFERENCES


APPENDIX A: ADVERTISEMENT

DO YOU SUFFER FROM AN ACHING OR BURNING PAIN BETWEEN YOUR SHOULDER BLADES?

Are you between the ages of 18 and 40 years old?

You may qualify to participate in a research study aimed to treat active rhomboid myofascial trigger points at pain tolerance versus pain threshold.

Treatment is conducted at the Supervised UJ clinic, Sherwell road, Doornfontein.

Please contact Fatima Ismail if you are interested

<table>
<thead>
<tr>
<th>Fatima Ismail</th>
<th>071 193 1745</th>
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<td>Fatima Ismail</td>
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APPENDIX B: INFORMATION FORM

DEPARTMENT OF CHIROPRACTIC

INFORMATION FORM

My name is Fatima Ismail and I am currently a Chiropractic student, completing my Masters Degree at the University of Johannesburg. I would like to thank you for volunteering in this study entitled “The effectiveness of treatment at pain threshold versus pain tolerance using ischaemic compression”.

The aim of this study is to determine whether pressure applied at pain threshold or at pain tolerance is more effective during ischaemic compression of an active myofascial trigger point.

On the first visit, you will be questioned about your medical history and undergo a physical examination as well as a neck examination. You will also be examined for active myofascial trigger points in your rhomboid muscles. The most active trigger point will then be selected and pressure readings will be taken with a hand held algometer. You will then be placed randomly into one of two groups. A total of six treatments of ischaemic compression will be administered twice a week over a three week trial period. Only measurements will be taken at the seventh and final visit.

Pressure readings will be taken with a hand held algometer at the first, fourth and seventh visits. As a participant of this study you will be exposed to the substantial benefits of ischaemic compression while only experiencing mild to moderate pain or discomfort during the 30 second treatment time. Ischaemic compression serves to increase blood perfusion...
to the area with the consequent removal of waste products from the involved muscle thereby greatly relieving the participant from the pain caused by the active myofascial trigger points.

The research study will take place at the University of Johannesburg Chiropractic Day Clinic. Your privacy will be protected as only the doctor, participant (you) and clinician will be in the treatment room. Your anonymity will be ensured as your personal information will be converted into data and therefore cannot be traced back to you. Standard doctor/patient confidentiality will be adhered to at all times when compiling the research dissertation.

All procedures will be explained to you and all participation is entirely on a voluntary basis; withdrawal at any stage will not cause you any harm. Risks are minimal with ischaemic compression. Discomforts may include tenderness and slight bruising (Shacksnovis and Korporaal, 2005). Benefits include the relief of pain and muscle spasm as well as local hyperaemia (Hou et al., 2002). All the necessary precautions will be taken to ensure patient safety. Results of this study will be made available to you on request.

University of Johannesburg’s ethics clearance number:

**AEC31-01-2013**

Should you have any concerns or queries regarding the current study, the following persons may be contacted.

<table>
<thead>
<tr>
<th>Researcher:</th>
<th>Name</th>
<th>Telephone number</th>
</tr>
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<tr>
<th>Supervisor:</th>
<th>Name</th>
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<tr>
<td>Dr Caroline Hay</td>
<td></td>
<td>011 559 6500</td>
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<tr>
<th>Co-supervisor:</th>
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<tbody>
<tr>
<td>Dr Christopher Yelverton</td>
<td></td>
<td>011 559 6218</td>
</tr>
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</table>
APPENDIX C: CONSENT FORM

DEPARTMENT OF CHIROPRACTIC

CONSENT FORM

I have fully explained the procedures and their purpose. I have asked whether or not any questions have arisen regarding the procedures and have answered them to the best of my ability.

Date: ______________________          Researcher: ______________________________

I have been fully informed as to the procedures to be followed and have been given a description of the discomfort risks and benefits expected from the treatment. In signing this consent form I agree to this form of treatment and understand my rights and that I am free to withdraw my consent and participation in this study at any time. I understand that if I have any questions at any time, they will be answered.

