The relative effect of manipulation and core rehabilitation in the treatment of acute mechanical lower back pain in athletes

By

Jennifer Campbell

Dissertation submitted in partial compliance with the requirements for the Master's Degree in Technology: Chiropractic at Durban Institute of Technology.

I, Jennifer Campbell, do declare that this dissertation is representative of my own work in both conception and execution, except where specific assistance is sought and duly acknowledged

Jennifer Campbell **_Date_**

APPROVED FOR EXAMINATION

Supervisor **_Date_**

Dr. C Korporaal (M.Tech: Chiropractic, CCSP, CCFC, ICCSD)

Co-supervisor **_Date_**

Dr. R White (M.Tech Chiropractic)

DEDICATION

I dedicate this dissertation to my family who has given me incredible support, guidance and encouragement through out my life. You have given me the greatest opportunity and without you I would not be where I am today. For that I am eternally grateful.

Thank you to Jay for your unwavering support and belief in me. Your constant motivation, as well as patience in me, during my studies, and this dissertation, has helped keep me going and for that I am very appreciative.

ACKNOWLEDGEMENTS

My thanks go to the following people:

- My supervisor, Dr. Charmaine Korporaal, thank you for your constant support and willingness to help. I appreciate the time you've dedicated to me in helping me throughout my dissertation. Without your valuable input this dissertation would not have been possible. Special thanks as well to all your guidance and excellent mentorship since 1st year.

- My co-supervisor, Dr. Rowan White, thank you for your input, encouragement and time taken in helping me with my dissertation.

- To Pat and Linda in reception - thank you for all your help and support in my years at the clinic. It was a pleasure working with you, and I am going to miss those decadent tea parties.

- Mrs. Ireland, thank you for your help and effort in operating the research program so efficiently.

- All the patients, who participated in my study – without your participation, none of this, would have been possible.

- To all the clinicians at Durban University of Technology (DUT), lecturers and colleagues, I thank you for your encouragement and assistance throughout my time at DUT, and those in the clinic throughout the research process.

- All my fellow classmates, without your support this would not have been possible.

ABSTRACT

Objectives

The objectives were to compare the relative effect of manipulation and core rehabilitation in the treatment of acute mechanical lower back pain in athletes.

Project Design:

The study design was a randomized controlled parallel group trial. A quantitative study was performed, by making use of a pre – and post experimental investigation (Nansel et al. 1993 and Naidoo, 2002).

Setting:

Participants presenting with acute low back pain with an onset of 7 days or less, to the Chiropractic Day Clinic at the Durban University of Technology.

Subjects:

Thirty athletic participants, either male or female, between the ages of 18 and 45 years presented at the initial consultation which included participant screening and establishment of their suitability for the study. These were then divided into either group A (which received a manipulation) or group B (which received core exercises).

Outcome measure:

A correct contraction of the core stability muscles was maintained, with a decrease in pressure (in mm Hg) on a Pressure Biofeedback Unit, and an increase in length of time (in seconds).

Results:

It was found that there was no significant difference between the manipulation and the core rehabilitation groups. Although both groups showed improvement with regards to their acute mechanical low back pain, the core rehabilitation group improved at a significantly faster rate than the manipulation group with regards to endurance on the stabilizer.

Conclusions:

Both treatments were equally beneficial for most of the quantitative outcomes measured in this study. However, for the outcome of time on the stabilizer, the core rehabilitation group improved at a significantly faster rate than the manipulation group (p=0.006).

ABBREVIATIONS

AMI	-	Arthrogenic muscle inhibition
BMI	-	Body mass index
EMG	-	Electromyelogram
LBP	-	Low back pain
L1	-	Lumbar vertebrae one
L4	-	Lumbar vertebrae four
SI	-	Sacro-iliac
SMT	-	Spinal manipulative therapy
PBU	-	Pressure biofeedback unit
TrA	-	Transversus abdominus muscle

TABLE OF CONTENTS

CHAPTER THREE – METHODOLOGY AND MATERIALS

CHAPTER FOUR – RESULTS AND DISCUSSION

APPENDICES

LIST OF TABLES

LIST OF FIGURES

Chapter Two

Chapter Four

DEFINITIONS:

ACUTE PAIN

This refers to a pain that has a rapid onset and pronounced symptoms, all of which are of short duration (less than 4 weeks) (Guerriero et al. 1999).

