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THE EFFECT OF COSTOVERTEBRAL ADJUSTMENT VERSUS ISCHAEMIC COMPRESSION OF RHOMBOID MUSCLES FOR INTERSCAPULAR PAIN

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

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Dr D. Whelan
DECLARATION

I, Jared Ashley Irwin do hereby declare that this is my own unaided work except where otherwise indicated in the text. This dissertation is being submitted for the degree of Masters Degree in Technology at the University of Johannesburg. It has not been previously submitted for any degree or examination at any other Technikon or University.

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DEDICATION

To my parents, Wesley and Bernadine, you have given everything to help me reach my dream. You mean the world to me, and I am truly blessed. There is nothing that I can repay you with for what you have done for me. You stood by me through the difficult times and supported me through the good times, and supported every decision I made. There are no words to describe how grateful I am to have you as my parents. All I can say from the bottom of my heart is thank you, and I hope I have made you proud of the man that I have become.

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To my fellow classmates and future colleagues Jessica Wood, Glen Paton, Markus Linzbacher and Leonie Bosch. We have finally become the people we dreamed of. Thank you for all the memories that helped us achieve our goal through many hard years of studying.

“The doctor of the future will give no medicine but will interest his patients in the care of the human frame, in diet and the cause and prevention of disease”

Thomas Edison
ACKNOWLEDGEMENTS

To Dr Chris Yelverton, not just my supervisor but a great mentor, thank you for allowing me to follow my dream. You have always pushed me as a person and a student to be the best chiropractor that I can be. Your constant guidance, advice and support have taught me that it is easier to stand up for what I believe in than to stand up for what I don’t believe in. Your hard work and dedication to chiropractic over the years has brought me to where I am today. Thank you.

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ABSTRACT

PURPOSE: The aim of the study was to compare the effectiveness of treating inter-scapular pain with either chiropractic adjustments delivered to the costovertebral joints, with ischaemic compression of the myofascial trigger points of the rhomboid muscles, or using a combination treatment protocol in order to determine which of the three treatment protocols was superior.

DESIGN: This study was a comparative study consisting of three groups of ten participants each. Participants were between the ages of eighteen and forty-five and there was an equal male to female ratio. Prior to becoming a participant in this study individuals were assessed according to the inclusion and exclusion criteria. A McGill Pain Questionnaire, numerical pain rating scale, a clinical case history, full physical examination, a cervical regional examination were completed. The method of treatment for each participant was determined by random group allocation. Group 1 received chiropractic adjustments to the costovertebral joints, Group 2 received ischaemic compression to the myofascial trigger points of the rhomboid muscles. Group 3 received a combination treatment involving both chiropractic adjustments to the costovertebral joints together with ischaemic compression of the rhomboid trigger points. Subjective and objective readings were based on the above treatment protocols. All participants received a total of six treatments over a three week period.

MEASUREMENTS: Subjective measurements were obtained by the Numerical Pain Rating Scale and the McGill Pain Questionnaire. Objective measurements were obtained using the hand-held pressure algometer. The data was collected on the first, fourth and seventh consultations.

OUTCOME: With regards to the subjective readings, the results from the McGill Pain Questionnaire for the intragroup analysis indicated that the ischaemic compression group showed the greatest improvement over time (84.06%). No statistically significant differences were noted for the intergroup analysis. The intragroup analysis of the Numerical Pain Rating Scale indicated that the adjustment group showed the greatest improvement over time (78.70%). The intergroup analysis indicated that there were no statistically significant differences.
With regards to the objective measurements, the intragroup analysis of the pressure algometer readings indicated that the ischaemic compression group showed the greatest improvement over time (35.44%). Once again, there were no statistically significant differences with the intergroup analysis.

**CONCLUSION:** Based on the results of this study, it showed that all three treatment groups were effective in decreasing inter-scapular pain and disability. Although all three treatment protocols have shown to be effective, and have shown similar improvement, intergroup analysis indicates that statistically there is no treatment protocol that is seen to be more superior or more effective in treating inter-scapular pain.
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1.1 PROBLEM AND ITS SETTING

Inter-scapular pain is a common cause of upper back pain and one of the regions with the least amount of research conducted, compared with the cervical and lumbar regions. The thoracic spine has largely been neglected. This may be attributed to the technical difficulties associated with movement analysis in the region and the belief that the thoracic spine is less commonly implicated in clinical pain syndrome (Schiller, 2001).

According to Gatterman (2004), costal joint dysfunction is characterised by pain during inspiration which will generally appear due to sudden, unguarded movement such as an explosive cough or sneeze or more severe such as a crushing blow to the chest. Unilateral thoracic spine pain is the most common presenting symptom, pain is accentuated by deep inspiration, coughing or by sneezing. Costovertebral dysfunction can be indicated by the presence of a prominent rib head with localized tenderness, as well as an absence of joint movement found on motion palpation.

As with any joint in the human body the costovertebral joint where the ribs articulate with the spinal column can undergo dysfunction, which will produce sharp or stabbing pain upon deep respiration. Thoracic pain can also be caused by the straining of postural muscles that support the upper extremities, common sites of myofascial pain are the rhomboids and the trapezius muscles (Gatterman, 2004).

A myofascial trigger point is defined as: “A focal area of hyper-irritability in a muscle that is locally sensitive to pressure and can refer symptoms, usually pain, to other areas of the body”. Myofascial trigger points are reported to exist in almost every soft tissue of the body (Muscolino, 2009).

Myofascial pain syndrome is pain originating from a specific muscle or muscle group. It is characterised by intense, deep muscular pain referred from hypersensitive trigger points in muscle bellies or their tendinous attachment (Gatterman, 2004). Pain referral from trigger points in the rhomboid muscles concentrate along the vertebral (medial) border of the scapula and the paraspinal muscles (Travell and Simmons, 1999). Thoracic myofascial pain syndrome occurs when there are trigger points found in the inter-scapular muscles, which can be due to static loading of the rhomboids
with the arms in the elevated position. Inter-scapular pain can be aggravated by fatigue, static loading and stress (Gatterman, 2004).

According to Raj and Paradise (2004), a trigger point is characterised by a palpable taut band, a focal spot of tenderness in the taut band, painful restricted range of motion, referred pain to a regional site, muscle weakness without atrophy and pain or altered sensation on compression of the tender nodule. Change in muscle tone is an important feature of the subluxation complex. Muscle hypertonicity can perpetuate and prolong joint restriction. It is hypothesised that an adjustment may be able to change the muscle spasm cycle (Esposito and Philipson, 2005).

1.2 PROBLEM STATEMENT

As patients seeking chiropractic treatment for inter-scapular pain, the initial treatment from the chiropractor could involve correcting the presence of rhomboid muscle tension, correcting rib dysfunction at the costovertebral joints or a combination treatment protocol can be involved.

The confusion in treatment protocols can be attributed to the trigger points found in rhomboid muscles which are due to tension, stress and poor posture (Anderson, 2006). In addition the presence of inter-scapular pain can also be caused by rib dysfunction in the mid thoracic spine, which can frequently be misdiagnosised. This can cause an improper treatment protocol given to the patient (Dalton, 2011). When rib dysfunction involving the costovertebral joints occurs, there is an increase in this muscle tension noted in the paraspinal, rhomboid muscles, trapezius muscles and/or levator scapular muscles (Hyde and Gengenbach, 2007). This study in turn could establish which treatment protocol is superior in the treatment of inter-scapular pain.

1.3 AIM OF THE STUDY

The aim of the study was to compare the efficacy of treating inter-scapular pain with either chiropractic adjustments to the costovertebral joints, ischaemic compression of rhomboid trigger points, or a combination treatment involving both chiropractic adjustment and ischaemic compression in order to determine which of the three treatment protocols was superior.
1.4 POSSIBLE OUTCOMES OF THE STUDY

Inter-scapular is a source of severe pain in patients presenting to the chiropractic practice, and it had been reported that further investigation regarding non-invasive treatment was required. This would add value to the chiropractic profession as well as clinical practice. The outcome of this study might provide additional value to the formation of treatment protocols when patients present with inter-scapular pain.

The outcome of this study could determine which treatment protocol in the form of chiropractic adjustments to the costovertebral joints, ischaemic compression to the rhomboid muscles or a combination treatment of both chiropractic adjustment and ischaemic compression was superior for the treatment of inter-scapular pain.
CHAPTER TWO – LITERATURE REVIEW

2.1 INTRODUCTION

This literature review will explain all aspects involved with the rhomboid major and minor muscles, rhomboid myofascial pain syndrome and costovertebral syndrome in detail. This chapter will describe the anatomy of the rhomboid muscles and the ribs. Rhomboid myofascial pain syndrome as well as costovertebral syndrome will be explained in terms of its aetiology and clinical presentation. Treatment of costovertebral syndrome will be discussed with specific emphasis on costovertebral chiropractic adjustments, ischaemic compression to the rhomboid musculature or a combination treatment involving both chiropractic adjustments and ischaemic compression. This will give insight and explain the basis behind this research dissertation and its aims.

2.2 SKELETAL MUSCLE

2.2.1 Skeletal muscle structure

Muscle tissue is a specialised tissue that facilitates movement in the body (Coetzee, Loots and Meiring, 2003).

Histologically (Figure 2.1), skeletal muscle is composed of long and cylindrical multinucleated cells arranged in bundles. Multinucleated skeletal muscle cells result from fusion of embryonic mononucleated myoblasts. These cells show cross-striations and the nuclei are found on the periphery of the cell within the cell membrane. Contraction of the multinucleated cells is quick and voluntary. Contraction is caused by the interaction of thick myosin filaments and thin actin filaments that slide upon one another. This is also known as the sliding filament theory (Junqueira and Carneiro, 2005).
Inside the muscle fibre, transverse tubules encircle myofibrils (Figure 2.2). Myofibrils are cylindrical structures that can actively shorten. They are responsible for skeletal muscle fibre contraction. Myofibrils are anchored at each end of the muscle fibre to the internal surface of the sarcolemma. The external surface of the sarcolemma is attached to the collagen fibres of the skeletal muscle tendon or aponeurosis. This will cause the entire cell to shorten and pull on the tendon during myofibril contraction (Martini, 2004).

The myofibrils consist of bundles of myofilaments. Myofilaments fill a large portion of the sarcoplasm. Myofilaments are composed of actin, which forms the largest part of the thin filaments, and myosin, which forms the largest part of the thick filaments. Myofilaments are arranged into sarcomeres. Sarcomeres are known as the functional units of the muscle fibre. Contraction of the skeletal muscle is caused by the interaction between the thick and thin filaments (Guyton and Hall, 1997).
Skeletal muscle fibres exhibit a pattern of alternating light (I) and dark (A) bands (Figure 2.3b). The darker A bands contain the thick filaments and is located at the centre of the sarcomere. Within the A band there is a zone of overlap where thick and thin filaments overlap. The I band contains thin filaments but no thick filaments. It extends from the A band of one sarcomere to the A band of the next sarcomere (Figure 2.3c). Z lines are the boundary between two adjacent sarcomeres that interconnect the thin filaments of the adjacent sarcomeres (Figure 2.3d). Transverse tubules surround each sarcomere (Junqueira and Carneiro, 2005).

Transverse tubules (refer to T-tubules in Figure 2.2) run transversely to the myofibrils and starts at the cell membrane. The transverse tubules penetrate the entire muscle cell from one side to the other side. The transverse tubules are exposed to the surrounding tissue where they originate at the cell membrane. This enables them to communicate with the fluid surrounding the muscle (Guyton and Hall, 1997). When an action potential spreads over the muscle fibre and the transverse tubules, it stimulates the release of calcium ions from the sarcoplasmic reticulum causing muscle spasm (Young and Heath, 2000).
Triads are located on each side of the M line at the zone of overlap. As a result, the calcium that is released by the sarcoplasmic reticulum enters the regions where the thin and thick filaments interact (Martini, 2004).

Figure 2.3 Schematic diagram illustrating the myofibril (Marieb and Hoehn, 2007)

Skeletal muscle is divided into three categories according to the amount of myoglobin located in the sarcoplasm. Skeletal muscles may contain all three types of fibres but the amount of fibres depends on the function of the muscle (Coetzee, Loots and Meiring, 2003).

The three different types of skeletal muscle fibres are:

Red muscle fibres: The muscle fibres are red due to the considerable amount of myoglobin present in the sarcoplasm. These fibres are small in diameter and contain a large amount of mitochondria.
Contraction of this muscle fibre is a slow reaction but the contraction can be maintained for long periods of time (Coetzee, Loots and Meiring, 2003).

White muscle fibres: The muscle fibres appear white due to less myoglobin in the sarcoplasm. The fibres are larger in diameter with less mitochondria. Contraction of these muscle fibres is fast and powerful but the muscles fatigue quickly (Coetzee, Loots and Meiring, 2003).

Intermediate muscle fibres: These fibres contain red and white muscle fibres (Coetzee, Loots and Meiring, 2003).

2.2.2 The muscle spindle

Muscle spindles are found in all striated muscles and are located within the muscle belly. It sends information regarding muscle length and rate of change of its length to the nervous system (Coetzee, Loots and Meiring, 2003). Muscle spindles are located around intrafusal fibres (Junqueira and Carneiro, 2005). The pointed ends of the muscle spindle attach to the glyocalynx of the surrounding extrafusal muscle fibres. The central portion of each muscle spindle only has a few actin and myosin filaments and does not contract like the ends do. It only functions as a sensory receptor. The end (contracting) portions are excited by small gamma motor nerve fibres located in the anterior horns of the spinal cord (Guyton and Hall, 1997).

Muscle spindles relay sensory nerve impulses all of the time. It also regulates muscle contraction by activating motor neurons via the stretch reflex in order to resist muscle stretch (Martini, 2004). Stretching the muscle spindles will increase the rate of firing. Shortening the spindle will decrease the rate of firing (Guyton and Hall, 1997).

2.2.3 The Golgi tendon organ

Golgi tendon organs are located in muscle tendons at the muscle-tendinous junctions and transmit information regarding the degree of tension in the tendon (Coetzee, Loots and Meiring, 2003). The Golgi tendon organ is an encapsulated proprioceptive sensory receptor. A few muscle tendon fibres pass through the Golgi tendon organ. Tension that develops within this small bundle of muscle tendon fibres will stimulate the organ (Guyton and Hall, 1997). The Golgi tendon organ functions to
monitor external muscle tension and the rate of change during muscle contraction (Martini, 2004). The Golgi tendon organ inhibits the muscle spindle and therefore allows the muscle to relax. The function of the Golgi tendon is to protect muscles, tendons and ligaments from injury (Guyton and Hall, 1997).

2.3 THE RHOMBOID MUSCLES

2.3.1 The Anatomy of the Rhomboid muscle

a. Origin and Insertion

The rhomboid muscle is made up of two separate muscles, the rhomboid major and rhomboid minor muscles. These muscles lie deep to the trapezius muscle and form a broad parallel band that pass inferio-laterally from the vertebrae to the medial border of the scapula. Because of their anatomical considerations the rhomboid muscles adduct and elevate the scapula. Attachment of the rhomboid major fibres to the lower vertebral border of the scapular will allow the scapula to rotate medially, turning the glenoid fossa down. It also assists in forceful adduction and extension of the arm by stabilising the scapula in the retracted position (Travell and Simons, 1999). It can therefore be said that the rhomboid muscles retract and rotate the scapula, depressing the glenoid cavity (Moore, 1999).

The rhomboid minor muscle (Figure 2.4) is the more cephalad of the two muscles and attaches to the ligamentum nuchae and to the spinous process of the C7 and T1 vertebrae, and below to the vertebral border (medial) of the scapula at the root of its spine. the rhomboid major muscle is found below the rhomboid minor muscle and attaches above to the spinous processes of T2 to T5 vertebrae, and below to the vertebral border of the scapula between the spine of the scapula and its inferior angle (Travell and Simons, 1999).
b. Innervation

The rhomboid major and minor muscles are innervated by the dorsal scapular nerve through the upper trunk of the brachial plexus from C5, and sometimes C4 roots (Travell and Simons, 1999).