Date: _______________________ Participant: ______________________________
APPENDIX D: CASE HISTORY FORM

UNIVERSITY OF JOHANNESBURG
CHIROPRACTIC DAY CLINIC

CASE HISTORY

Date: ______________

Patient: ___________________________ File No: __________

Age: _____ Sex: ________ Occupation: ______________

Student: __________________________ Signature: __________

Complies with Inclusion criteria of the research:

Clinician: __________________________
Signature: __________________________

Examination:
Previous: UJ Other
Current: UJ Other

X-ray Studies:
Previous: UJ Other
Current: UJ Other

Clinical Path. Lab:
Previous: UJ Other
Current: UJ Other

Case status:
PTT: Conditional:
Signed off: Final sign out:

Recommendations:

1
**Students case history**

1. **Source of history:**

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

3. **Present illness:**
   - Location
   - Onset
   - Duration
   - Frequency
   - Pain (character)
   - Progression
   - Aggravating factors
   - Relieving factors
   - Associated Sx's and Sg's
   - Previous occurrences
   - Past treatment and outcome
4. Other complaints:

5. Past history
   General health status
   Childhood illnesses
   Adult illnesses
   Psychiatric illnesses
   Accidents/injuries
   Surgery
   Hospitalisation

6. Current health status and lifestyle
   Allergies
   Immunizations
   Screening tests
   Environmental hazards
   Safety measures
   Exercise and leisure
   Sleep patterns
   Diet
   Current medication
   Tobacco
   Alcohol
   Social drugs
7. Family history:
   Immediate family:
   Cause of death
   DM
   Heart disease
   TB
   HBP
   Stroke
   Kidney disease
   CA
   Arthritis
   Anaemia
   Headaches
   Thyroid disease
   Epilepsy
   Mental illness
   Alcoholism
   Drug addiction
   Other

8. Psychosocial history:
   Home situation
   Daily life
   Important experiences
   Religious beliefs

9. Review of systems:
   General
   Skin
   Head
Eyes
Ears
Nose/sinuses
Mouth/throat
Neck
Breasts
Respiratory
Cardiac
Gastro-intestinal
Urinary
Genital
Vascular
Musculoskeletal
Neurologic
Haematologic
Endocrine
Psychiatric
APPENDIX E: FULL PHYSICAL EXAMINATION

UNIVERSITY OF JOHANNESBURG
CHIROPRACTIC DAY CLINIC

PHYSICAL EXAMINATION

(NOTE: only if Cervical Spine Regional is complete)

Underline abnormal findings in RED.

Date: ____________________

Patient: ____________________ File No: ______________

Clinician: ____________________ Signature: ______________

Student: ____________________ Signature: ______________

Height: _______  Weight: _______  Temp: _______

Rates:  Heart: _______  Pulse: _______  Respiration: _______

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<th>Arms:</th>
<th>L</th>
<th>R</th>
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<tbody>
<tr>
<td></td>
<td>Legs:</td>
<td>L</td>
<td>R</td>
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</table>

General Appearance:

________________________________________________________________________

________________________________________________________________________

________________________________________________________________________

________________________________________________________________________

________________________________________________________________________
STANDING EXAMINATION

1. Minor’s sign
2. Skin changes
3. Posture: Erect
   Adam’s
4. Ranges of motion (Thoracolumbar Spine)
   T/L spine: Flexion: 90° (fingers to floor)
   Extension: 50°
   R. lat. flex: 30° (fingers down leg)
   L. lat. flex: 30° (fingers down leg)
   Rot. to R: 35°
   Rot. to L: 35°

/ = pain-free limitation
// = painful limitation

5. Romberg’s sign
6. Pronator drift
7. Trendelenburg’s sign
8. Gait:
   - rhythm
   - balance
   - pendulousness
   - on toes
   - on heels
   - tandem
9. Half squat
10. Scapular winging
11. Muscle tone
12. Spasticity/Rigidity
13. Shoulder:
   symmetry
   ROM
   - glenohumeral
   - scapulo-thoracic
   - acromioclavicular
   - elbow
   - wrist
14. Chest measurement:
   - inspiration
   - expiration
   | L | R |
   | cm | cm |

15. Visual acuity

16. Breast examination:
   Inspection:
   - skin
   - size
   - contour
   - nipples
   - arms overhead
   - hands against hips
   - leaning forward
   Palpation
   - axillary lymph nodes
   - breast incl. tail

**SEATED EXAMINATION**

1. Spinal posture
2. Head
   - hair
   - scalp
   - skull
   - face
   - skin
3. Eyes:
   Observation
   - conjunctiva
   - sclera
   - eyebrows
   - eyelids
   - lacrimal glands
   - nasolacrimal duct
   - position and alignment
   - corneas and lenses
   - corneal reflex

   ocular movement
   | L | III | IV | VI | R | III | IV | VI |

   visual fields
   accommodation
   Opthalmoscopic
   Examination
   - iris
   - pupils
   - red reflex
   - optic disc
   - vessels
   - general background
- macula
- vitreous
- lens