ARTHROGENIC MUSCLE INHIBITION (AMI)

AMI is defined as the inability of a muscle to recruit all motor units of a muscle group to their full extent during a maximum effort voluntary muscle contraction. It is a natural response designed to protect the joint from further damage (Suter et al. 2000).

CLINICAL INSTABILITY

Clinical instability is defined as a significant decrease in the capacity of the stabilizing system of the spine to maintain the intervertebral neutral zones within physiological limits, which results in pain and disability (Panjabi, 1992).

CORE MUSCLE STRENGTH

With regards to this research, muscle strength was determined by how much the participant could reduce the pressure on the PBU, therefore, giving the examiner a negative reading.

CORE STABILITY

It is a generic description for the training of the abdominal and lumbopelvic region (Marshall et al. 2005).

CORE STRENGTHENING / STABILIZATION

This term has come to mean lumbar stabilization, which in essence describes the muscular control required around the lumbar spine to maintain functional stability (Akuthota et al. 2004).

JOINT DYSFUNCTION

Joint dysfunction is depicted as an area showing disturbances of function without structural change, thereby affecting quality and range of motion. It represents disturbances in function that can be represented by a decrease in motion, increase in motion or an abnormal motion (Peterson and Bergman, 2002: 41).

For the purposes of this research, a joint dysfunction includes a decrease in the quality and range of motion, as well as localized muscle spasm and pain to the area of dysfunction (Gatterman, 1995).

MANIPULATION

"A manipulation or adjustment is a passive manual manoeuvre during which the joint complex is suddenly carried beyond the normal physiological range of movement and through the elastic barrier without exceeding the boundaries of anatomical integrity. The usual characteristic is a dynamic specific thrust of controlled velocity and amplitude given at the end of a normal passive range of movement to exceed this elastic barrier into the range of the para-physiological space. It is usually accompanied by a cracking noise" (Sandoz, 1976 and Vernon et al. 2005)

MECHANICAL LOW BACK PAIN (LBP)

Low back pain is pain, muscle tension or stiffness localised below the costal margin and above the inferior gluteal folds, with or without leg pain (sciatica) and is defined as acute when it persists for less than 12 weeks (Van Tulder et al. 1997 and Magee, 2002).

PALPATION

Palpation is a procedure in which the hands are used to assess the mobility of the joints. It is the palpatory diagnosis that covers a collection of manual examination techniques used to assess tenderness, shape, size, consistency, position and inherent mobility of the tissues beneath (Gatterman, 1990: 63).

Palpatory procedures are divided into static and motion palpation. Static palpation is performed with the patient in the stationary position, whereas motion palpation is performed during active and passive joint movement, in addition to involving the evaluation of accessory joint movements (Gatterman, 1990: 63).

CHAPTER ONE

1. INTRODUCTION

1.1 THE PROBLEM AND ITS SETTTING

Lower back pain (LBP) is a major international problem and there are epidemiological and statistical studies documenting the high incidence and prevalence of LBP (Manga et al. 1993). Surveys suggest that the lifetime prevalence of LBP ranges from 60-90% with a 5% annual incidence (Kirkaldy-Willis and Burton, 1992). Similarly the lifetime incidence of LBP in Indian and Coloured communities in South Africa was found to be 78.2% and 76.6% respectively (Docrat, 1999) and in Black South Africans, it was found to be 57.6% (Van der Meulen, 1997).

This may result from a steady reduction in physical activity associated with, or as a result of, the modern fast paced life for most individuals. This could be attributed to a decrease in manual labour and increase in labour-saving devices, which has resulted in the once strong muscle system that is responsible for maintaining our “good’ postures and movements, becoming progressively more inactive as time goes by - resulting in weakened core stability in many individuals. This commonly results in an increasing incidence of LBP (Back Facts, 2000).

The assumption in the literature is that athletes are protected from back pain due to their higher level of abdominal muscle strength (Biering-Sorensen, 1984). It is also thought that people with an increased level of physical fitness from sports participation have a lower risk of LBP as a lack of exercise may play an important role in the development of LBP (Descarreaux et al. 2002). This is due to the fact that most people believe regular exercise will improve their appearance and general health, but few realize the positive effects that good physical conditioning can have on their lower back pain (Biering-Sorensen, 1984). Exercise - based rehabilitation programs that aim to improve "stability" of the lumbar spine are utilized widely in the management of patients with LBP (Richardson and Jull, 1995; Taylor and O'Sullivan, 2000; McGill, 2001; Richardson et al. 2004). These exercises which target the core muscles in the early phases of management (Hides et el. 2001), can reduce LBP intensity, improve functional disability, mobility and endurance (Arokoski, 2001).