The dorsal scapular nerve originates at the dorsal aspect of the anterior ramus of the fifth cervical nerve root close to the intervertebral foramen (Snell, 2004). It then descends downward and
backward where it pierces the middle scalene muscle and runs deep to the levator scapulae and rhomboids, supplying both (Gray, 1994).

c. Blood supply

The principal blood supply (Figure 2.5) to the rhomboid muscles is from dorsal scapular artery which may originate independently, from the third or less common second portion of the subclavian artery (Moore and Dalley, 2010). The dorsal scapular artery runs deep to the levator scapulae muscle to the medial border of the scapula from where it courses beneath the rhomboids along the medial border of the scapula to the inferior angle. It supplies the levator scapulae and rhomboids muscles (Gray and Carter, 2001).

Figure 2.5 Vascular supply around the shoulder girdle (Gray and Carter, 2001)
2.3.2 Functional unit

The rhomboid muscles work interchangeably with the levator scapulae and upper trapezius muscle to elevate the scapula (Travell and Simmons, 1999). It also works with the levator scapulae and latissimus dorsi during scapular rotation (Musculino, 2009). Basmajian and Deluca (1985), stated the rhomboid muscles work together with the middle trapezius muscles to assist in abducting the arm to 90° and in the early state of flexion in the arm at the shoulder joint.

Scapular adduction by the rhomboids and middle trapezius muscles is opposed directly by the serratus anterior and indirectly by the pectoralis major musculature (Travell and Simmons, 1999). The referral pattern of trigger points located in the rhomboid muscles must be distinguished from trigger
points in the surrounding supraspinatus, infraspinatus, levator scapulae and the scalene muscles (Musculino, 2009).

Figure 2.7 Referral pain pattern for the rhomboid muscles (Travell and Simons, 1999)

2.3.3 Activation and perpetuation of rhomboid muscle trigger points

The major activating and perpetuating factors for the rhomboid muscles are a chronic overload due to prolonged periods of working in a hunched over position with the head-forward posture, as well as postural overload due to an overly contracted pectoralis major muscle (Finando and Finando, 2005).
Holding the arm in abduction or flexion above 90° for a prolonged period of time, as seen when painting overhead can lead to trigger point formation. Other activating and perpetuating factor includes continuous stretching of the muscle due to a prominence of the scapula on the convex side in upper thoracic scoliosis (Travell and Simmons, 1999). Working at a computer screen that is positioned incorrectly or reading with the head inclined forward, car accidents, a cold draft on the neck and being stressed psychologically can also result in trigger point formation (Musculino, 2009). The weight of a strap from a heavy bag, purse or backpack carried on one shoulder can initiate and maintain rhomboid trigger point formation. Chest surgery (mainly a mastectomy), asymmetrical or rotated pelvis and leg length inequality can also perpetuate the formation of rhomboid trigger points (Starlanyl and Sharkey, 2013).

Overuse of the shoulder and arm may cause rhomboid muscle strains or spasm, mainly during overhead activities like serving a tennis ball or reaching to put an object on a high shelf (Smith, Padgett, Kaufman, Harrington, An and Irby, 2004). It can also occur from activities such as poor posture mainly from prolonged use of a computer. Rhomboid muscle strain causes pain in the upper back, between the scapula and the spine, a muscle spasm usually feels like a knot or tightness in the muscle (Rouzier, 2005).

2.3.4 Examination and location of rhomboid muscle trigger points

The rhomboid muscles are best examined for myofascial trigger points with the participants in a seated position with their arms hanging forward. This allows the muscle to be in a more relaxed position and also aids in abducting the scapula. The muscle fibre direction helps distinguish the rhomboids from the overlying trapezius muscle. The muscle is palpated for taut bands and the mid-belly region of each taut band examined for trigger point tenderness. The referred pain is reproduced by deep palpation (Travell and Simmons, 1999). The pain referral pattern concentrates along the vertebral (medial) border of the scapula between the scapula and the paraspinal muscles. The pain may resemble that of the levator scapulae muscle, however there is no neck component and neck restriction present (Travell and Simmons, 1999). Another examination method can be used, where the muscle is placed in a more relaxed position. This position also allows the lifting of the muscle off the scapula, making it easily accessible.
2.4 MYOFASCIAL PAIN SYNDROME

2.4.1 Introduction

The term “myofascial” is derived from the word “myo” which means muscle and “fascia” which means connective tissue. Myofascial pain is an intense, deep muscular pain referred from hypersensitive trigger points in the muscle bellies and their tendinous attachments (Gatterman, 2005).

The word syndrome is defined as: “A group of symptoms that collectively indicate or characterise a disease, psychological disorder or other abnormal condition” (Houghton et al, 2010). A pain syndrome is defined as neurogenic, musculoskeletal, sympathetic, visceral or psychogenic in origin (Raj and Paradise, 2004). So therefore a Myofascial Pain Syndrome consists of pain, muscle stiffness and decreased range of motion (Patrick, Stevens, Walker and Zempsky, 2013).

This syndrome presents with a persistent aching pain which is referred to a localised area of the body from trigger points in one or more muscles in that region (Baldry, 2001). It is said that the hallmark finding of this syndrome is the myofascial trigger point (Gerwin, Shannon, Hong, Hubbard and Gervitz, 1997). Myofascial pain syndrome is a very common disorder (Rudin, 2003).

This condition is diagnosed on physical examination by identifying the characteristics of the myofascial trigger point and eliminating other causes that may mimic this syndrome (Alvarez and Rockwell, 2002). The pain experienced does not correspond to a dermatomal, myotomal or sclerotomal pattern (Rachlin, 1994).

2.4.2 Myofascial trigger points

The presence of myofascial trigger points can cause a myofascial pain syndrome. These trigger points are defined as a hyperirritable area in a skeletal muscle that contains a hypersensitive taut band (Travell and Simmons, 1999). This trigger point is painful during compression, stretching and periods of overload (Fernandez-De-Las-Penas, Alonso-Blanco and Miangolarra-Page, 2007). A distinct pattern of referred pain and tenderness, altered muscle functioning and autonomic changes can accompany pain on compression of the trigger point (Travell and Simons, 1983).
According to Raj and Paradise (2004) a trigger point has the following characteristics:

- Taut palpable band
- Focal spot tenderness of a nodule in the taut band
- Painful and restricted range of motion
- Referred pain to a regional site upon activation of the trigger point
- Recognition of the pain by pressure on the tender nodule
- Reproducibility of pain pattern
- Visual or tactile identification of a local twitch response on activation of the trigger point
- Muscle weakness with the absence of muscle atrophy
- Pain or altered sensation on compression of the nodule
- Symptoms of autonomic dysfunction.

Myofascial trigger points are extremely common and nearly everyone suffers their effects at one time or another in their lives. Individuals in their adult years of maximum activity are most likely to suffer from the pain syndromes of myofascial trigger points. These trigger points are not life threatening despite their painfulness, but can and do alter quality of life (Travell and Simons, 1983). Trigger points are most commonly found in skeletal muscles, skeletal muscle tendons, joint capsules, and ligaments around the joints, periosteum and skin (Finando and Finando, 2005). Trigger points are activated after prolonged muscle contraction, environmental factors such as extreme cold or heat. Other factors that can lead to trigger point activation include emotional stress or muscle inactivation due to injury (Mense and Simons, 2001).

**2.4.3 The Mechanism of Myofascial Triggers Point Development**

The aetiology of trigger point formation remains unclear. The energy crisis theory and motor endplate hypothesis have become the most widely accepted theories (Gunn, 1997).

**a. Energy Crisis Theory**

The energy crisis theory was developed to better understand the pathophysiology of myofascial trigger points and is consistent with electrodiagnostic findings (Travell and Simons, 1999).
The energy crisis theory (Figure 2.8) was required to encompass and give reason that could explain the following:

- The absence of motor unit action potentials in the taut band of the trigger point when the muscle was at rest
- The sensitisation of nociceptors in the trigger point
- The fact that the trigger points are often affected by muscle overload
- The effectiveness of any therapeutic technique that restores the muscles full stretch length (Travell and Simons, 1999).

This theory was the first to attempt to explain the formation of myofascial trigger points (Bengtsson, Hendrikkson and Larsson, 1986; Simons 1997). It suggests that repetitive microtrauma, macrotrauma and an increased demand on the muscle will result in an excessive release of calcium ions which will result in increased contractile activity and shortening of the sarcomere. An increase in calcium can also result from a marked increase in acetylcholine from the motor end plate (Travell and Simons, 1983).

Acetylcholine is a neurotransmitter that binds to acetylcholine receptors on the skeletal muscle and acts to activate muscles (Martini, 2004). The increased acetylcholine released from a dysfunctional motor endplate, initiates abnormal depolarisation of the post-synaptic membrane. This results in an indefinite period of sustained contraction therefore accounting for the absence of action potentials in the motor unit of the taut palpable band. Sustained contraction will result in decreased blood supply to the muscle with resulting decreased oxygen supply (Simons, 1997).

The decreased oxygen and blood supply to the muscle causes a local energy crisis. This local energy crisis will stimulate the production of vasoactive products causing sensitisation of the local nociceptors (Travell and Simons, 1999). The products produced include bradykinin, histamine and prostaglandins and they play a role in the production of pain due to direct stimulation of sensory nerves (Huguenin, 2004).

Trauma to the sarcolemma and sarcoplasmic reticulum are mostly short term and therefore the increased contractile activity is only temporary. The increased calcium released due to increased
acetylcholine from the motor endplate is more long term and cause muscle contraction to be prolonged (Travell and Simons, 1999).

Figure 2.8 A modified diagram illustrating the energy crisis theory (Travell and Simons, 1999)

b. Motor endplate dysfunction hypothesis

This hypothesis refers to dysfunction in the region of the motor endplate as the foundation for myofascial trigger point formation. The term neuromuscular junction is used interchangeably with the term endplate. The term ‘endplate’ refers to the physical structure whereas the ‘neuromuscular junction’ refers to the functional significance of the structure (Travell and Simons, 1999).

When a motor endplate becomes dysfunctional, various mechanisms can cause it to exist as a trigger point. The sustained muscle contraction compresses local sensory nerves and reduces the axoplasmic transport of molecules that normally inhibit acetylcholine release (Hohmann and Herkenham, 1999; Gessa, Casu and Carta 1998). The sustained muscle contraction also compresses blood vessels reducing oxygen supply to the muscles. The decreased blood supply coupled with the increased metabolic supply cause a rapid depletion of adenosine triphosphate (Travell and Simons, 1999).
The energy crisis theory and motor endplate dysfunction hypothesis can co-exist together (Huguenin, 2004). This is due to a synaptic junction being present between the muscle cell and motor endplate.

c. Integrated trigger point theory

This is the combination of the two theories, it combines the electrophysiological and histopathological sources. This theory indicates that a trigger point is essentially a region of many dysfunctional endplates. And that each dysfunctional endplate is associated with a section of muscle fibre that is maximally contracted (a contracted knot) (Travell and Simons, 1999).

Needle electromyography (EMG) studies show that minute loci present within the myofascial trigger point produces characteristic electrical activity. Active loci are clustered within the myofascial trigger point and are found at the motor endplate zone (Hubbard and Berkhoff, 1993).

The spontaneous spikes in electrical activity are characteristic of active loci within trigger points and are recognised on electromyographers as normal “endplate” potentials. However, physiological experiments have shown that these potentials are not normal, but are a result of a grossly abnormal increase in ACh release by the nerve terminal. It can be seen that the contracted knot is located at an endplate and this is the cause of the endplate dysfunction (Travell and Simons, 1999).

Experiments have shown that the endplate potentials are as a result of an abnormal increase in acetylcholine (ACh) released by the nerve terminal (Travell and Simons, 1999). The EMG noise observed is believed to be the increased rate of release of ACh from the nerve terminal. A small amount of muscle activity is capable of producing action potentials that are propagated a small distance from the cell membrane of the muscle. This propagation may lead to the activation of a few contractile elements that may cause a small degree of muscle shortening (Huguenin, 2004).

A painful muscle condition develops which leads to a decrease in the flexibility of the muscle, decreased range of motion and eventually general disability (Gatterman, 2005).

The following hypothesis proposes a likely relationship between the dysfunctional endplate and the contracted knot. The hypothesis provides a model that can be used to design critical experiments with which to verify, refine, or refute the hypothesis (Travell and Simons, 1999).
The hypothesis is based on a continuous excessive ACh release from a dysfunctional motor nerve terminal into the synaptic cleft. Impaired cholinesterase function would potentiate the effect. The excessive ACh activates ACh receptors in the postjunctional membrane to produce greatly increased numbers of miniature endplate potentials. These potentials are so numerous that they superimpose to produce endplate noise, and a sustained partial depolarisation of the postjunctional membrane. The excessive demand for production of ACh in the motor nerve terminal would increase its energy demand. The increased activity of the postjunctional membrane and sustained depolarisation would impose an additional local energy demand. Increased numbers of subsarcolemma mitochondria and abnormal mitochondria have been noted repeatedly in past studies, this mechanism may be responsible for the presence of many ragged red fibres in muscles with characteristics that are compatible with the presence of myofascial trigger points (Travell and Simons, 1999).

The voltage gated calcium channels that releases calcium from the sarcoplasmic reticulum occurs due to the depolarisation of the T tubules at the triad where the T tubules communicate with the sarcoplasmic reticulum. The T tubule is part of the same sarcoplasmic membrane that forms the postjunctional membrane. The sustained depolarisation of this membrane is one mechanism that might account for the tonic increase in the release of calcium from the sarcoplasmic reticulum to produce that local sarcomere contracture of the contraction knots. The volume increase occupied by the contraction knot would explain why clinicians describe a palpable nodule at the trigger point together with a narrower taut band. This contraction process appears to occur in the immediate area of the endplate. A sustained release of calcium from the sarcoplasmic reticulum would increase the energy demand of the calcium pumps in the sarcoplasmic membrane that return the calcium into the sarcoplasmic reticulum. The sustained contracture of the sarcomeres in the contraction knot would greatly increase the local energy and oxygen demand (Travell and Simons, 1999).

The energy crisis in the vicinity of the endplate can cause the release of neuroactive substances that sensitize and modify the function of any sensory and autonomic nerves in the region. Small blood vessels, sensory nerves, and autonomic nerves are normally part of the same neurovascular bundle or complex that includes the motor nerve. Sensitization of local nociceptors can account for the tenderness of the trigger point, the referred pain originating at the trigger point, and the origin of a local twitch response. Experimental evidence suggest that autonomic nervous system activity can
strongly modulate the abnormal release of acetylcholine from the nerve terminal, completing what then becomes a self sustaining vicious cycle (Travell and Simons, 1999).

2.5 ISCHAEMIC COMPRESSION

Ischaemic compression or acupressure is the application of progressively stronger, firm digital pressure applied to a trigger point for the purpose of eliminating the point's tenderness (Travell and Simons, 1983).

Ischaemic compression involves the compression of the trigger points with direct pressure on the trigger points or by pinching the trigger point. Ischaemic compression is delivered by sustained pressure for a period of seconds to a minute on a trigger point to ease the tension in the taut bands of a muscle (Montanez-Aguilera and Valtuena-Gimeno, 2010). The purpose of the compression is to initially increase the blockage of blood to the area so that upon release there will be resurgence of blood. This washes away waste products (Fritz and Grosenbach, 2009). The pressure on the myofascial trigger point is continued until a state of tension relief is achieved which implies inactivation of the trigger point (Raj and Paradise, 2004). After the direct pressure, the practitioner lengthens the muscle to re-establish an optimal length-tension relationship (Fritz and Grosenbach, 2009).

Application of ischaemic compression can be done using fingers, thumbs or elbows. Ischaemic compression may induce some trauma to the tissue involved and minor bruising may occur (Hains, 2002). Bruising is minimised during treatment as pressure is only applied for short periods of time (Peterson and Bergmann, 2002).

Studies have shown short term positive effects in an application of ischaemic compression for 60 seconds (Fryer and Hodgson, 2005). This concludes that ischaemic compression increases active range of motion and decreases sensitivity of the myofascial trigger point (Montanez-Aguilera and Valtuena-Gimeno, 2010).

Ischaemic compression produces its effects by a combination of the following factors:

**Reflex Vasodilation** – by temporarily occluding the trigger point’s blood supply and then releasing the digital pressure from the trigger point is causes a reactive increase in blood supply. This flushes any inflammatory exudates and pain metabolites such as serotonin and histamine out of the muscle, breaks down scar tissue, and reduces muscle tone (Anderson, 1997). The extra blood flow through
the trigger point nourishes the muscle, the nerve endings are desensitised, and scar tissue is broken down so that the muscle fibres can move better. As the trigger point settles, there will be an accompanying decrease in referred pain (Travell and Simons, 1983).