4. Ears:
   - auricle
   - ear canal
   - drum

   - auditory acuity
   - Weber test
   - Rinne test

5. Nose:
   - External
   - Internal
     - septum
     - turbinates
     - olfaction

6. Sinuses (frontal & maxillary):
   - tenderness
   - transillumination

7. Mouth and pharynx:
   - lips
   - buccal mucosa
   - gums and teeth
   - roof
   - tongue
     - inspection
     - movement
     - taste
     - palpation

   - pharynx
     - CN X
     - inspection

   - carotid arteries (thrills, bruit)
   - Cranial Nerves
     - CN V
     - CN VII
     - CN VIII (nystagmus)
     - CN IX
     - CN XI
     - CN X11

8. Peripheral vasculature:
   - Inspection
     - skin
     - nail beds
     - pigmentation
     - hair loss
- Palpation
  - pulses: femoral, dorsalis pedis, popliteal, radial, post. Tibial, brachial
  - lymph nodes: epitrochlear, femoral (horizontal & vertical)
  - temperature (feet and legs)

- Manual compression test
- Retrograde filling (Tredelenburg) test
- Arterial insufficiency test

10. Musculoskeletal:
   (i) ROM
   - hip

<table>
<thead>
<tr>
<th>L</th>
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<tr>
<td>Apparent</td>
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<tr>
<td>Actual</td>
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- knee
- ankle

(ii) leg length

- Co-ordination
  - point to point
  - dysdiachokinesia

9. TMJ
- Inspection
  - ROM
  - deviation
- Palpation
  - crepitus
  - tenderness
10. Thorax
   • Inspection
     - skin
     - shape
     - respiratory distress
     - rhythm (respiratory)
     - depth (respiratory)
     - effort (respiratory)
     - intercostals supraclavicular retraction
   • Palpation
     - tenderness
     - masses
     - respiratory expansion
     - tactile fremitus
   • Percussion
     - lungs (posterior)
     - diaphragmatic excursion
     - kidney punch
   • Auscultation
     (i) breath sounds
     - vesicular
     - bronchial
     (ii) adventitious sounds
     - crackles (rales)
     - wheezes (rhonchi)
     - rubs
     (iii) voice sounds
     - broncophony
     - whispered pectoriloquy
     - egophony
   • Cardiovascular
     - auscultation (aortic murmurs)
     - Allen’s test

SUPINE EXAMINATION

1. JVP
2. PMI
3. Auscultation heart
   (L. lat. Recumbent)
4. respiratory excursion
5. percussion chest
   (anterior)
6. breast palpation
7. Abdominal Examination
   • Inspection
     - skin
     - umbilicus
     - contour
     - peristalsis
     - pulsations
     - hernias (umbilical/incisional)
• Auscultation  
  - bowel sound  
  - bruit  

• Percussion  
  - general  
  - liver  
  - spleen  

• Palpation  
  - superficial reflexes  
  - cough  
  - light  
  - rebound tenderness  
  - deep  
  - liver  
  - spleen  
  - kidneys  
  - aorta  
  - intra-/retro-abdominal wall mass  
  - shifting dullness  
  - fluid wave  

• Acute abdomen  
  - where pain began and now  
  - cough  
  - tenderness  
  - guarding/rigidity  
  - rebound tenderness  
  - roving’s sign  
  - psoas sign  
  - obturator sign  
  - cutaneous hyperaesthesia  
  - rectal exam  
  - Murphy’s sign  

MENTAL STATUS

(i) Appearance and behaviour  
  - level of consciousness  
  - posture and motor behaviour  
  - dress, grooming, personal hygiene  
  - facial expression  
  - affect  

(ii) Speed and language  
  - quantity  
  - rate  
  - volume  
  - fluency  
  - aphasia (pm)  

(iii) Mood  

(v) Memory and attention  
  • orientation (time, place, person)  
  • remote memory
(vi) Higher cognitive functions

- recent memory
- new learning ability
- information and vocabulary
- (general and specialised knowledge)
- abstract thinking