LBP has frequently been found to be one of the major causative factors in reduced training time in endurance athletes (Bono, 2004). This may be due to the hypothesized relationship between poor co-ordination of paraspinal muscles and LBP. These changes are suggested to be due to a de-conditioning syndrome, resulting from disuse, pain and illness, as well as muscle spasm and reflex inhibition of the core muscles (Arokoski et al. 2001).

This concurs with the previous work of Panjabi (1992) who indicates that the spinal stabilization system consists of three subsystems- the active subsystem, the passive subsystem and the neural control subsystem. A dysfunction, disease or injury in any one of these parts may lead to spinal instability. This is defined as an increase in the neutral zone around a joint or a decrease in joint stiffness, leading to excessive muscular activity which is required to prevent injury (Liebenson, 1997). Jull and Richardson (2000) indicate that the functional unit that is frequently associated with the development of LBP due to impairments in muscle strength and motor control are the core stabilizers, which are required to maintain normal posture and stability during periods of activity. This is supported by Evans and Oldreive (2000), who found that patients with LBP had a reduced endurance and protective ability of TrA. In addition, it was noted that there was wasting and inhibition of the other core stabilizer and co-contractor - Multifidus in chronic LBP (Hides et al. 1994). Thus, a comprehensive strengthening or facilitation of these muscles has been advocated as a way to prevent and rehabilitate various lumbar spine and musculoskeletal disorders, as well as a way to enhance athletic performance (Akuthota and Nadler, 2004).

Based on clinical presentation, treatment programs have primarily focused on pain reduction, that often only provides temporary relief. If, however, impairments of the deep muscles of the trunk and back are present, these muscular impairments may possibly persist after pain has dissipated and return to manifest as chronic LBP at some later stage (Jull and Richardson, 2000).

Randomized clinical controlled trials indicate the use of manipulation as one of the most effective approaches in the treatment of patients with LBP of mechanical origin (Di Fabio, 1992; Bronfort et al. 2004).

A recent study in the rehabilitation and physical activity literature has emphasized core stability exercises for acute LBP, and as balance, strength and flexibility improves, the episodes and intensity of acute LBP diminish (Graves et al. 2004). This is in line with the suggestions that correction of predisposing factors such as poor posture, faulty lifting techniques, abnormal biomechanics, muscle inflexibilities and poor conditioning is essential to treatment, especially to athletes (Drezner and Herring, 2001).

More recent research however, could not conclude that a combination of manipulation and stabilization was more effective than manipulation alone (Boden, 2002). This proposed research therefore, endeavoured to assess a rehabilitation program when compared with manipulation in athletes for the treatment of acute mechanical LBP.

1.2 AIMS AND OBJECTIVES OF THE STUDY

The aim was to compare the relative effect of manipulation and core rehabilitation in the treatment of acute mechanical back pain in athletes.

1.2.1 Objectives

1.2.1.1 Objective one

The first objective was to determine the relative effect of manipulation (group A) and core rehabilitation (group B) in athletes with acute mechanical LBP in terms of the subjective findings.

Hypothesis 1

It was hypothesized that manipulation and core rehabilitation would have no effect on the pain experienced by the athletes with acute mechanical LBP. No significant difference was expected to be found between the two groups.

1.2.1.2 Objective two

The second objective was to determine the relative effect of manipulation (group A) and core rehabilitation (group B) in athletes with acute mechanical LBP in terms of the objective findings.

Hypothesis 2

It was hypothesized that manipulation and core rehabilitation would have no effect on core muscle strength and endurance in athletes with acute mechanical LBP. No significant difference was expected to be found between the two groups.

1.2.1.3 Objective three

The third objective was to determine any correlations between the subjective and objective outcomes for Group A and Group B.

Hypothesis 3

It was hypothesized that the improvement of core muscle strength and / or endurance would not correlate with the clinical indicators i.e. decrease in NRS, RMQ, as well as surface EMG readings and an increase in algometer readings.