**Ischaemic nerve block** – the digital pressure applied to the trigger point causes a temporary ischaemia thus depriving the area of oxygen, this causes a reduction in action potential and block noxious sensory afferent input to the higher levels of the nervous system (Anderson, 1997).

Ischaemic compression does not require any specialised equipment. It is also well tolerated by patients and it is not strenuous on the doctor. The digital pressure induced by the doctor should produce local or referred pain. Although local or referred pain is produced, the treatment should be tolerable. The patient should not feel the need to break the contact or protect the muscle by contracting it (Montanez-Aguilera and Valtuena-Gimeno, 2010).

### 2.6 THE ANATOMY OF THE RIBS

The thoracic region is the part of the body found between the neck and the abdomen. It also consists of the thoracic cage (rib cage). The thoracic cage consists of horizontal bars formed by the 12 pairs of ribs and costal cartilage, it’s also supported by the vertical sternum and the thoracic vertebrae (Moore and Dalley, 2010).

#### 2.6.1 Osteology of the ribs

The ribs are curved, flat bones that form most of the thoracic cage. They are light in weight and are highly resilient. Each rib has a spongy interior that contains bone marrow which produces red blood cells.

There are three type of ribs, and can be classified as typical or atypical (Moore and Dalley, 2010).

**The three different types of ribs**

- **True (vertebrocostal) ribs**: these are the 1st to 7th ribs and they attach directly to the sternum through their own costal cartilage.
- **False (vertebrochondral) ribs:** these are the 8th, 9th and usually the 10th ribs. Their cartilages are attached to the cartilage of the rib above them, therefore their connection with the sternum is indirect.

- **Floating (vertebral, free) ribs:** these are the 11th, 12th and sometime 10th ribs. The rudimentary cartilage of these ribs do not connect even indirectly to the sternum, instead they end in the posterior abdominal musculature (Moore and Dalley, 2010).

**a. Typical Ribs (3rd to 9th rib)**

These ribs have more or less identical features, each presenting a head, neck and corpus (comprising the tubercle and shaft with its angel) (Coetzee, 1987).

These ribs have a wedged-shaped head and two facets separated by the crest of the head, one facet for the articulation with the corresponding vertebrae and one facet for the vertebra superior to it. The neck of these ribs connects the head of the rib with the body at the level of the tubercle (Moore and Dalley, 2010). This tubercle is found at the junction of the neck and the body, it consists of a smooth round inferiomedial articular facet that articulates with the corresponding transverse process of the vertebrae to form the costotransverse joint. It also has a rough superolateral articular part that provides attachment for the costotransverse ligament which attaches to the tip of its own corresponding vertebra transverse process (Coetzee, 1987).

The body (shaft) of the rib is thin, flat, and curved, mostly at the costal angle where the rib faces anteriolaterally. The angle also demarcates the lateral limit of attachment of the deep back muscles to the ribs. The concave internal (medial) surface of the body has the characteristic costal groove which runs parallel to the inferior border of the rib, which always for some protection for the intercostals nerves and vessels (Moore and Dalley, 2010). The rounded upper border of this groove gives attachment to the innermost intercostals muscles. The superior border of each rib gives attachment laterally to the external intercostals muscles and medially to the internal intercostals muscles, its inferior border is sharp and gives attachment to the internal and external intercostals muscles (Coetzee, 1987).
b. Atypical Ribs (1\textsuperscript{st}, 2\textsuperscript{nd}, and 10\textsuperscript{th}-12\textsuperscript{th} ribs)

The 1\textsuperscript{st} rib is the broadest, shortest and most sharply curved of the seven true ribs (Moore and Dalley, 2010). Its rib head consists of a single round facet for articulation with the first thoracic vertebra. The neck is flattened from above downwards, the tubercle has the two usual facets and it coincides with the angle. At the tubercle this rib is bent, so that the head is directed downwards. The shaft has a single uniform curve without angles or twist, its surfaces look upwards and downwards, and its borders are directed medially and laterally. At the centre of the upper surface of the rib which interrupts the medial border is where the anterior scalene tubercle is found, its gives an attachment site for the anterior scalene muscle. The wide swallow groove for the subclavian vein is found anterior to the tubercle, while behind the tubercle is where you will find a similar groove for the subclavian artery with the lower trunk of the brachial plexus behind it (Coetzee, 1987).

There is a rough area in front of this groove that gives origin to a part of the subclavius, the large area behind the groove allows attachment for the scalene medius (Coetzee, 1987).

The 2\textsuperscript{nd} rib has a thinner, less curved body and is substantially longer than the 1\textsuperscript{st} rib. Its head has two facets for articulation with the bodies of T1 and T2 vertebrae, its main feature making it an atypical rib is the rough area on its upper surface. This rough surface is the tuberosity of the serratus anterior from which part of that muscle originates (Moore and Dalley, 2010). On the outer convexity of the 2\textsuperscript{nd} rib there is a rough eminence, to the posterior part of which the scalenus posterior is inserted (Coetzee, 1987).

The 10\textsuperscript{th} rib is typical rib except for the fact that it has a single facet instead of the double facet on its head, to allow for articulation to occur at a single vertebra (Coetzee, 1987).

The 11\textsuperscript{th} and 12\textsuperscript{th} rib each have a single facet on their heads, they are distinguished from the 10\textsuperscript{th} rib by their small size, and by the fact that they do not have tubercles or articular facets for the transverse processes of the vertebrae. They are distinguished from each other by the fact that the 11\textsuperscript{th} rib has traces of an angle and costal groove, the 12\textsuperscript{th} rib is shorter and is devoid of any characters, being merely a curved flat spicule (Coetzee, 1987).
2.6.2 Anatomy of the Costovertebral Joints

At each level of the thoracic vertebral column a pair of ribs is connected with the vertebra by two synovial joints, the costovertebral and the costotransverse joints (Kapandji, 1974).

The costovertebral joint is made up on the vertebral side by two costal facets, one on the superior border of the lower vertebra and the other on the inferior border of the upper vertebra. A solid angle is formed by these facets, whose base consists of the annulus fibrosis of the intervertebral disc. The corresponding facets of the head of the rib are slightly convex and also form a solid angle between each other which fits exactly into the angel formed by the costal facets of the vertebrae. The interosseous ligament attached to the head of the rib between the two articular facets and to the intervertebral disc, divides this joint which is surrounded by a single capsule into two distinct cavities, one superior and one inferior (Kapandji, 1974).

The costovertebral joint is reinforced by the radiate ligament of the rib head. It consists of three bands, the superior band and inferior band which attach to the adjacent vertebral bodies, and the intermediate band which will insert into the annulus fibrosis of the intervertebral disc (Kapandji, 1974).

2.6.3 Movement of the ribs at the costovertebral joints

The costovertebral joint and the costotransverse joint form a joint couple mechanically linked. The most common movement is rotation about an axis passing through the centre of each joint. An axis running through the centre of the costotransverse joint and the centre of the costovertebral joint, acts as a swivel for the rib which is thus suspended from the vertebral column at the two centre points. The direction of this axis with respect to the sagittal plane determines the direction of the movement of the rib (Kapandji, 1974).

With the lower ribs the axis lies almost parallel to the sagittal plane and so elevation of the rib increases the transverse diameter of the thorax. When the rib rotates about this axis, it will become less oblique and more transverse, and as a result its most lateral border moves laterally which will represent the increase in the transverse diameter of the thoracic outlet (Kapandji, 1974).

With the upper ribs the axis lies close to the frontal plane, therefore elevation of these ribs increases the anterioposterior diameter of the thorax. In effect, when the anterior end of the rib is raised, it is
displaced anteriorly. Therefore elevation of the ribs increases the transverse diameter of the lower thorax and the anterioposterior diameter of the upper thorax. In the midzone, the costovertebral joints have an axis which lies obliquely at an angle of 45° to the sagittal plane so that both diameters are increased (Kapandji, 1974).

The axes of the ribs allow three basic yet specific types of motion to occur: bucket handle, pump handle and calliper motion.

**Bucket-handle motion**: the handle of the bucket is fixed at both ends. The axis is closer to a sagittal plane in the lower ribs. One end of the bucket handle fixed at the vertebral end, the majority of the rib elevation occurs through upward excursion of the lateral position. This motion increases the transverse diameter of the rib cage (DiGiovanna, Schiowitz and Dowling, 2005).

**Pump-handle motion**: the term pump handle motion is derived from the similarity between the rib motion and that of an old fashion water pump. One end is fixed, and the free end describes an arc. The costovertebral-costotransverse axis of the upper ribs lies close to the coronal plane. As the ribs moves about the axis, they increase the anterioposterior diameter of that portion of the rib cage (DiGiovanna, Schiowitz and Dowling, 2005).

**Caliper motion**: the eleventh and twelfth ribs have only costovertebral articulations. Because there are no transverse process limitations, the motion of these ribs is calliper-like along a horizontal plane. This motion produces slight changes in both the anteroposterior and the transverse dimensions (DiGiovanna, Schiowitz and Dowling, 2005).

All ribs have both pump-handle and bucket-handle movements. Cephalic ribs have more pump-handle motion and caudal ribs have more of a bucket–handle movement. While the 11th and 12th ribs only allow for a calliper motion to occur (DeStefano, 2011).

### 2.7 COSTOVERTEBRAL SUBLUXATION SYNDROME

Normal vertebral rotation can become impaired due to the presence of a rib dysfunction, due to fact the ribs should move with the rotation transverse process. The intertransverse muscles may be a cause of dysfunction in the mid-to-lower thoracic area and can be determined by intertransverse palpation during lateral bending (Yao, 2010).
The etiology of costovertebral subluxation syndrome is due to the dysfunction of a rib from trauma or microtrauma, there may be a history of trauma to the chest, motor vehicle accident, sudden unguarded movement, a cough, a sneeze or it could be secondary to scoliosis (Gatterman, 2005). These dysfunctions are featured by the misalignment of the costal processes to the vertebral body and transverse processes independent of the vertebral motion-unit subluxation that is the primary, or this could be the misalignment of the costal process relative to the vertebral bodies and transverse processes. As a result of vertebral motion-unit subluxation which is secondary (Yao, 2010). The presence of painful, difficult, and/or restricted respiratory movements of the ribs, shearing stresses to the capsular ligaments and synovial, inducing a vertebral motion-unit subluxation and/or are contributory to the chronicity of a dysfunction, induction of a spinal curvature and/or contribute to the chronicity of the curvatures present, and may cause irritation of the sympathetic ganglia and rami communicantes (Yao, 2010).

The signs and symptoms are described as a dull or sharp stabbing episodic pain in the thorax aggravated by movement, reparatory coughing or sneezing (Gatterman, 2005). This pain manifests when the rib loses the ability to properly coordinate movement with the rest of the ribs and spine as part of a functional unit (Dalton, 2011). The pain is usually very rapid following a fall, push, a missed step, a stretch, sneeze or cough. Transient but sharp neuralgia angina or dyspnoea may be reported. With costovertebral subluxation syndrome the sharp pain in the inter-scapular region is accompanied by muscle spasm in the rhomboid musculature (Bahr, 2012). Intercostals muscle spasms, and tissue resistance are also found at the rib angle and overlying the vertebral attachment (Yao, 2010).

The examination findings of a patient presenting with costovertebral subluxation syndrome are palpable muscle guarding over the area of dysfunction (Dalton, 2011). Rib misalignment, site tenderness, a loss of joint pay at the costovertebral joint with failure of the rib to open and close, palpated at the rib angle are also identified (Gatterman, 2005). Exam findings will also show a unilateral asymmetry which can be palpated below the axilla, by noting one rib is shallower to the rib above or below which can indicate some type of detraction mechanism is involved (Yao, 2010). An asymmetry of the rib angle may also be noticeable on imaging techniques (Gatterman, 2005).

These findings indicate an alteration in the costovertebral or costotransverse articulation (dysfunction), and rotation alterations of the vertebral body to which the rib is attached. These detractions may also cause overlying muscle changes such as hypertonicity (Yao, 2010).
The posterior rib dysfunction occurring at the ribs costovertebral joint can result in a decrease in chest excursion, which can be determined by motion palpation of the thoracic cage during deep inspiration with the patient either standing or prone (Yao, 2010).

The assessment of a rib dysfunction is done by first, tractioning the patient’s skin of the lateral thorax towards the spine with a broad bilateral palmar contact. Then place both thumbs near the dorsal midline on the ribs which are undergoing examination. As the patient inhales deeply, note if both thumbs move equally. If the rib rises and the interspace opens, it is considered normal. If it remains down or down to some extent compared with the opposite side, it is considered "locked." Thumb motion restricted unilaterally suggests the side of dysfunction (Yao, 2010).

Springing the ribs P-A of the patient in a prone and relaxed position creates stress at the vertebral connections aggravates symptoms and causes an immediate apprehensive muscle-guarding response in sprain and subluxation (Yao, 2010). Once a rib dysfunction has been assessed and corrected, normal tone restored to overlying paraspinal muscles, and once these articular structures recover normal movement within the kinetic chain, deep paraspinal work in the area is painless and enjoyable (Dalton, 2011).

Management of costovertebral subluxation syndrome would consist of chiropractic adjustment therapy delivered to the costovertebral joint, soft tissue therapy to surrounding muscles, postural retraining and stabilization of excess motion with a rib belt if chronic (Gatterman, 2005). Bahr (2012), states that chiropractic adjustment therapy delivered to the costovertebral joints shows the most immediate result with relief of pain and overlying muscle spasms.

Differential diagnosis costovertebral subluxation syndrome could be a rib fracture, anomalies, tumours, metabolic disorders, degeneration, arthritides, and referred pain from visceral disorders (Gatterman, 2005).

2.8 THE CHIROPRACTIC VERTEBRAL SUBLUXATION COMPLEX

The chiropractic vertebral subluxation complex (VSC) is a theoretical model that describes dysfunction of a motion segment. It incorporates the complex interaction of pathological changes in
nerves, muscles, ligaments, local blood supply and connective tissue. This is in contrast to the mechanistic view of the chiropractic subluxation as a static misalignment of the articular facets (Gatterman, 2005). Literature also refers to a subluxation as a motion segment in which the alignment, physiological functions and/or movement integrity has been altered (Peterson and Bergmann, 2002).

The VSC is a conceptual model, not a definitive entity, existing only if all components are present (Gatterman, 2005). Gatterman (2005), states this subluxation complex encompasses pathological changes in the context of spinal biomechanics, biochemistry, physiology and anatomy. And that it generates symptoms such as pain and visceral or autonomic symptoms.

**Theoretical components**

There are five theoretical components to the VSC. All are interactive with one another yet each represents a distinct pathophysiological process that contributes to the overall picture (Gatterman, 2005).

1. **Neuropathophysiology** refers to the neural consequences associated with the subluxation complex resulting in muscle hypertonia, increased sympathetic facilitation and sensory dysaesthesias, or muscle atrophy, sympathetic atonia and anaesthesia (Gatterman, 2005).

2. **Kinesiopathology** refers to the disordered movement component of the VSC, encompassing articular hypomobility, hypermobility or loss of joint play (Gatterman, 2005). Such a functional imbalance may result in altered stress distribution within the vertebral motion segment. The mechanical irritation that ensues may result in neurogenic and non-neurogenic pain. Individual structural elements (muscle, disc, facet, ligaments or nerve) may experience concentration of local stresses within functional consequences and tissue-specific symptom production. The result is a state of dysfunction that can lead to local inflammatory or biomechanical changes. If neural elements become inflamed or compromised, the remote symptoms may also appear in the peripheral distribution of the nerve (Triano, 2001).

3. **Myopathology** may manifest as muscle hypertonicity which may be a postural compensatory mechanism or secondary to the neuropathophysiology component of the VSC (Gatterman, 2005).

4. A **histopathology component** refers to the cellular flow associated with the inflammatory process. Oedema may accumulate within the confines of the intervertebral foramen impeding
the flow of circulating fluids, compounding the neuropathophysiology component of the subluxation complex (Gatterman, 2005).

5. Biochemically, prostaglandin E-2, histamine, bradykinin, potassium ions, leukotriene B-4, 5-hydroxytryptamine and cytokines will accumulated in stressed tissue (Gatterman, 2005).