### NEUROLOGICAL EXAMINATION (LUMBAR SPINE)

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<th>DERMATOMES</th>
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<td></td>
<td></td>
<td>Hip Flexion (L1/L2)</td>
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APPENDIX F: CERVICAL SPINE REGIONAL FORM

UNIVERSITY OF JOHANNESBURG
CHIROPRACTIC DAY CLINIC

REGIONAL EXAMINATION
CERVICAL SPINE

Date: ______________________

Patient: ______________________  File No: ______________________

Clinician: ______________________  Signature: _________________

Student: ______________________  Signature: _________________

OBSERVATION

• Posture
• Size
• Swellings
• Scars
• Discolouration
• Hairline
• Bony and soft tissue contours
• Shoulder level
• Muscle spasm
• Facial expression

5. RANGE OF MOTION

Flexion = 45° - 90°
Extension = 55° - 70°
L/R Rotation = 70° - 90°
L/R Lat Flexion = 20° - 45°
PALPATION

- Lymph nodes
- Trachea
- Thyroid gland
- Pulses/thrills
- Tenderness
- Muscle Tone
- Active MF Trigger Points
  - SCM
  - Trapezius
  - Scaleni
  - Levator Scapulae
  - Posterior Cervical musculature

ORTHOPAEDIC EXAMINATION

1. Doorbell Sign
2. Max. Cervical Compression
3. Spurling’s manoeuvre
4. Lateral Compression (Jackson’s test)
5. Kemp’s Test
6. Cervical Distraction
7. Shoulder abduction Test
8. Shoulder depression Test
9. Dizziness rotation Test
10. Lhermitte’s Sign
11. O’ Donoghue Manoeuvre
12. Brachial Plexus Tension
13. Carpal tunnel syndrome:
   - Tinel’s sign
   - Phalen’s Test
14. TOS:
   - Halstead’s test
   - Adson’s test
   - Eden’s (traction) test
   - Hyperabduction (Wright’s) test – Pec minor
   - Costoclavicular test

Remarks:

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<td>CAROTIDS</td>
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<td>SUBCLAVIAN ARTERIES</td>
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<tr>
<td>WALLENBERG’S TEST</td>
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COMMENTS:
### MOTION PALPATION

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<td>Ext LF</td>
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### NEUROLOGICAL EXAMINATION

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<th>Left</th>
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<td>C6</td>
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| Bioeps C5 |
| Brachioradialis C6 |
| Triceps C7 |

91
APPENDIX G: SOAP NOTE

CHIROPRACTIC DAY CLINIC

SOAP NOTE:

<table>
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<tr>
<th>Patient:</th>
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<tbody>
<tr>
<td>File No:</td>
<td>Student:</td>
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<tr>
<td>Date:</td>
<td>Clinician:</td>
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S: O:

A: P:

Comments:

Patient: | Visit No: |
<table>
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</thead>
<tbody>
<tr>
<td>File No:</td>
<td>Student:</td>
</tr>
<tr>
<td>Date:</td>
<td>Clinician:</td>
</tr>
</tbody>
</table>

S: O:

A: P:

Comments:
APPENDIX H: NUMERICAL PAIN RATING SCALE SCORE SHEET

Name: _______________________________
Group: ______________________________
Visit: ______________________________

This is a scale of 0 to 10.
A score of 0 indicates no pain at all.
A score of 10 indicates the “worst pain” that you have experienced.
Please mark with a cross how you would describe your pain at the moment.

**Numerical Pain Rating Scale**

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
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<tbody>
<tr>
<td>No pain</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Severe pain</td>
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### APPENDIX I: PRESSURE ALGOMETER SCORE SHEET

#### Visit One

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<thead>
<tr>
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<tbody>
<tr>
<td></td>
<td>Pain threshold</td>
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<tr>
<td></td>
<td>Pain tolerance</td>
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#### Visit Four

<table>
<thead>
<tr>
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<th>Algometer reading</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Pain threshold</td>
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<tr>
<td></td>
<td>Pain tolerance</td>
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#### Visit Seven

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<tr>
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<th>Algometer reading</th>
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<td>Pain threshold</td>
</tr>
<tr>
<td></td>
<td>Pain tolerance</td>
</tr>
</tbody>
</table>
KEY:  

Group 1 = Pain threshold  

Group 2 = Pain tolerance  

PRS = Pain rating scale from 1 – 10  

Algometer = kg/cm$^2$