1.3 RATIONALE AND BENEFITS

1. Health Researchers have shown that LBP is one of the most costly health problems in the world today (Manga et al. 1993). “However, the cost effectiveness of chiropractors addressing LBP is overwhelming when compared with medical and other forms of therapy” (Bronfort et al. 2004). Therefore, chiropractors can help in resolving this costly health problem.

2. Core stability has been indicated to have an effect on mechanical lower back pain, hence a relationship between core stability and LBP may exist (Hodges et al. 1996), although causality cannot be inferred.

3. Manipulation has been shown to be effective in the treatment of LBP (Bronfort et al. 2004), and endurance training of the core muscles has been recognized as a crucial preventative measure of first time and recurrent LBP (Liebeson, 1997). In athletic endeavours, muscle endurance appears to be more important than pure muscle strength (Akuthota and Nadler, 2004). However, core rehabilitation as a primary intervention has yet to be compared to manipulation in order to determine the relative clinical effectiveness of each modality.

4. Emphasis is placed on training and strengthening of extremities involved in a particular activity in most sports training routines and, although this is beneficial, without sufficient trunk strength and stability, the strength of the limbs cannot be effectively applied (Hedrick, 2000).

5. Despite its widespread use, research into core strengthening is meagre (Akuthota and Nadler, 2004). Hence, this study aims to contribute to the body of knowledge pertaining to core muscle strength and its relationship to LBP in athletes.

1.4 CONCLUSION

In the remaining chapters, the researcher will outline pertinent literature around the topic that will be presented in chapter 2. Chapter 3 will describe the methodology of the study in detail and chapter 4 will present the statistics, results and subsequent conclusions drawn. Chapter 5 will present the final analysis and the recommendations.

CHAPTER TWO

2. REVIEW OF THE RELATED LITERATURE

2.1 INTRODUCTION

In this chapter, I will provide an overview of the current literature that is relevant to this study. I will outline the appropriate anatomy and biomechanics of structures related to LBP, as well as the current epidemiological data on LBP, followed by treatment options. Finally, I will highlight the basic concepts and theories related to core stabilization and its association with LBP.

2.2 INCIDENCE AND PREVALENCE OF LOWER BACK PAIN

LBP is a major international problem and there are epidemiological and statistical studies documenting the high incidence and prevalence of lower back pain. Surveys suggest that the lifetime incidence of LBP ranges from 60-90% with a 5% annual incidence. For persons younger than 45 years, mechanical LBP represents the most common cause of disability and it is the third most common cause of disability in persons aged older than 45 years (Manga et al. 1993 and Hills, 2005).

Similarly, Docrat (1999) studied the epidemiology of LBP in Indian and Colored communities in South Africa and found the lifetime prevalence to be 78.2% in the Indian community and 76.6% in the Colored community whilst the lifetime incidence of LBP in Black South Africans was found to be 57.6% (Van der Meulen, 1997). While there is no mortality associated with mechanical LBP, morbidity in terms of lost productivity, use of medical services and cost to society is staggering (Hills, 2005).

The three most common types of LBP, which could either be acute or chronic, or traumatic or non-traumatic, are lumbar facet syndrome, sacro-iliac syndrome and lumbar radicular syndrome, which may be discogenic or biomechanical in origin (Schaefer and Faye, 1990). The causes of LBP in active people are diverse, with the lower back being a frequent site of injury in a number of sports including; running, dancing, swimming and soccer etc. (Drezner and Herring, 2001). With running being one of the most popular recreational activities (Wang et al. 1993), it is estimated that 36% of these athletes suffer from musculoskeletal injuries associated with this sport. Various reasons for this muscle tightness have been postulated, however, and more importantly, this muscle tightness is considered a predisposing factor in muscle injuries (Wang et al. 1993). Therefore, the more an athlete runs (increasing factor exposure), the greater the likelihood of them developing muscle tightness, which can predispose to LBP (Wang et al. 1993).

It is therefore essential for the clinician to understand the basic anatomy and pathomechanics of the lower back region, in order to understand the mechanical syndromes. In this respect, the anatomy of the low back will now be presented.