Mechanical and chemical irritants stimulates local nociceptors with a resultant barrage of afferent input to the dorsal horn of the spinal cord, resulting in somato-autonomic and somato-somatic reflexes via the autonomic and motor nervous system. Myofascial trigger points, muscle imbalance and spinal muscle deconditioning are hypothesised to result from reflex stimulation of the anterior and lateral horn cells of the spinal cord, resulting in reflex muscle spasm and sympathetic hyperactivity respectively. This represents just one of the many proposed cause and effects of the VSC (Gatterman, 2005).

As joint subluxation leads to kinesiopathology, it results in hypomobility of the motion segment. Structural elements (such as muscle) are compromised and irritated as a result of joint dysfunction. This can lead to neuropathology, and will eventually also lead to anatomical, physiological, biochemical and degenerative changes, inflammation and histopathology (Esposito and Philipson, 2005).

Therefore it is proposed that effective treatment of the myopathophysiololgy component of the subluxation complex in the form of rhomboid myofascial trigger points should therefore also include restoring segmental mobility (kineosiopathology) at the costovertebral joint underlying the rhomboid muscles in the thoracic spine.

This is further explain by the myofascial cycle which according to Peterson and Bergmann (2011), is a central complicating feature of many of the internal and external derangements of the motion segment is the induction of a self-perpetuating myofascial cycle of pain and muscle spasm. Articular soft tissues are richly supplied by mechanoreceptors and nociceptors, and traction or injury to these structures may lead to the initiation of local muscle splinting. With time the continued muscle contraction may lead to further muscle fatigue, ischemia, pain, and maintenance of muscle spasm and joint dysfunction (Figure 2.9).

High-velocity adjustments are suggested as treatments that may be effective in interrupting this cycle. Many theorises exist as to the mechanism by which the adjustment relieves muscle spasm. Both are
speculated to induce a reflex response in muscle, one through direct action on muscle and the other reflexly through joint distraction (cavitation). The direct muscle model speculates that quick traction and excitation of the golgi tendon organ, located in the muscle tendon junction, acts as brakes to limit excessive joint movement and possible injury by inhibiting motor activity. The concept is that adjustments induce a strong stretch on the muscle tendon complex, activate the golgi tendon organ, and induces reflex muscle relaxation (autogenic inhibition). High-velocity adjustments induce enough force to stimulate these structures and induce a burst of somatic afferent receptor activity. Based on this information, it seems reasonable to assume that joint and soft tissue mechanoreceptors and nociceptors have the potential to play a material role in the inhibition of muscle spasm and the interruption of painful myofascial cycles and joint dysfunction (Peterson and Bergmann, 2011).

![Figure 2.9 The self-perpetuating cycle of myofascial pain and muscle spasm (Peterson and Bergmann, 2011)](image)

**2.9 THE CHIROPRACTIC ADJUSTMENT**

The purpose of chiropractic is described as being the restoration of normal tone of the nervous system (Esposito and Philipson, 2005). The chiropractic adjustment is the primary treatment tool used by Chiropractors as a form of spinal adjustment therapy, which is joint specific (Haldeman, 1992; Leach, 2004; Gatterman, 2004). The chiropractic adjustment reverses dysfunctions and corrects
neurological dysfunction (Esposito and Philipson, 2005). A movement restriction is referred to as a dysfunction of the joint, joint locking or joint blockage (Gatterman, 2005).

A chiropractic adjustment is defined as the application of a high velocity, low amplitude thrust designed to move a joint beyond its physiological limit into the paraphysiological space without exceeding the limits of anatomic integrity and motion often resulting in an audible release (Eriksen, 2004; Gatterman, 2005; Haldeman, 1992). This is thought to affect the movement component of the subluxation complex directly with an indirect effect on the other components of the complex, this allows for restoration and normalisation of joint with the ultimate goal to promote homeostasis of the body (Gatterman, 2005).

Peterson and Bergmann (2002), describes joint adjustment therapy as any procedure where the hands are used to mobilise, adjust, manipulate, apply traction or influence the joints of the body with the aim of influencing the patient’s health. They also state that the main objective of the chiropractic adjustment is to improve health and function through the alleviation of musculoskeletal pain, aberrant joint alignment and function. The chiropractic adjustment effects the joint dysfunctions, muscle spasms, periarticular fibrosis and adhesions.

![Figure 2.10 Sandoz’s model for joint range of motion (Esposito and Philipson, 2005)](image)
Sandoz (1970) describes joint range of motion as four zones and two barriers (Figure 2.10):

- Zone one represents active range of motion and is produced by active muscles
- Zone two represents passive range of motion and extends to the elastic barrier of resistance. This is where joint play is examined
- Zone three represents the paraphysiological space. This space extends from the elastic barrier of resistance to the limit of anatomical integrity. Movement into this space will result in joint cavitation
- Zone four represents pathological movement. This is movement of the joint past the limit of anatomical limit of integrity (Esposito and Philipson, 2005).

The audible release or “crack” heard during an adjustment occurs when the joint is moved past the elastic barrier of resistance into the paraphysiological space. Joint injury occurs when the joint is moved past the physiological space to the limit of anatomic integrity. This will result in injury to the ligaments and joint capsule (Peterson and Bergmann, 2002).

The audible release does not necessarily signify that a successful adjustment has been accomplished, but only that an articulation has been opened and sub-atmospheric gas passed out of occlusion to produce a bubble that pops (Oppenheim, Spitzer and Segal, 2005).

Esposito and Philipson. (2005), describe three events that occur when passing through the elastic barrier of resistance into the paraphysiological space:

- Sudden separation of joint surfaces
- An audible cracking sound
- The presence of a radiolucent space in the joint.

There has been a resurgence of interest in the thoracic spine from a clinical perspective. However, much of the clinical theory particularly in relation to the effects of chiropractic adjustment on thoracic spine is untested (Herzog, Conway, Zhang, Ga, Guimaraes, 1995).

2.10 THE EFFECTIVENESS OF CHIROPRACTIC ADJUSTMENTS

Chiropractic adjustment therapy has been widely recognised and successfully used as a conservative treatment modality for spinal dysfunction and pain (Schiller, 2001).
The physical effects of an adjustment are represented by a cavitation, joint and muscle mechanoreceptor stimulation, increased active and passive range of motion, breaking up articular adhesions and it is hypothesized to overcome the elastic barrier of resistance to cause separation of articular surfaces (Gatterman, 1990).

The neurophysiological effects of an adjustment are pain inhibition, muscle relaxation and stimulation of the autonomic system (Gatterman, 1990). The different autonomic nervous system responses include vasomotor changes, pseudo motor activity and changes in visceral regulation control. This is due to the stimulation of the nerves exiting the intervertebral foramen which results in the spinal pain gate mechanism being inhibited, which leads to pain inhibition and muscle relaxation of nerves related to areas that have been adjusted (Bergman, Peterson and Lawrence, 1993, Esposito and Philipson, 2005).

A number of biomechanical changes produced by the vertebral movement during a spinal adjustment have been hypothesized. The mechanical force introduced during the adjustment may directly alter segmental biomechanics by releasing trapped meniscoids, releasing adhesions or by reducing distortions in the annulus fibrosis of the spinal column. The mechanical changes elicited by an adjustment can provide sufficient energy to restore a segment to a lower energy level, thus intern reducing mechanical stresses or strains on soft tissue overlying the segment and to allow for restoration of joint mobility and joint play (Pickar, 2002).

2.11 REFLEXOGENIC EFFECTS OF THE CHIROPRACTIC ADJUSTMENT

Changes in muscle tone are an important feature of the subluxation complex. Muscle hypertonicity can perpetuate and prolong joint restriction. It is hypothesised that an adjustment may be able to change the muscle spasm cycle (Esposito and Philipson, 2005).

According to Wyke (1984), joint dysfunction is often associated with muscle spasm as a result of increased motor output. Chiropractic adjustments may prevent trigger point formation by reducing motor output. It has even been suggested that chiropractic adjustments may convert active trigger points into latent trigger points.

A chiropractic adjustment has the potential to normalise joint mechanics and terminates the altered neurogenic reflexes associated with joint dysfunction. The adjustment may reduce strain on the joint,
reduce muscle spasm and stop nociception from these tissues to the spinal cord (Peterson and Bergmann, 2002).

### 2.11.1 The reflex theory of the chiropractic adjustment

Of particular interest to this study is the reflex theory of the adjustment. The chiropractic adjustment is considered a biomechanical aberration within a vertebral motion segment. Such a dysrelationship is proposed to stimulate sensory receptors in spinal and paraspinal muscles, ligament and joints capsules, the impulses of which presumably activate neural reflex centres within the spinal cord or higher centres. These receptors are responsive to mechanical (position, motion and tissue tension), inflammatory (pain) and temperature changes. Impulses generated from these receptors then cause somato-somatic responses resulting in muscle spasm, or somato-visceral responses in sympathetic and parasympathetic nerves resulting in autonomic phenomena (Haldeman, 2000).

These reflexes activate central reflex pathways and specific somato-somatic reflexes in experimental animals. It has been shown that these reflexes may be brought about by chiropractic adjustment at the spinal level (Haldeman, 2000). This tends to support why chiropractors palpate for a tight muscle bundle as one indication of where to apply the adjustment (De Vocht, Pickar and Wilder, 2005). But there remains minimal evidence that these reflexes persist for sufficient time to bring about long-term relief (Haldeman, 2000).

With any high velocity chiropractic adjustment the force applied to the segment when its related muscles are in secondary or protective spasm, produces muscle relaxation if the impulse succeeds in removing the focal stimulus for the reflex (Schafer and Faye, 1990).

Reflex relaxation of hypertonic muscles by sudden stretching is a theory of particular interest to the present study. It has been believed that adjustments can normalise abnormal muscle tone, which is often attributed to the stimulation of inhibitory afferents to the dorsal horn from mechanoreceptors (Evans, 2002). However, the evidence regarding the effect of the chiropractic adjustment on somatomotor activity reveals both excitatory and inhibitory effects (Pickar, 2002).

Several studies appear to provide evidence that chiropractic adjustment produces a decrease in resting paraspinal electromyographic (EMG) activity (Fryer, Morris and Gibbons, 2004). De Vocht et
al (2005), investigated the immediate effects of the spinal adjustment on EMG activity in areas of localised tight muscle bundles in the lumbar paraspinal muscles in low back pain patients.

Resting EMG activity levels within the tight muscle bundles immediately decreased in the majority of cases after receiving a chiropractic adjustment to the associated vertebral dysfunction (De Vocht et al, 2005; Fryer, Morris and Gibbons, 2004).

In one symptomatic patient with spontaneous muscle activity in the thoracic spine, Suter, Herzog, Conway and Zhang (1994), observed reduced paraspinal EMG activity within 1 second after a thoracic spinal adjustment. This indicates the effect of muscle relaxation following a chiropractic adjustment to muscles overlying subluxated joints in the thoracic spine.

However according to Evans (2002), this is unlikely as he is of the opinion that sudden stretch of musculature produced by adjustments will excite rather than inhibit the motor neuron. He cited a study on the effects of spinal high-velocity, low amplitude thrust manipulation which was performed on the facets joints of anaesthetised cats at levels C3/C4 were distracted and an increase in the EMG activity of the cervical and upper limb musculature was recorded. This is likely a protective role initiated by the capsule mechanoreceptors and affected by the muscles, elicited by way of relax arcs. A synergy of the passive (capsule-ligamentous) and active (muscular) joint restraints is therefore achieved. Due to the rich innervations of the capsules of humans, it is reasonable to suggest that a similar synergistic relationship occurs in humans (Evans, 2002).

Lastly, Dunning and Rushton (2008), demonstrated an excitatory effect of a C5/C6 chiropractic adjustment on the motor activity of the biceps brachi muscle, which is not attached to the spine at its origin or insertion. The C5/C6 segment corresponds with the segmental innervations of the biceps brachi muscle. The resting EMG activity ipsilateral to the side of the application of the chiropractic adjustment was higher that the contralateral side, but EMG increases from the resting state were observed bilaterally (Dunning and Rushton, 2008).

The studies therefore indicate that chiropractic spinal adjustments can both increase the excitability of motor pathways in the spinal cord and depress the inflow of sensory information from muscle spindles (Pickar, 2002).
The pain relief theory of the chiropractic adjustment

The pain relief theory proposes that the chiropractic adjustment can bring about hypoalgesia by stimulating spinal structures which reduces muscle pain thresholds via central facilitations. There is now evidence that patients undergoing treatment with spinal adjustments describe pain relief that exceeds that obtained with other treatment methods, however the observed changes may also be due to psycho-physiological mechanism and may not be due to effects of the adjustment on spinal pain (Haldeman, 2000).

Ruiz-Sáez, Fernandez-de-las-Penas, Blanco, Martinez-Segura and Garcia-Leon (2007), investigated the immediate effect of a single cervical spine adjustment on the pressure pain threshold of latent trigger points. The treatment group received an actual adjustment showed a trend towards an increase in pressure pain threshold. The control group received a sham adjustment, and the subjects in this group showed a trend towards a decrease in pressure pain threshold levels.

Vernon, Aker, Burns, Vilijakaanen and Short (1990), measured changes in the pressure/pain threshold after spinal adjustment using the pressure algometer. In a case study, spinal adjustment increased the average pressure/pain threshold of six tender spots in the neck region by approximately 50% (from 2 kg/cm² to 2.9 kg/cm²) (Pickar, 2002).

The neurophysiological mechanism by which spinal adjustments therapy may evoke changes in pressure pain threshold in myofascial trigger points remain to be fully elucidated. Reduction of chemical algogenic mediators, activation of segmental inhibitory pathways and or central descending inhibitory pathway activation mechanisms have been suggested (Ruiz-Sáez et al., 2007).

2.12 CONCLUSION

In this chapter we have addressed the literature that is the most pertinent to this study. Emphasis has been placed on the anatomy and physiology of skeletal muscle; the anatomy and myofascial trigger points of the rhomboid muscles, myofascial pain syndrome, costovertebral subluxation syndrome, and the anatomy and biomechanics of the thoracic ribs.

The benefits from chiropractic adjustments have been discussed previously. This justifies the use of chiropractic adjustments as part of the treatment protocol for inter-scapular pain caused by rib dysfunction seen in costovertebral subluxation syndrome. Chiropractic adjustments have positive
effects on the structures directly related to the joints causing a increase in joint mobility, improved blood circulation, reduction in the perception of pain, improvement of muscle hypertonicity and a decrease in trigger point severity. With regards to myofascial trigger points, motor dysfunction is a feature of myofascial trigger points (Dommerholt, Gerwin and Shah, 2004). This highlights the relationship between the inter-scapular pain and myofascial trigger points.

In this chapter, ischaemic compression has been discussed with regards to effects and mechanism of use on myofascial trigger points. Ischaemic compression is safe and effective in reducing the severity and tenderness of the myofascial trigger point (Fernandez-De-Las-Penas, Alonso-Blanco, Alguacil-Diego and Miangolarra-Page, 2006).

In a study by Segeel, Hay and Lawson (2008), on the effects of chiropractic adjustment therapy and dry needle therapy in the treatment of inter-scapular pain due to active rhomboid minor and major trigger points. The purpose of this study was to determine the effectiveness of chiropractic adjustment therapy against soft tissue therapy (in the form dry needling) in the treatment of inter-scapular pain. The results of the study indicated that both groups showed effectiveness, whether it was chiropractic adjustment therapy or chiropractic adjustment therapy combined with soft tissue release. Both groups showed a decrease in inter-scapular pain by the end of the study.

The study will give a better understanding of the effectiveness of treating inter-scapular pain by costovertebral adjustment therapy versus ischaemic compression of rhomboid myofascial trigger points.
CHAPTER THREE – METHODOLOGY

3.1 INTRODUCTION

This chapter serves to give a detailed explanation on how this study was conducted and carried out.

3.2 STUDY DESIGN

This study was a quantitative comparative study with random group allocation.

3.2.1 Sample size and selection

A sample group of thirty participants who suffer from inter-scapular pain were recruited to participate in this study. Participants between the ages of 18-45 who met the inclusion criteria were randomly divided into three groups of ten participants each ensuring equal male to female and age ratios. Participants used for this study were recruited by advertisements (Appendix A) placed in the private sector and around the University of Johannesburg Doornfontein campus. The study was conducted at the University of Johannesburg Chiropractic Day Clinic.

Prior to inclusion in the study, the participants had to undergo an examination done by the researcher. The examination included a full case history (Appendix H), a full physical examination (Appendix I), cervical regional examination (Appendix J), McGill Pain Questionnaire (Appendix E) and completing the Numerical Pain Rating Scale (Appendix F).

Participants were included or excluded from the study based on the following results:

3.2.2 Inclusion criteria

To be included into this study, participants had to comply with the following inclusion criteria:
• Either gender
• Between the ages of 18-45 years of age, because at 50 years of age there is a risk of developing fractures related to osteoporosis which is a contra-indication for chiropractic adjustment therapy (Bickley, 2007).

• Rhomboid trigger point diagnosis according to the follow criteria:
  - The presence of a hyperirritable palpable nodule within a taut band of muscle (Travell and Simons, 1999).
  - Spot tenderness over palpable nodule (Travell and Simons, 1999).
  - When pressure is applied over spot tenderness, characteristic referral pain will arise. Referred pain along the vertebral border of the scapula between the scapula and paraspinal muscles (Travell and Simons, 1999).

• Costovertebral syndrome diagnosis according to the following criteria:
  - Sharp and stabbing pain, accentuated by deep inspiration, coughing and sneezing (Gatterman, 2004).
  - Continues soreness at the costovertebral angle which can radiate to the lateral and anterior chest wall (Gatterman, 2004).
  - An absence of joint movement of the ribs determined by motion palpation (Gatterman, 2004).
  - Prominent rib heads and localized tenderness which may indicate rib dysfunction (Gatterman, 2004).

### 3.2.3 Exclusion criteria

Participants were excluded if they have the following:

• Chiropractic adjustment contra-indications (Appendix D).
• Participants in other forms of chiropractic or medical treatment (during the duration of the study), which may interfere with the study, for example the use of muscle relaxants, other medication and fractures of the ribs.
All participants who met the inclusion criteria were accepted into the study and were required to read the participant information form (Appendix B) and sign the consent form (Appendix C).

### 3.2.4 Random Group Allocation

Participants who met the inclusion criteria were randomly allocated to Group 1, Group 2 or Group 3 by picking a number i.e. 1, 2 or 3 out of a hat, this would identify the group selected by the participant. The thirty participants were divided into three groups consisting of ten participants each. There were 5 females and 5 males in each group. Group 1 and Group 3 received chiropractic adjustments to the rib restrictions found in the upper thoracic spine T1-T5 levels as determined by motion palpation.

Group 2 received ischaemic compression to the rhomboid muscle trigger point only. Group 3 received both chiropractic adjustments to the rib restrictions and ischaemic compression to the rhomboid muscles.

### 3.3 TREATMENT APPROACH

#### 3.3.1 First and follow up visits

Each participant in each group was treated six times out of a total of seven visits over a period of three weeks. Each participant was treated twice a week with at least two days between treatments for three weeks. The third week consisted of three visits with the last visit used only for data collection.

**a. First (Initial) visit**

On the first visit the participant was given a participant information form (Appendix B) and was required to sign the consent form (Appendix C). When suitability for the study was determined the participant was required to complete McGill Pain Questionnaire (Appendix E) and the Numerical Pain Rating Scale (Appendix F). A full case history (Appendix H), a full physical examination (Appendix I), cervical spine regional examination (Appendix J) and motion palpation of the costovertebral joints of the upper thoracic spine were performed after which the SOAP note (Appendix K) was completed. The algometer was used to determine the severity of the rhomboid muscles trigger points (Appendix
The participants received chiropractic adjustments to restrictions of the upper costovertebral joints found at T1-T5 as determined by motion palpation, ischaemic compression of the rhomboid muscles, or participants will receive a combination treatment of chiropractic adjustments and ischaemic compression.

b. Follow-up visits

Participants were required to complete the McGill Pain Questionnaire (Appendix E) and the Numerical Pain Rating Scale (Appendix F) on the fourth and seventh visits. Algometer readings were also taken on the fourth and seventh visits (Appendix G). The costovertebral joint restrictions were re-accessed via motion palpation, and corrected with chiropractic adjustment therapy on the second to sixth visits for participants in Group 1 and Group 3. Myofascial trigger points were also re-accessed via digital palpation and ischaemic compression for 60 seconds was applied to the trigger points on the second to sixth visits for participants in Group 2 and Group 3.

3.3.2 Assessment

Each participant had to undergo a myofascial trigger point examination of the rhomboid muscles. There are three trigger points found in the rhomboid muscles (refer to Figure 2.7). The myofascial trigger point of rhomboid minor is found near the medial border of the scapula above the root of the scapula spine. The myofascial trigger points of rhomboid major are found along the medial border of the scapula, from below the root of the scapula spine to the inferior angle of the scapula.

3.3.3 Digital Palpation of Rhomboid Trigger Points

The participant was placed in a position to allow the rhomboid muscles to lengthen to a point of observable increase in resistance to movement. With the participant in this position the normal muscle fibres are still slack, but the fibres of any taut band are placed under further tension, which allows them to be easily identified from normal fibres. Cross fibre palpation was used to identify any taut bands, this fibre examination was through flat palpation. If a taut band was identified, palpation along the taut band was used to search for the focal point and, when this point was identified the
participant was asked if the point was tender under pressure and whether it reproduced the pain (Travell and Simons, 1999).

3.3.4 Intervention

a. Ischaemic compression

With Group 2 and Group 3 participants when a trigger point in the rhomboid muscle had been determined via specific trigger point location and flat palpation, the trigger point was compressed via ischaemic compression. The thumb was then placed using flat palpation on the trigger point, either hand could have been used (Travell and Simons, 1999). The researcher found it best to use the left hand for a trigger point located in the left rhomboid muscle and the right hand for a trigger point in the right rhomboid muscle.

The participants in Group 2 and Group 3 received ischaemic compression over the rhomboid muscle trigger point which was found on flat palpation examination.

Ischaemic Compression was applied via sustained digital pressure, with a thumb contact, for a period of a minute.

An algometer reading was taken on the first, fourth and seventh visits in order to evaluate the trigger point severity.

Ischaemic compression was done on six out of the seven visits, ischaemic compression with flat palpation over the trigger point reproducing the pain referral pattern. The ischaemic compression was held for 60 seconds (Fryer and Hodgson, 2005).

b. Chiropractic adjustment methods for the costo vertebral joints of the thoracic spine.

Participants in Group 1 received chiropractic adjustments to restrictions found at the costo vertebral joints found at levels T1-T5 as determined by motion palpation, by assessing the bucket-handle motion (Peterson and Bergmann, 2002). The participants in Group 3 received a combination of ischaemic compression over the rhomboid muscle trigger point after receiving adjustments to restrictions found at the costo vertebral joints found at levels T1-T5 as determined by motion...
palpation. The chiropractic adjustment techniques used on these participants for costovertebral joint restrictions were the thenar costal drop, the hypothenar costal push and the covered-thumb push (Peterson and Bergmann, 2002).

3.4 SUBJECTIVE DATA

Each participant was required to complete two questionnaires on the first visit. These questionnaires were: the McGill Pain Questionnaire (Appendix E) and the Numerical Pain Rating Scale (Appendix F). Both were completed again before treatment on the fourth treatment visit and on the seventh final data collection visit. The McGill Pain Questionnaire and the Numerical Pain Rating Scale were used to verify any subjective changes in the participant's observation regarding their neck pain specifically during the three week research study.

3.4.1 The McGill Pain Questionnaire

The McGill Pain Questionnaire (MPQ) provides a quantitative profile of three aspects of pain: sensory- discriminative, motivational-affective, and cognitive-evaluation. Originally it was used for pain evaluation but can also be used as a diagnostic tool. It is the leading instrument for describing the various dimensions of pain. It consists of different adjectives describing pain arranged in different sub-classes describing various aspects or types of pain. The participant only chooses the words that describes their feeling or sensations at that moment, and is relatively easy to follow and score. There is also a short-form McGill Questionnaire that has lesser descriptors of pain and that was used in this study (Appendix E). It has a total of 15 descriptors and each descriptor is ranked on an intensity scale with 0 being none and 3 being severe. This questionnaire is sensitive to treatment-related changes in the pain report, and the test-retest reliability is high (Maire, 2002).

3.4.2 Numerical Pain Rating Scale

The Numerical Pain Rating Scale (Appendix F) is a scale from 0-10 used to determine the participant's pain level. It was completed before the treatment on the first and fourth visits and on the seventh data collection visit. Participants were asked to grade their pain level experienced at that particular moment on a scale of 0 to 10. Zero indicating “no pain” and 10 indicating the “worst imaginable pain”. This scale is considered valid and reliable by Bolton and Wilkinson (1998) and
Yeomans (2000). The scores of the questionnaire remained confidential. The participant’s scores were not given to them nor did they see their answers from the previous questionnaire before answering the next.

3.5 OBJECTIVE DATA

Objective data was measured using the hand-held algometer. Objective data was collected before treatment on the first and fourth visits and during data collection on the seventh final visit. It measures the minimum pressure that causes pain (Rachlin, 1994).

3.5.1 The Algometer

The pressure algometer (Figure 3.1) is known as the pressure threshold meter. It is a force gauge, spring operated plunger calibrated in kg/cm² fitted with a rubber disc of 1 cm² surface. The algometer is designed to determine the pressure threshold and tissue compliance (Fischer, 1987). Pressure threshold is defined as the minimum pressure needed to cause pain (Rachlin, 1994). The algometer is a useful device used in practice to determine the severity of a muscle trigger point. It was proven valid and reliable by Fischer (1987) when a comparison of corresponding muscles on opposite sides failed to elicit significant differences.

Figure 3.1 A replica model of the pressure algometer used in the study (www.sciencedirect.com)
The pressure algometer reading of the rhomboid muscle trigger point was measured. Initially, flat palpation was used to locate the trigger point and reproduce the pain referral pattern of the rhomboid muscle. The algometer was then placed over the same trigger point in the rhomboid muscle at an angle of 90° to the skin. Pressure was then applied downwards on the trigger point until the participant indicated the presence of pain threshold. The algometer was removed and the reading in kg/cm² was noted. The readings were taken before treatment on the first and fourth visits and on the seventh final data collection visit.

3.6 STATISTICAL ANALYSIS

The subjective and objective data was collected by the researcher over the study period. The results were given to the statisticians at the STATKON department at the University of Johannesburg. If assumptions for normality per group using the Shapiro-Wilks Test were met: Inter-group analysis (the comparison between groups) was performed using either the One way ANOVA test if normality was present or the Kruskal-Wallis test if normality was not met. The Kruskal-Wallis test would indicate a difference between the groups, the Mann-Whitney Test was then used to establish where the difference occurs. Intra-group analysis was done using the Friedman test. This indicated whether or not there were significant changes over time. The Wilcoxon Signed Rank test determined where the differences were noted.

3.7 ETHICAL CONSIDERATIONS

All participants that partook in this particular study received a participant information form (Appendix B) and were requested to read and sign the participant consent form (Appendix C) specific to this study. The information and consent forms outlined the names of the researcher, purpose of this study and benefits of partaking in this study, participant assessment and treatment procedures. Any risks, benefits and discomfort pertaining 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 by ensuring their anonymity (no names or data) and confidentiality (standard doctor confidentiality) while compiling the research dissertation. The participants’ files were stored in a strong room at the University of Johannesburg Chiropractic Day Clinic. The participants were informed that their participation was voluntary and they were free to withdraw from the study at
any stage. Should the participant have any questions, they were explained by the researcher. The participants were requested to read the information form and sign the consent form, signifying 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, benefits included decreased inter-scapular pain, decreased muscle spasm found within rhomboid musculature. Some participants experienced slight pain and discomfort of the thoracic spine after the chiropractic adjustments and ischaemic compression, which was normal.

Participants were referred if any problems arose.
CHAPTER FOUR – RESULTS

4.1 INTRODUCTION

This chapter was used to present the results obtained during the clinical trial of this study. All the objective and subjective data was statistically analysed and plotted on line graphs, in order to determine whether there were any statistically significant improvements in any of the three groups, as well as to determine whether there were any significant differences between the groups. The efficacy of the treatment protocol was also determined by the degree to which there was a significant difference in the subjective findings. It was, therefore, measured in all three groups using the McGill Pain Questionnaire and the Numerical Pain Rating Scale, the efficacy was determined by the degree to which the participant’s disability percentage differed during the treatment times.

The main objective measure was the hand held pressure algometer, to determine the pain threshold that causes pain in the participant over the rhomboid trigger points at the end of each treatment protocol. This would determine which treatment protocol would be most beneficial.

The statistical results only represent a small group of subjects and therefore no assumptions can be made with respect to the population as a whole.

The statistical analysis was conducted on a 95% confidence level. The probability level (p-value) was set at $p \leq 0.05$. If the p-value is $\leq 0.05$, a statistical significant finding is observed. If the p-value is $> 0.05$, it can be stated that there is no statistical significant finding present.

4.2 DEMOGRAPHIC DATA ANALYSIS
Table 4.1 Demographic Data within the sample of 30 participants

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean Age in Years</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adjustment Group</td>
<td>23.70</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Ischaemic Compression Group</td>
<td>23.90</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Combination Group</td>
<td>22.30</td>
<td>5</td>
<td>5</td>
</tr>
</tbody>
</table>

4.2.1 Gender Tests

The Gender Cross Tabulation and Chi-Square tests were performed to determine the equal distribution of males and females throughout the research trial period.

Table 4.1 shows the mean age of the participants in each group. It also shows that there was an equal distribution of males and females within the sample and within each group. The Chi-Square Tests indicate that the $p = 1.00$, therefore $p > 0.05$ and no statistical significant difference was noted.

4.2.2 Age Tests

Participants between the ages of 18 and 45 were used for the study. Equal age ratios were ensured in each group with the mean age of the participants being 23.30 years. The minimum age was 20 and the maximum was 28.

There was no statistically significant difference between the ages of groups with $p = 1.00$ ($p > 0.05$).

4.3 SUBJECTIVE DATA ANALYSIS

The subjective data of this study included the Numerical Pain Rating Scale and the McGill Pain Questionnaire.
The Friedman test was used for the intragroup analysis, this test was used to form a comparison of each group individually over time. Further intragroup analysis was required if the values indicated to be statistically significant by using the Wilcoxon Signed Rank test.

The Bonferroni Adjustment was added to the intragroup analysis, this would test the smallest p-value of each variable against a significant level of \(0.05 ÷ 2 = 0.025\), it also tests the largest p-value of each variable against a significant level of \(0.05 ÷ 1 = 0.05\).

The use of the Bonferroni Adjustment is to lower the p-value in order to prevent data from incorrectly appearing significant. This also ensures accuracy of the data.

The Kruskal-Wallis test was also used for intergroup analysis, this test was used to determine whether there is normal distribution between groups.

### 4.3.1 Numerical Pain Rating Scale

**a) Intragroup Analysis of the Numerical Pain Rating Scale**

<table>
<thead>
<tr>
<th>Non-parametric Friedman Test for Intragroup Analysis of the NPRS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group</strong></td>
</tr>
<tr>
<td>Adjustment Group</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Ischaemic Group</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>
The mean value of the adjustment group was 6.10 for the first reading, 3.80 for the second reading and 1.30 for the third reading. An improvement of 78.70% was noted between the first and third readings. The \textbf{p-value} = 0.000 (p ≤ 0.05) which indicates statistically significant differences.

The ischaemic compression group had a mean value of 5.70 for the first reading, 3.60 for the second reading and 1.60 for the third reading. This indicated a difference of 71.90% between the first and third readings. The \textbf{p-value} = 0.000 (p ≤ 0.05) indicating that there were statistically significant differences.

The combination group had a mean value of 6.50 for the first reading, 4.40 for the second reading and 2.00 for the third reading. An improvement of 69.23% was noted between the first and third readings. The \textbf{p-value} = 0.000 (p ≤ 0.05) therefore statistically significant changes have also occurred.

Table 4.2 illustrates that when the Friedman test was used when comparing the NPRS scores, the \textbf{p} ≤ 0.05. All three groups indicated to have statistically significant differences.

It was necessary to establish when these changes occurred within each of the three groups as there were statistically significant changes. The non-parametric Wilcoxon Signed Ranks test was used to determine if the changes occurred during the first (visit 1) and second (visit 4) readings or between the second (visit 4) and third (visit 7) readings.