2.3 ANATOMY

The lumbosacral spine consists of five lumbar vertebrae, the sacrum and the coccyx. It is an intricate structure comprising of bony elements, linked by ligaments and joint capsules and is governed by layers of muscles (Kirkaldy-Willis and Burton, 1992).

2.3.1 BONY ANATOMY

2.3.1.1 The lumbar spine

The five lumbar vertebrae are identified by their large bodies, sturdy laminae and absent costal facets. These vertebrae have large kidney shaped bodies, which increase in size from L1 to L5 as the load that they support increases towards the inferior end of the vertebral column (Moore and Dalley, 1999).

The vertebral arch is a horseshoe-shaped structure formed by the laminae and pedicles. There are seven processes that project from this arch. These are the paired superior and inferior articular processes, the spinous process and two transverse processes (Kirkaldy-Willis and Burton, 1992). The thick and broad spinous processes are hatchet-shaped and point posteriorly. The transverse processes, originating from the laminae-pedicle junction, are slender, long and flattened on their anterior and posterior surfaces (Moore and Dalley, 1999). The transverse process together with the spinous process serves as a site for muscles and ligaments to attach (Kirkaldy-Willis and Burton, 1992). The articular processes, which are also large, thick and strong, facilitate flexion, extension and lateral bending of the spine. However, they prohibit rotation (Moore and Dalley, 1999).

Interactive Spine - Chiropractic edition ©
2001 Primal Pictures Ltd

Figure 1: The Lumbar Spine

(Permission received from Primal Pictures Ltd: Copyright Primal Pictures Ltd)

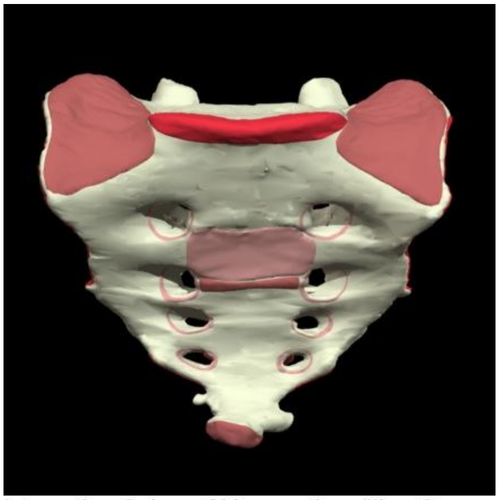
2.3.1.2 The sacrum

The sacrum is a fusion of the five sacral segments that results in a triangular wedged-shaped bone. The sacral base, which is formed by the S1 superior surface, has two superior facets that articulate with L5. Laterally, it articulates with the ilium. The sacral apex points downwards to articulate with the coccyx by means of a disc. The sacral tubercles, located in the midline, correlate with the spinous processes of the fused vertebrae, whereas the tubercles on the posterolateral aspect correlate with the transverse processes. The sacrum provides strength and stability to the pelvis and transmits the weight of the body to the pelvis. It supports the vertebral column and forms the posterior part of the pelvis (Moore and Dalley, 1999).

Interactive Spine - Chiropractic edition ©
2001 Primal Pictures Ltd

Figure 2: The Sacrum

(Permission received from Primal Pictures Ltd: Copyright Primal Pictures Ltd)



Interactive Spine - Chiropractic edition ©
2001 Primal Pictures Ltd

Figure 3: The Sacrum

(Permission received from Primal Pictures Ltd: Copyright Primal Pictures Ltd)

2.3.2 THE LUMBAR ZYGOPOPHYSEAL JOINT (lumbar facet joint)

2.3.2.1 Anatomy

The lumbar zygopophyseal joint is a typical synovial joint. This joint is formed by the articulation between the superior articular process of the vertebral body below and the inferior articular process of the vertebral body above, and is classified as a synovial (diarthrodial) planar joint (Moore and Dalley, 1999). The lumbar facet joints are biplanar, with the major posterior parts approximated to the sagittal plane. The exception is the lower lumbar facet joints that rotate toward the coronal plane at the lumbosacral junction (Giles and Singer, 1997). Each joint is surrounded by an articular capsule posterolaterally and this capsule is thick and fibrous and covers the dorsal aspect of the joint. The anterior and medial aspects of the joint are covered by the ligamentum flavum. The accessory ligaments unite the laminae, transverse process and spinous process in order to help stabilize the joint (Moore and Dalley, 1999). The synovial membrane lines the articular capsule, ligamentum flavum and the synovial joint folds supply the joint surface with synovial fluid (Giles and Singer, 1997).