The Bonferroni Adjustment was used. In this instance, the Bonferroni Adjustment tests the smallest p-value against a significance level of 0.05 ÷ 2 = 0.025 and the largest p-value against a significance level of 0.05 ÷ 1 = 0.05.
Table 4.3 Non-parametric Wilcoxon Signed Ranks Test for the Intragroup Analysis of the NPRS

<table>
<thead>
<tr>
<th>Visit Number</th>
<th>Adjustment Group</th>
<th>Ischaemic Compression Group</th>
<th>Combination Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>p-value between visit 4 and visit 1</td>
<td>0.005 thus ≤ than 0.025</td>
<td>0.005 thus ≤ than 0.025</td>
<td>0.004 thus ≤ than 0.025</td>
</tr>
<tr>
<td>p-value between visit 7 and visit 1</td>
<td>0.004 thus ≤ than 0.025</td>
<td>0.005 thus ≤ than 0.025</td>
<td>0.004 thus ≤ than 0.025</td>
</tr>
</tbody>
</table>

Table 4.3 indicates that the three groups showed statistically significant differences for the NPRS between visits 1 and 4 with the p-values for the adjustment group being 0.005 (p ≤ 0.025), 0.005 for the ischaemic compression group (p ≤ 0.025) and 0.004 for the combination group (p ≤ 0.025). Between visits 1 and 7, the p-value for the adjustment group was 0.004 (p ≤ 0.025), for the ischaemic compression group the p-value was 0.005 (p ≤ 0.025) and the combination group had a p-value of 0.004 (p ≤ 0.025) which also indicates a statistical significance between visit 1 and visit 7.

b) Intergroup Analysis of the Numerical Pain Rating Scale

Table 4.4 Non-Parametric Kruskal-Wallis Test for the Intergroup Analysis of the NPRS

<table>
<thead>
<tr>
<th>Reading</th>
<th>Mean Rank/p-value</th>
<th>Adjustment Group</th>
<th>Ischaemic Compression Group</th>
<th>Combination Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (Visit 1)</td>
<td>Mean Rank</td>
<td>15.50</td>
<td>13.50</td>
<td>17.65</td>
</tr>
<tr>
<td>p-value</td>
<td>0.495 thus p &gt; 0.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean Rank</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>--------</td>
<td>-----------</td>
<td>-------</td>
<td>-------</td>
<td></td>
</tr>
<tr>
<td>2 (Visit 4)</td>
<td>14.55</td>
<td>13.65</td>
<td>18.30</td>
<td></td>
</tr>
<tr>
<td>p-value</td>
<td>0.431 thus p &gt; 0.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 (Visit 7)</td>
<td>13.60</td>
<td>14.85</td>
<td>18.05</td>
<td></td>
</tr>
<tr>
<td>p-value</td>
<td>0.479 thus p &gt; 0.05</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The non-parametric Kruskal-Wallis test was also used for the intergroup analysis of NPRS. Table 4.4 shows that there were no statistically significant differences for visit 1, as the p-value was 0.495 (p > 0.05). There was also no statistically significant difference for reading 2 (visit 4) with the p-value being 0.431 (p > 0.05). There was also no statistically significant differences for reading 3 (visit 7) for the NPRS as the p-value is 0.479 (p > 0.05). Therefore no differences between the groups were present due to no statistical significance found.

A decrease of 4.80 was noted for the adjustment group, 4.10 for the ischaemic compression group and 4.50 for the combination group between the first and seventh consultations.

There was no statistically significant change for the first reading between the three groups with a p-value = 0.495 (p > 0.05). The second reading showed no statistically significant change with p-value = 0.431 (p > 0.05). The third reading had a p-value = 0.479 (p > 0.05), indicating that there was no statistically significant change between the three groups for the readings of the Numerical Pain Rating Scale.

4.3.2 McGill Pain Questionnaire

a) Intragroup Analysis of the McGill Pain Questionnaire
Table 4.5 Non-Parametric Friedman Test for Intragroup Analysis of the McGill Pain Questionnaire

<table>
<thead>
<tr>
<th>Group</th>
<th>Reading Number</th>
<th>Mean</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adjustment Group</td>
<td>1 (Visit 1)</td>
<td>5.30</td>
<td>0.000 thus p ≤ 0.05</td>
</tr>
<tr>
<td></td>
<td>2 (Visit 4)</td>
<td>2.50</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 (Visit 7)</td>
<td>1.10</td>
<td></td>
</tr>
<tr>
<td>Ischaemic Compression</td>
<td>1 (Visit 1)</td>
<td>6.90</td>
<td>0.000 thus p ≤ 0.05</td>
</tr>
<tr>
<td>Group</td>
<td>2 (Visit 4)</td>
<td>3.20</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 (Visit 7)</td>
<td>3.10</td>
<td></td>
</tr>
<tr>
<td>Combination Group</td>
<td>1 (Visit 1)</td>
<td>7.70</td>
<td>0.000 thus p ≤ 0.05</td>
</tr>
<tr>
<td></td>
<td>2 (Visit 4)</td>
<td>4.20</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 (Visit 7)</td>
<td>2.00</td>
<td></td>
</tr>
</tbody>
</table>

The adjustment group had a mean value of 5.30 for the first reading, 2.50 for the second reading and 1.10 for the third reading. An improved pain rating of 78.70% was noted between the first and seventh readings. The **p-value = 0.000** (**p ≤ 0.05**), thus indicating statistically significant differences.

The ischaemic compression group had a mean value of 6.90 for the first reading, 3.20 for the second reading and 3.10 for the third reading. An improvement of 84.06% was noted between the first and seventh readings. The **p-value = 0.000** (**p ≤ 0.05**), indicating that there were statistically significant differences.

The combination group had a mean value of 7.70 for the first reading, 4.20 for the second reading and 2.0 for the third reading. An improvement of 74.03% was noted between the first and seventh readings. The **p-value = 0.000** (**p ≤ 0.05**), therefore also indicating statistically significant changes occurring.
Table 4.5 illustrates that when the Friedman test was used when comparing the McGill Pain Questionnaire scores, the p-values are ≤ 0.05. This indicates that all three groups had statistically significant differences.

The results showed statistically significant changes and therefore it is necessary to establish when these changes occurred within each of the three groups. The non-parametric Wilcoxon Signed Ranks test was used to determine if the changes occurred during the first (visit 1) and second (visit 4) readings or between the second (visit 4) and third (visit 7) readings.

This was achieved by using the Bonferroni Adjustment. The Bonferroni Adjustment tests the smallest p-value against a significance level of 0.05 ÷ 2 = 0.025 and the largest p-value against a significance level of 0.05 ÷ 1 = 0.05. The use of the Bonferroni Adjustment is to lower the alpha value in order to prevent data from incorrectly appearing statistically significant. This also ensures accuracy of the data.

Table 4.6 Non-Parametric Wilcoxon Signed Ranks Test for the Intragroup Analysis of the McGill Pain Questionnaire

<table>
<thead>
<tr>
<th>Visit Number</th>
<th>Adjustment Group</th>
<th>Ischaemic Compression Group</th>
<th>Combination Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>p-value between visit 4 and visit 1</td>
<td>0.006 thus ≤ than 0.05</td>
<td>0.004 thus ≤ than 0.05</td>
<td>0.004 thus ≤ than 0.05</td>
</tr>
<tr>
<td>p-value between visit 7 and visit 1</td>
<td>0.001 thus ≤ than 0.025</td>
<td>0.001 thus ≤ than 0.025</td>
<td>0.002 thus ≤ than 0.025</td>
</tr>
</tbody>
</table>

Table 4.6 indicates that the three groups showed statistically significant differences for the McGill Pain Questionnaire between visits 1 and 4 with the p-values for the adjustment group being 0.006 (p ≤ 0.05), 0.004 for the ischaemic compression group (p ≤ 0.05) and 0.004 for the combination group.
(p ≤ 0.05). Between visits 1 and 7, the p-value for the adjustment group was **0.001** (p-value < 0.025), for the ischaemic compression group the p-value was **0.001** (p ≤ 0.025) and the combination group had a p-value of **0.002** (p ≤ 0.025).

b) Intergroup Analysis of the McGill Pain Questionnaire

Table 4.7 Non-Parametric Kruskal-Wallis Test for the Intergroup Analysis of the McGill Pain Questionnaire

<table>
<thead>
<tr>
<th>Reading</th>
<th>Mean Rank/p-value</th>
<th>Adjustment Group</th>
<th>Ischaemic Compression Group</th>
<th>Combination Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (Visit 1)</td>
<td>Mean Rank</td>
<td>16.10</td>
<td>17.05</td>
<td>13.35</td>
</tr>
<tr>
<td></td>
<td>p-value</td>
<td>0.432 thus p &gt; 0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 (Visit 4)</td>
<td>Mean Rank</td>
<td>15.90</td>
<td>17.00</td>
<td>13.60</td>
</tr>
<tr>
<td></td>
<td>p-value</td>
<td>0.412 thus p &gt; 0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 (Visit 7)</td>
<td>Mean Rank</td>
<td>13.10</td>
<td>18.45</td>
<td>14.95</td>
</tr>
<tr>
<td></td>
<td>p-value</td>
<td>0.465 thus p &gt; 0.05</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The Kruskal-Wallis test is a non-parametric test used to compare independent groups of 3 or more. The use of mean rank values is preferred over the use of the raw values for this test.

In Table 4.7 it can be observed that there was no statistically significant difference for the adjustment group as the p-value was **0.432** (p > 0.05). The p-value for the ischaemic compression group was **0.412** (p > 0.05) and the p-value for the combination group was **0.465** (p > 0.05). Therefore no differences between the groups were present due to no statistical significance found.
There was no statistically significant change for the first reading between the three groups with a p-value of 0.432 (p > 0.05). The second reading also showed no statistically significant change with a p-value of 0.412 (p > 0.05). The third reading had a p-value of 0.465 (p > 0.05), indicating that there was also no statistically significant change between the three groups for the readings of the McGill Pain Questionnaire.

4.4 OBJECTIVE DATA ANALYSIS

The objective data for this study was obtained using the pressure algometer.

4.4.1 Pressure Algometer

a) Intragroup Analysis of the Pressure Algometer Readings

Table 4.8 Non-Parametric Friedman Test for Intragroup Analysis of the Pressure Algometer

<table>
<thead>
<tr>
<th>Group</th>
<th>Reading Number</th>
<th>Mean</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adjustment Group</td>
<td>1 (Visit 1)</td>
<td>2.85</td>
<td>0.000 thus p ≤ 0.05</td>
</tr>
<tr>
<td></td>
<td>2 (Visit 4)</td>
<td>3.35</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 (Visit 7)</td>
<td>3.89</td>
<td></td>
</tr>
<tr>
<td>Ischaemic Compression</td>
<td>1 (Visit 1)</td>
<td>2.66</td>
<td>0.000 thus p ≤ 0.05</td>
</tr>
<tr>
<td>Group</td>
<td>2 (Visit 4)</td>
<td>3.20</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 (Visit 7)</td>
<td>4.12</td>
<td></td>
</tr>
<tr>
<td>Combination Group</td>
<td>1 (Visit 1)</td>
<td>2.78</td>
<td>0.000 thus p ≤ 0.05</td>
</tr>
<tr>
<td></td>
<td>2 (Visit 4)</td>
<td>3.35</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 (Visit 7)</td>
<td>4.12</td>
<td></td>
</tr>
</tbody>
</table>
The adjustment group had a mean value of 2.85 kg/cm² for the first reading, 3.35 kg/cm² for the second reading and 3.89 kg/cm² for the third reading. An improvement of 26.74% was noted between the first and seventh readings. The p-value = 0.000 (p ≤ 0.05), which indicates statistically significant differences.

The ischaemic compression group had a mean value of 2.66 kg/cm² for the first reading, 3.20 kg/cm² for the second reading and 4.12 kg/cm² for the third reading. This showed an improvement of 35.44% between the first and seventh readings. The p-value = 0.000 (p ≤ 0.05) which indicates statistically significant difference.

The combination group had a mean value of 2.78 kg/cm² for the first reading, 3.35 kg/cm² for the second reading and 4.12 kg/cm² for the third reading. An improvement of 32.52% was noted between the first and seventh readings. The p-value = 0.000 (p ≤ 0.05) therefore also indicating statistically significant changes occurring.

Table 4.8 illustrates that when the Friedman test was used when comparing the pressure algometer readings, the p-values are ≤ 0.05 for all three group in this study. This indicates that all three groups had statistically significant differences.

With these statistically significant changes occurring in all three groups, it is necessary to establish when these changes occurred within each of the groups.

The non-parametric Wilcoxon Signed Ranks test was used to determine if the changes occurred during the first (visit 1) and second (visit 4) readings or between the second (visit 4) and third (visit 7) readings.

This was achieved by using the Bonferroni Adjustment. The Bonferroni Adjustment tests the smallest p-value against a significance level of 0.05 ÷ 2 = 0.025 and the largest p-value against a significance level of 0.05 ÷ 1 = 0.05. The use of the Bonferroni Adjustment is to lower the alpha value in order to prevent data from incorrectly appearing statistically significant. This also ensures accuracy of the data.
Figure 4.9 Non-parametric Wilcoxon Signed Ranks Test with the Bonferroni Adjustment

<table>
<thead>
<tr>
<th>Visit Number</th>
<th>Adjustment Group</th>
<th>Ischaemic Compression Group</th>
<th>Combination Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>p-value between visit 4 and visit 1</td>
<td>0.005 thus ≤ than 0.05</td>
<td>0.005 thus ≤ 0.05</td>
<td>0.005 thus ≤ than 0.05</td>
</tr>
<tr>
<td>p-value between visit 7 and visit 1</td>
<td>0.005 thus ≤ than 0.025</td>
<td>0.005 thus ≤ 0.05</td>
<td>0.005 thus ≤ than 0.025</td>
</tr>
</tbody>
</table>

In Table 4.9 it can be concluded that all three groups showed statistically significant differences for the pressure algometer readings between visits 1 and 4 with the p-values being 0.005 for the adjustment group (p ≤ 0.05), 0.005 for the ischaemic compression group (p ≤ 0.05) and 0.005 for the combination group (p ≤ 0.05). The p-value for the adjustment group between visit 1 and visit 7 was 0.005 (p ≤ 0.025), 0.005 for the ischaemic compression group (p ≤ 0.025) and for the combination group the p-value was 0.005 (p ≤ 0.025), all indicating statistically significant differences.

b) Intergroup Analysis of the Pressure Algometer Readings

Figure 4.10 Non-parametric Kruskal-Wallis Test

<table>
<thead>
<tr>
<th>Reading</th>
<th>Mean Rank/p-value</th>
<th>Adjustment Group</th>
<th>Ischaemic Compression Group</th>
<th>Combination Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (Visit 1)</td>
<td>Mean Rank</td>
<td>17.00</td>
<td>12.60</td>
<td>16.90</td>
</tr>
<tr>
<td>p-value</td>
<td>0.443 thus p &gt; 0.05</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
The non-parametric Kruskal-Wallis test was used for the intergroup analysis of the pressure algometer readings. In Table 4.10 it can be observed that the p-value for reading 1 (Visit 1) was 0.443 (<i>p > 0.05</i>), the p-value for reading 2 (Visit 4) was 0.823 (<i>p > 0.05</i>) and the p-value for reading 3 (Visit 7) was 0.297 (<i>p > 0.05</i>). These p-values indicate that there were no statistically significant differences between all three groups at readings 1, 2 and 3.

There was no statistically significant change for the first reading between the three groups with a p-value of 0.443 (<i>p > 0.05</i>).

The second reading also showed no statistically significant change with a p-value of 0.823 (<i>p > 0.05</i>). The third reading had a p-value of 0.297 (<i>p ≤ 0.05</i>), indicating that there was no statistically significant change between the three groups for the readings of the pressure algometer.
CHAPTER FIVE: DISCUSSION

5.1 INTRODUCTION

This chapter discusses the results with reference to the statistical analyses presented in Chapter 4.

5.2 DEMOGRAPHIC DATA

The trial consisted of 30 participants with an equal male to female ratio between the groups and within the groups. The gender distribution was purposely set at 15 males and 15 females. This is done as upper thoracic (inter- scapular) pain shows no gender bias (Marx, Hockberger and Walls, 2010).

The mean age of the sample was 23.30 years old. The adjustment group had a mean age of 23.70, the ischaemic compression group had a mean age of 23.90 and the combination group had a mean age of 22.30. The range of participants recruited for this study was 20 to 28 years of age.

Frontera, Silver and Rizzo (2008), state that any thoracic sprain or strain acute or sub-acute that causes pain in the thoracic region is due to bone or soft tissue injury. These thoracic injuries causing upper thoracic pain can occur in all age groups, but there is an increased prevalence among patients of working age. Frontera et al. (2008), reiterates Marx et al. (2010), finding that upper thoracic pain is not gender specific and is evenly distributed between the sexes.

However Hutson and Speed (2011), also state that upper thoracic pain is commonly seen in the younger adult age groups, this is due to their high levels of activity.

Therefore, the demographic data of this study indicated a fair representation of the population which are commonly affected with thoracic or inter-scapular pain.
5.3 SUBJECTIVE DATA

This study made use of two methods for evaluating the subjective data. The subjective data signifies what the participant was experiencing at the time by means of assigning data into numerical formats. This was achieved using the McGill Pain Questionnaire and the Numerical Pain Rating Scale.

5.3.1 McGill Pain Questionnaire

a) Initial Pain and Disability Comparison of the MPQ

For the initial reading of the McGill Pain Questionnaire (MPQ), the adjustment group had a p-value of 0.728 (p > 0.05), the ischaemic compression group had a p-value of 0.405 (p > 0.05) and the combination group had a p-value of 0.814 (p > 0.05). These readings indicate that there was no difference between the three groups for the MPQ indicating that all three groups started on the same levels of disability and were therefore comparable.

b) Intragroup Analysis of the MPQ

The non-parametric Friedman test was used for the intragroup analysis of the MPQ. The test was used to determine changes in mean values over time for all three groups. Table 4.5 indicates that all three groups had statistically significant differences with p-values of 0.000 (p ≤ 0.05).

Due to these statistically significant differences, the non-parametric Wilcoxon Signed Ranks test was used with the Bonferonni Adjustment to establish when these changes occurred within the three groups. Between visits 4 and visit 1, the adjustment group had a p-value of 0.006 (p ≤ 0.05), the ischaemic compression group had a p-value of 0.004 (p ≤ 0.05) and the combination group had a p-value of 0.004 (p ≤ 0.05). Between visits 7 and 1, the adjustment group had a p-value of 0.001 (p ≤ 0.025), the ischaemic compression group had a p-value of 0.001 (p ≤ 0.025) and the combination
group had a p-value of \textbf{0.002} \,(p \leq 0.025). Table 4.6 indicates that all three groups showed statistically significant differences for the McGill Pain Questionnaire between visits 1 and 4 and visits 1 and 7.

All three of the groups showed a great percentage of improvement over the three week trial with regards to the McGill Pain Questionnaire. The ischaemic compression group had the greatest improvement with **84.06%**, closely followed by the adjustment group with **79.25%** and the combination group with an improvement of **74.03%**. Therefore this indicates that all three are effective as a treatment protocol for inter-scapular pain.

As stated earlier the McGill Pain Questionnaire provides the researcher with a quantitative profile of the three aspects of pain, this being sensory-discriminative, motivational-effective and cognitive evaluative (Maire, 2002). This can be perceived differently by the individual participants, which could explain why one group using the MPQ showed better results compared to the other subjective data obtained by the researcher.

In a recent study that investigated the effects of ischaemic compression on the myofascial trigger points of the trapezius muscle, it stated that ischaemic compression by means of slowing down the blood supply and forcing the tension out of the muscle resulted in desensitization of the nerve endings in the surrounding skin. This could explain why the ischaemic compression group may have demonstrated better results with the MPQ at the end of the 3 week trial, as the participants had a lower level of perceived pain (Iqbal, Khan and Miraj, 2010).

The adjustment groups in this study had similar but slightly reduced results with regards to the McGill Pain Questionnaire, as this could be due the presence of the most common side effect following a chiropractic adjustment. This side effect is the mild discomfort found in the treatment area immediately after the adjustment, this could explain why the adjustment groups had a slightly reduced improvement following the treatment. Other side effects could include tenderness and tiredness in the treatment area (Lester, 2012).
In a study where the MPQ was used to assess the nature, and intensity of labour pain, it showed that there was a great variety between subjects to what they perceive as being painful. This can be explained by one psychological factor, pain experienced by the patient previously. Subjects who have had previously experienced significant levels of pain unrelated to childbirth had a low or moderate levels of labour pain. Subjects who reported little experience of pain unrelated to childbirth had high levels of pain (Niven and Gijsbers, 1984).

This could explain the different readings in each group, as some participants may have a higher or lower level of perceived pain.

c) Intergroup Analysis of the MPQ

The non-parametric Kruskal-Wallis test was used for the intergroup analysis by making use of the mean ranks. Table 4.4 shows that there were no statistically significant differences between the three groups as the p-value for the adjustment group was 0.432 \( (p > 0.05) \), 0.412 \( (p > 0.05) \) for the ischaemic compression group and the p-value for the combination group was 0.465 \( (p > 0.05) \). The non-parametric Mann-Whitney test was not used as there were no statistically significant differences.

The adjustment group presented with a mean score of 5.3 at the first reading with an improvement to a mean score of 1.1 at the last reading. The ischaemic compression group presented with a mean score of 6.9 at the first reading with an improvement to a mean score of 3.1. The combination group initially presented with a mean score of 7.7 and improved to a reading with a mean score of 2.0.

All three groups showed an almost similar decrease in pain, indicating all three treatment protocols are effective. In a study by Hoiriis, Pfleger, McDuffie, Cotsonis and Elsangak (2004), on the effects of chiropractic adjustments compared to muscle relaxants for subacute low back pain. This study showed that chiropractic adjustments were far superior to the administration of muscle relaxants in reducing pain caused by muscle spasm. As myofascial trigger points are related to local tenderness and muscle spasm (Fisher, 1988). This therefore indicates that a chiropractic adjustment to an underlying joint is just as effective as ischaemic compression in treating muscle spasm, which is related to the formation of myofascial trigger points in spasmodic muscle.
In a study by Fernandez-De-las-Pans, Alonso-Blanco, Fernandez-Carnero and Carlos Miangolarra-Page (2006), determined the effects of ischaemic compression on the tenderness of active or latent myofascial trigger points. The results of this study showed that ischaemic compression was very effective in reducing myofascial trigger point tenderness. And as tenderness and muscle spasm are seen in areas of myofascial trigger points. This therefore concludes that chiropractic adjustments delivered to underlying joints and ischaemic compression have the same treatment outcome, which is reducing muscle spasm caused by myofascial trigger points.

Therefore this could explain why there was no real difference found between the 3 different treatment groups with regards to statistical analysis done by using the Kruskall-Wallis test. Each group was benificial but no superior treatment protocol was identified.

5.3.2 Numerical Pain Rating Scale

a) Initial Pain and Disability Comparison of the NPRS

For the initial reading of the NPRS, the adjustment group had a p-value of 0.107 (p ≤ 0.05), the ischaemic compression group had a p-value of 0.149 (p > 0.05) and the combination group had a p-value of 0.177 (p > 0.05). These values indicate that all three groups started off with the same levels of pain and disability and were therefore comparable.

b) Intragroup Analysis of the NPRS

The non-parametric Friedman test was used for the intragroup analysis of the NPRS. This test was used to determine changes in mean values over time for all three groups. Table 4.2 indicates that all three groups had statistically significant differences with p-values of 0.000 (p ≤ 0.05).

Due to these statistically significant differences, the non-parametric Wilcoxon Signed Ranks test was used with the Bonferonni Adjustment to establish when these changes occurred within the three groups. Between visits 4 and 1, the adjustment group had a p-value of 0.005 (p ≤ 0.05), the
ischaemic compression group had a p-value of **0.005** \( (p \leq 0.05) \) and the combination group had a p-value of **0.004** \( (p \leq 0.05) \). Between visits 7 and 1, the adjustment and combination groups had had p-values of **0.004** \( (p \leq 0.025) \). While the ischaemic compression group had a p-value of **0.005** \( (p \leq 0.025) \) between visit 7 and visit 1. Table 4.3 represents that all three groups showed statistically significant differences for the NPRS between visits 1 and 4 and visits 1 and 7.

The adjustment group had the greatest improvement with **78.70%** followed closely by the ischaemic compression group with **71.90%**. The combination group showed an improvement of **69.23%**.

With the NPRS the decrease in pain can be attributed to the chiropractic adjustment. When the normal range of motion is restored to joint, then pain caused by restricted motion has a significant decrease (Gatterman, 1990). The adjustment has an effect on the nerves related to areas that have been adjusted and leads to pain inhibition and muscle relaxation (Bergmann, 1993; Esposito & Philipson, 2005).

In a recent study which explored the clinical treatment effect of mechanical versus manual adjustments for acute lower back pain. This study was carried out for patients with lower back pain using mechanical or manual adjustments over the duration of 4 weeks. Measurements were done using the Numerical Pain Rating Scale (NPRS) and the Oswestry Lower Back Pain Disability Index Questionnaire (ODI). The study showed that manual chiropractic adjustments had a slightly greater amount of pain reduction at the 4th consultation with regards to the numerical pain rate rating scale (NPRS) (Schnieder, 2008).

The analgesic effects experienced by the adjustment group is similar to the results obtained by Schiller (2001) who demonstrated a statistically significant positive effect of thoracic chiropractic adjustment on mechanical mid-thoracic pain in a single blind, placebo controlled study, as measured by NPRS. In a study obtained by Carlyle, Moodley and Hollinshead (2010), who demonstrated a statistical significant reduction of latent upper trapezius myofascial trigger point pain intensity following cervicothoracic chiropractic adjustments. Similarly, Costello (2008) demonstrated the analgesic effects of upper thoracic spinal adjustments on pain secondary to cervical radiculopathy, likely due to the reflex central pain inhibitory mechanism.
The analgesic effects is postulated to be caused by a sudden stretch of the shortened rhomboid muscle fibres, (secondary to MTrP formation), resulting in the activation of central descending inhibitory pain mechanism by afferent impulses from stimulation of mechanoreceptors within the upper-thoracic facet joint capsules (Gatterman, 2005).

With the ischaemic compression group, a study involving ischaemic compression of a trigger point compared to placebo ultrasound, showed that ischaemic compression was very successful in reducing pain (Gemmell, Miller and Nordstrom, 2008). With ischaemic compression the deep pressure applied to the muscle intentionally decreases the blood supply temporarily in the myofascial trigger point area in order to increase local blood flow. This leads to elimination of waste products, therefore increasing the local oxygen supply which allows the affected tissue to heal (Arnau-Masanet, Barrios-Pitarque, Bosch-Morell, Montanez-Aguilera, Pecos-Martin and Valtuena-Gimeno, 2010). The application of ischaemic compression creates mechanical disruption of the locked actin-myosin myofibril cross links within the myofascial trigger points (Perle, Schneider, and Seaman, 1999) and causes a decrease in the sensory afferent input of noxious stimuli to the brain via the pain gate theory (Martin, Wilcox and Moodley, 2008). Pain relief from ischaemic compression has been linked with altered spinal reflex mechanisms, resulting in decreased muscle spasm therefore decreasing the readings of the NPRS (Ingber, Kostopoulos, Larkin and Nelson, 2008).

As stated earlier in this chapter, the chiropractic adjustment and ischaemic compression can cause a reduction on pain experienced by the participant. Both Subjective data readings showed an improvement over time. The NPRS of certain groups were slightly less improved, this can be due to external factors. External factors such as altered position which can aggravate the pain, a full bladder or the temperature of the room can have a negative effect on the NPRS (Wells, Pasero and McCaffery, 2008). This could account for the less impressive readings in the NPRS seen in this study in certain groups.

c) Intergroup Analysis of the NPRS

The non-parametric Kruskal-Wallis test was used for the intergroup analysis by making use of the mean rank values of the different data. In table 4.4 it can be observed that there were no statistically
significant differences for visit 1 with the p-value being 0.495 (p > 0.05) and for visit 4 the p-value was 0.431 (p > 0.05). There was also no statistically significant differences for reading 3 (visit 7) for the NPRS as the p-value was 0.479 (p > 0.05). The non-parametric Mann-Whitney test was not used as there were no statistically significant differences.

In a randomised clinical trial where the treatment of acute neck pain was assessed by comparing chiropractic cervical and thoracic spine adjustments, there were no statistically significant differences between the two groups for the NPRS readings with a p-value of 0.213 (p > 0.0125), using the Bonferroni Adjustment (Puente dura, Landers, Cleland, Mintken, Huijbregts & Fernandez De Las Penas, 2011). This indicates that the intergroup results for the NPRS readings seen from this study are comparable and similar to the results of a randomised clinical trial.

With reference to the studies discussed above, it can be deduced that the chiropractic adjustments and ischaemic compression will have a decrease on the perceived levels of pain as assessed by the NPRS. External factors still play a role in the NPRS as stated by Wells et al. (2008).

It is evident in the results that all three groups improved in a similar way with regards to the NPRS. Statistically using the Kruskal-Wallis test, this test indicated no treatment protocol was deemed superior as all three groups improved in a similar manner, slight differences could be accounted for by external factors affecting the participant and the NPRS.

5.4 OBJECTIVE DATA

5.4.1 Pressure Algometer

a) Initial Pain and Disability Comparison of the Pressure Algometer

For the initial readings of the pressure algometer, the adjustment group had a p-value of 0.240 (p > 0.05), the ischaemic compression group had a p-value of 0.511 (p > 0.05) and the combination group
had a p-value of 0.154 (p > 0.05). This indicates that all three groups started on the same objective pain level as assessed by the pressure algometer, as there were no statistically significant differences (p > 0.05). These findings indicate that the initial pain and disability between the three groups were comparable.

b) Intragroup Analysis of the Pressure Algometer

The non-parametric Friedman test was used for the intragroup analysis of the pressure algometer. Table 4.8 indicates that all three groups had statistically significant differences as each group had a p-value of 0.000 (p ≤ 0.05).

Due to these statistically significant differences of all three groups, the non-parametric Wilcoxon Signed Ranks test was used with the Bonferonni Adjustment to establish when these changes occurred. Between visits 4 and 1, the adjustment group, ischaemic compression group and the combination group had a p-value of 0.005 (p ≤ 0.05). Between visits 7 and 1, the adjustment group, ischaemic compression group and combination group had a p-value of 0.005 (p ≤ 0.025). Table 4.9 represents that the all three groups showed statistically significant differences for the pressure algometer readings between visits 1 and 4 and visits 1 and 7.

The ischaemic compression group had the greatest increase in the mean value from 2.66 kg/cm² to 4.12 kg/cm², an improvement of 35.44%. The combination group had the second greatest increase in the mean value from 2.78 kg/cm² to 4.12 kg/cm². This was an improvement of 32.52%. The adjustment group had the least increase in the mean value from 2.85 kg/cm² to 3.89 kg/cm², an improvement of 26.74%.

In a recent study by Schmidt (2014), the effect of manual compressive therapy on latent myofascial trigger point pressure pain threshold. Manual compression was held for 90 seconds, and pressure pain threshold was measured after 5 minutes with the algometer. This study showed that manual compression therapy was reliable in increasing pressure pain threshold in subjects with latent myofascial trigger points of the upper back. Because this study found the manual compressive
therapy is a reliable treatment in increasing the pain pressure threshold in subjects with latent trigger points in the short term. This significant change for the better which is an increase in the pressure pain threshold in the subjects after application, combined with the desensitization of the nerve endings in the surrounding skin following ischaemic compression therapy as stated by Iqbal, Khan and Miraj, (2010), could explain why the ischaemic compression group showed the most improvement and best results of the three treatment group with regards to the pressure algometer.

In a study performed to determine the effectiveness of the manual pressure release of latent upper trapezius myofascial trigger points concluded that manual pressure release is effective based on a measurable increase in the pressure pain threshold values measured objectively with a pressure algometer (Fryer and Hodgson, 2005).

Herzog, Scheele and Conway (1999), also stated that the reflexogenic mechanism of the chiropractic adjustments is believed to cause a relief of pain and a decrease in the muscle hypertonicity directly at the area where the adjustments was applied. This will cause a decrease in the pressure algometer readings as the muscle spasm is reduced.

c) Intergroup Analysis of the Pressure Algometer

The non-parametric Kruskal-Wallis test was used for the intergroup analysis by making use of the mean ranks. Table 4.10 shows that there were no statistically significant differences between the three groups as the p-value for reading 1 was 0.225 (p > 0.05), 0.117 for reading 2 (p > 0.05) and 0.080 for reading 3 (p > 0.05). The non-parametric Mann-Whitney test was not used as there were no statistically significant differences.

In a study by Cagnie, Dewitte, Coppieters, Van Oosterwijck, Cools and Danneels (2013), that was done to determine the short term effect of ischaemic compression for trigger points on muscle strength, mobility, pain sensitivity and disability in the office workers and the effect on disability and general pain at 6 months. This study showed an increase in pressure pain threshold using a pressure algometer with a statistically significant p-value of p = 0.001.
In another study by Fernandez De Las Penas, Campo, Camero and Page (2005), the use of ischaemic compression on latent or active trigger in upper trapezius muscles resulted in an improvement of the pressure pain threshold measured by an algometer and visual analogue scale (Chaitlow, 2006).

In a study by Schiller (2001), on the effectiveness of spinal manipulative therapy in the treatment of mechanical thoracic pain. It showed that there was a significant improvement in the readings obtained with the McGill Pain Questionnaire, Numerical Rating Scale and a pressure algometer.

It is evident in the results that all three groups improved in a similar way with regards to the pressure algometer. Statistically using the Kruskal-Wallis test, it showed no significance and no treatment protocol could be deemed the more superior one as all showed similar improvement.

With reference to the studies discussed above, it can be deduced that all three treatment groups had a positive effect on inter-scapular pain. Ischaemic compression had a slightly better increase in pain pressure threshold, however each group still showed an increase in the pain pressure threshold levels as assessed by the pressure algometer. The treatment protocols with the presence of chiropractic adjustments, showed a slightly lower improvement which can be associated with tenderness and slight discomfort following the chiropractic adjustment (Lester, 2012). This could be the reason why the pressure algometer readings did not increase as much, this is due to the additional tenderness and discomfort present.

5.5 DISCUSSION OF THE RESULTS

All of evidence presented in Chapter 2 indicates that there is a theoretical link between inter-scapular pain and rhomboid myofascial trigger points. As stated by Rouzier (2005), a muscle strain in the rhomboid musculature causes pain in the upper back, between the scapular and spine. There is also evidence that links myofascial trigger points of the rhomboids with costovertebral syndrome. As stated by Bahr (2012), costovertebral subluxation syndrome presents with rhomboid muscle spasm and a sharp pain in the inter-scapular region. This is also supported by the results of this trial, as
there were no statistically significant differences between the three groups on the intergroup analyses with regards to the different treatment protocols.

This link can be accounted for by the vertebral subluxation complex (VSC). As stated in Chapter 2 the dysfunction at any joint can affect surround nerves, muscles, ligaments, local blood supply and connective tissue. In this case the components of the VSC most commonly affected are the neuropathophysiology and myopathy components. This will result in hypertonia and hypertonicity in the overlying muscles following joint dysfunction/dysfunction at the costovertebral joints.

With regards to the evidence presented, the improvement of inter-scapular pain can be seen in all three groups. A chiropractic adjustment causes neurophysiologic effects to occur following the adjustment. The pain gate theory will inhibit pain receptor flow via enkephalin synaptic interneuron transmitter and they are inhibited by approximation of the joint surfaces as well as immobilisation of the joints as seen in the vertebral subluxation complex. Pain evocation results from the stimulation of nociceptors due to the vertebral subluxation. The normal functioning of mechanoreceptors inhibits the functioning of the nociceptors and the abnormal functioning of the mechanoreceptors increases stimulation of the nociceptors. Pain results when there is a decreased inhibition of the nociceptive activity (Wyke, 1985). The chiropractic adjustment is theorised to induce sensory afference which closes the gate therefore diminishing or alleviating the pain (Fishman, Ballantyne & Rathmell, 2009).

With ischaemic compression as pressure on the taut band is directed towards the underlying bone, this action squeezes fluid out of the immediate area, causing a temporary ischaemia (hence the term “ischaemic compression”). The body will react by sending blood to the area, causing a reactive hyperaemia as pressure is released. This brings oxygen and well need nutrients to the compressed area and flushes out the metabolic waste products. The slight stretching of the muscle or tendon that occurs as pressure is applied elicits an inhibitory response from the golgi tendon organ, facilitating relaxation of the treated area and causing relief of pain produced by the hypertrophic muscle (Stephens, 2006).
The results from this trial confirm that there is a relationship between inter-scapular pain, rhomboid myofascial trigger points and costovertebral syndrome. Each group had a positive effect on treating inter-scapular pain over time as seen in the intra-group analysis as each group showed statistical significance, however when comparing each to one another there was no statistical significance found.
CHAPTER SIX: CONCLUSION AND RECOMMENDATIONS

6.1 CONCLUSION

The purpose of this study was to determine which treatment protocol in the form of chiropractic adjustments to the costovertebral joints, ischaemic compression to the rhomboid muscles or a combination treatment of both chiropractic adjustment and ischaemic compression was superior for the treatment of inter-scapular pain.

The outcome of the clinical and statistical findings for this study on the chiropractic profession suggests that patients with inter-scapular pain can be treated effectively. All three groups in this study improved, however according to statistical analysis no one group showed superiority with regards to treatment protocol.

This indicated that any one of the three treatment protocols can be used in the treatment of inter-scapular pain provided there are no contra-indications present. If the patient is contra-indicated to chiropractic adjustment, ischaemic compression is a suitable alternative treatment protocol as seen in this study.

The chiropractic adjustment and process of ischaemic compression both have a positive effect on the suppression of pain and disability symptoms, when it is applied to the treatment of inter-scapular pain.

In conclusion, there is no superior treatment protocol from inter-scapular pain found in this study, as chiropractic adjustments to the costovertebral joint, ischaemic compression of rhomboid myofascial trigger points or a combination treatment have both shown to decrease pain and increase well being in the participant. This study has given the chiropractic profession three different yet, effective treatment protocols for patients suffering with inter-scapular pain.
6.2 RECOMMENDATIONS

The following recommendations should be taken into consideration for future research regarding the treatment of inter-scapular pain:

- A larger sample size to provide more information and relevance to the general population.

- The inclusion of a one month follow up visit after the study to compare the long term effects of all three treatment protocols.

- Take the subjective and objective data readings before and after the treatment to assess the immediate effects of the chiropractic adjustment and ischaemic compression on inter-scapular pain.

- A research study with two sample groups. One receiving costovertebral chiropractic adjustments with ischaemic compression. Another group receiving costovertebral chiropractic adjustment with dry needling to the rhomboid trigger points. This will provide a comparison between common treatments protocols currently used.

- Add an additional objective measurement of thoracic spine range of motion to assess the effects of thoracic spine chiropractic adjustments and ischaemic compression on the thoracic spine and to assess the range of motion of the thoracic spine by using the newly developed skin-surface device called the Spinal Mouse.
REFERENCES


DO YOU SUFFER FROM PAIN IN BETWEEN YOUR SHOULDER BLADES?

RESEARCH STUDY PROVIDING CHIROPRACTIC TREATMENT!!!

If you are between the ages of 18 and 45 years come take part in a research project involving the treatment of pain found in between shoulder blades.

Treatment will be conducted at the University of Johannesburg Doornfontein Campus, Gate 7, Sherwell Road.

If you are interested in receiving treatment contact:

Jared Irwin: 083 302 6161
APPENDIX B: PARTICPANT INFORMATION FORM

DEPARTMENT OF CHIROPRACTIC

INFORMATION FORM

I, Jared Irwin, hereby invite you to participate in my research study, entitled: The Effect of Costovertebral Adjustment versus Ischaemic Compression of Rhomboid Muscles for Inter-Scapular pain. I am currently a Chiropractic student, completing my Masters Degree at the University of Johannesburg.

Before agreeing to participate, it is important that you read and understand the following explanation of the aim of the study, the study procedure, benefits, risks, discomforts, and your right to withdraw from the study at any time. If you have any questions, do not hesitate to ask me. If you decide to partake in this study, you will be asked to sign this document to confirm that you understand the study. You will be given a copy to keep.

The aim of the study is to determine which treatment protocol is the most effective for pain found in between shoulder blades, with either Chiropractic adjustment to the joints found between the ribs and the spine (Costovertebral joints) or ischaemic compression of rhomboid muscle trigger points or a combination treatment involving the two.

The study will consist of three groups of ten participants per group, each participant will be examined and accepted according to the inclusion and exclusion criteria. The method of treatment will be determined by randomly assigning each participant to a group. Group 1 participants will receive chiropractic adjustments to the restrictions of the costovertebral joints found between T1-T5 levels during motion palpation. Group 2 will receive ischaemic compression to the rhomboid muscle trigger points found on the medial border of the shoulder blade (scapula). Group 3 will receive a combination treatment of costovertebral joint adjustments and ischaemic compression of rhomboid trigger points.

The study will not discriminate based on demographics such as gender and race. However, participants must be healthy to ensure validity and eliminate situations where spinal manipulations have been contra-indicated. The total time required for your participation in this study will be a maximum of three weeks which includes seven consultations.
The Chiropractic adjustment involves the restoration of normal joint motion. Abnormal joint motion will be detected by the researcher via motion palpation. The Chiropractic adjustment is a safe, non-invasive treatment technique. Chiropractic adjustments are normally done as part of routine chiropractic care and present a slight risk of discomfort. Chiropractic adjustments may result in post-adjustment stiffness. There is also a slight possibility of pain. The study is investigative and there may be other risk or side effects which are unforeseen or unknown. You should immediately contact me if any side effects occur throughout your participation in this study. Participants will be referred appropriately when necessary. 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. Your protection is that an experienced person performs the chiropractic adjustments and ischaemic compression under observation of a qualified chiropractor.

Potential benefits include a decrease in inter-scapular pain.

The research study will take place at the University of Johannesburg Chiropractic Day Clinic. Your privacy will be protected as only the researcher, patient (you) and qualified chiropractor will be in the treatment room. Your anonymity will be ensured as your personal information will be converted into data and therefore cannot be tracked to you. Standard doctor/patient confidentiality will be adhered to all times compiling the research dissertation. Results of this study will be made available to you on request.

This clinical study protocol has been submitted to the University of Johannesburg's, Higher Degrees Committee (HDC) and a written approval has been granted by the committee.

Researcher: Jared Irwin 083 302 6161
Supervisor: Dr C. Yelverton 011 559 6218
Co-Supervisor: Dr D. Whelan 083 294 9946

Ethics Clearance Number: AEC01-32-2014
APPENDIX C: PARTICIPANT CONSENT FORM

DEPARTMENT OF CHIROPRACTIC

CONSENT FORM

Your participation in this study is entirely voluntary and you can decline to participate, or stop at any time, without stating any reason. Your withdrawal will not affect your access to medical care. I will provide you with any additional information that becomes available during the study, which may affect your willingness to continue on the study. I retain the right to withdraw you from the study if it is considered to be in your best interest. If you did not give an accurate history, or did not follow the guidelines of the study and the regulations of the study facility, you may be withdrawn from the study at any time.

All information obtained during the course of this study, including clinic records, personal data and research data will be kept strictly confidential. Data that may be reported as scientific will not include any information that identifies you as a participant in this study. You will be informed of any finding of importance to your health or continued participation in this study but this information will not be disclosed to any third party without your written permission.

Informed consent:

☐ I hereby confirm that I have been informed by the researcher, Jared Irwin, about the nature, conduct, benefits and risks of this study.

☐ I have also received, read and understood the Participant Information Form regarding the study.

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

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

Date: _______________________ Participant: _________________________

I, Jared Irwin, herewith confirm that the above participant has been fully informed about the nature, conduct and risk of the above study.

Date: _______________________ Researcher: _______________________

I, the undersigned Jared Irwin, have read and have explained fully to the participant, named ______________________ the Participation Information Form and Consent Form.

I hereby certify that, the participant has agreed to participate in the study.

Date: _______________________ Participant: _________________________

Researcher: Jared Irwin 083 302 6161
Supervisor: Dr. C. Yelverton 011 559 6218
Co-Supervisor: Dr. D. Whelan 083 294 9946
APPENDIX D: CONTRA-INDICATIONS TO CHIROPRACTIC ADJUSTMENTS (Peterson and Bergman, 2002).

1. Vascular Complications
   - Vertebrobasilar insufficiency
   - Atherosclerosis
   - Aneurysms

2. Tumours

3. Osteoporosis

4. Bone infection
   - Tuberculosis of the spine
   - Osteomyelitis of the spine.

5. Traumatic injuries
   - Fractures
   - Instability
   - Dislocations
   - Unstable spondylolisthesis
   - Severe sprains

6. Arthritis
   - Ankylosing spondylitis
   - Rheumatoid arthritis
   - Osteoarthritis
   - Uncoarthritis

7. Psychological considerations
   - Malingering
   - Hysteria
   - Hypochondiasis
   - Alzheimer’s disease
<table>
<thead>
<tr>
<th></th>
<th>NONE</th>
<th>MILD</th>
<th>MODERATE</th>
<th>SEVERE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Throbbing</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Shooting</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Stabbing</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Sharp</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Cramping</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Gnawing</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Hot-Burning</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Aching</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Heavy</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Tender</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Splitting</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Exhausting</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Sickening</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Fearful</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Punishing-Cruel</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
APPENDIX F: NUMERICAL PAIN RATING SCALE

Place a mark on the pain scale below that represents your pain at this point in time. On a scale of 0 to 10, 0 means “no pain” and 10 means “worst possible pain”. The middle of the scale describes “moderate pain”. A two or three rating would be “mild pain” and a rating of seven or higher would indicate “severe pain”.

Visit 1:

<table>
<thead>
<tr>
<th>No pain</th>
<th>Moderate pain</th>
<th>Severe pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>10</td>
</tr>
</tbody>
</table>

Visit 4:

<table>
<thead>
<tr>
<th>No pain</th>
<th>Moderate pain</th>
<th>Severe pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>10</td>
</tr>
</tbody>
</table>

Visit 7:

<table>
<thead>
<tr>
<th>No pain</th>
<th>Moderate pain</th>
<th>Severe pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>10</td>
</tr>
</tbody>
</table>
APPENDIX G: PRESSURE ALGOMETER READINGS

Participants name:

File number:

Date:

RHOMBOID MINOR AND MAJOR MUSCLE

<table>
<thead>
<tr>
<th></th>
<th>First Visit</th>
<th>Fourth Visit</th>
<th>Seventh visit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reading 1 (kg/cm²)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading 2 (kg/cm²)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reading 3 (kg/cm²)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
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:
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 I: 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: ________

Blood pressure: | Arms: | L | R
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>L</td>
</tr>
<tr>
<td>Legs:</td>
<td></td>
<td>L</td>
</tr>
</tbody>
</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: 90º (fingers to floor)
   Extension: 50º
   R. lat. flex: 30º (fingers down leg)
   L. lat. flex: 30º (fingers down leg)
   Rot. To R: 35º
   Rot. To L: 35º

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: skin
   symmetry
   ROM - glenohumeral
   - scapulo-thoracic
   - acromioclavicular
   - elbow
14. Chest measurement:

- Inspiration
- expiration

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15. Visual acuity

16. Breast examination:

- 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:
   - conjunctiva
   - sclera
   - eyebrows
   - eyelids
   - lacrimal glands
   - nasolacrimal duct
   - position and alignment
   - corneas and lenses

- corneal reflex
- ocular movement

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

4. Ears:
   - auricle

   - Inspection
     - 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
     - CNX
     - inspection
• carotid arteries (thrills, bruit)
• Cranial Nerves
  - CNV
  - CNVII
  - CNVIII (nystagmus)
  - CNIX
  - CNXI
  - CNX11

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

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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 hyperesthesia
  - 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

(iii) Memory and attention

- orientation (time, place, person)
- remote memory
  - recent memory
  - new learning ability

(vi) Higher cognitive functions

- Information and vocabulary
- (general and specialised knowledge)
- Abstract thinking
## NEUROLOGICAL EXAMINATION (LUMBAR SPINE)

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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:

________________________________________________________________________

________________________________________________________________________

________________________________________________________________________

VASCULAR | LEFT | RIGHT
--- | --- | ---
BLOOD PRESSURE | | |
CAROTIDS | | |
SUBCLAVIAN ARTERIES | | |
WALLENBURG’S TEST | | |

COMMENTS:

________________________________________________________________________

________________________________________________________________________

________________________________________________________________________
### MOTION PALPATION

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### Appendix K: SOAP NOTE

#### CHIROPRACTIC DAY CLINIC

**SOAP NOTE:**

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