2.3.2.2 Function

The function of the lumbar zygopophyseal joints' is to control patterns of motion between the vertebrae, to protect the discs from shear forces, excessive flexion and axial rotation, as well as to provide support to the spinal column (Giles and Singer, 1997).

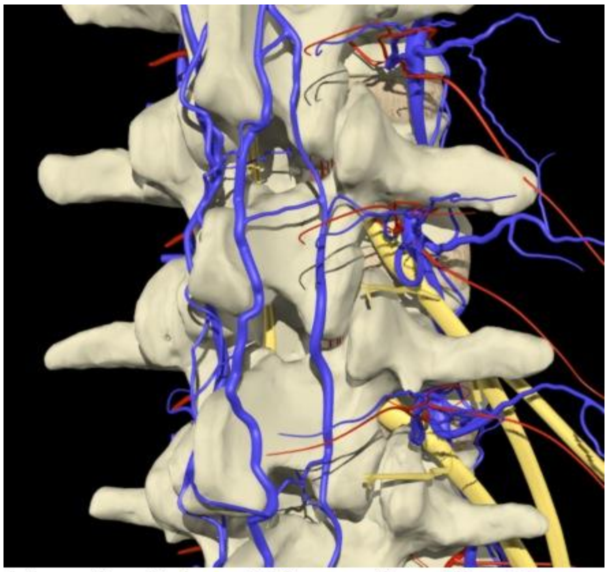
2.3.2.3 Innervation

The lumbar zygopophyseal joints' Innervation comes from the medial branch of the dorsal primary rami of the spinal nerves. Each articular branch supplies two adjacent joints, thereby supplying each joint with two nerves (Moore and Dalley, 1999). The median branch supplies the level of exit and the level below (Giles and Singer, 1997). Based on Hilton's law, the related connective tissue, muscles, skin and ligaments over a joint are supplied by the nerves to that joint. This implies that the neurological input and output of the joint will affect the surrounding structures and visa versa (Moore and Dalley, 1999).

There are three types of sensory receptors within the zygopophyseal joint (Leach, 1994; Gatterman, 1995):

Type: I	Globular corpuscles in the outer layers of the fibrous capsule. Very sensitive static and dynamic mechanoreceptors that continually fire even when the joint is not moving.
Type: II	Conical corpuscles in the deeper layers of the fibrous capsule. Less sensitive mechanoreceptors that fire only during movement.
Type: III	Larger corpuscles on the surface of the joint ligaments, thinly encapsulated mechanoreceptors.
Type: IV	Unmyelinated nerve fibers that weave throughout the capsule. Slow conducting nociceptive mechanoreceptors.

These receptors are often the origin of LBP when stimulated by noxious stimuli and thus result in the patterns of presentation of LBP seen in patients. This presentation will now be discussed in terms of the facet joint, a condition known as lumbar (posterior) facet joint syndrome (Kirkaldy-Willis and Burton, 1992).



Interactive Spine - Chiropractic edition ©
2001 Primal Pictures Ltd

Figure 4: Innervation of the Lumbar Facet Joint

(Permission received from Primal Pictures Ltd: Copyright Primal Pictures Ltd)

2.3.3 LUMBAR (POSTERIOR) FACET SYNDROME (facet syndrome)

2.3.3.1 Presentation

With lumbar facet joint syndrome, the pain is often localized and unilateral at the site of the facet joint involved (Kirkaldy-Willis and Burton, 1992). However, the classic presentation is generally LBP which may be referred to the groin, hip, buttock and posterior thigh to above the knee - mimicking radicular pain (Gatterman, 1990). In addition, pain of scleratogenous origin may account for a non-diffuse deep pain, where radicular pain is very specific allowing the patient to accurately trace the precise route of the involved nerve root (Giles and Singer, 1997). This presentation is thought to be secondary to the level of innervation (that is noxiously stimulated) of the facet joint involved together with the facet joint above and facet joint below (as these receive innervation from the same lumbar nerve root segment that is involved) (Sandoz, 1978).

2.3.3.2 Associated clinical signs

The classic signs are palpable muscular spasm with focal tenderness over the affected facet joint(s), with hyperextension movements of the back increasing the pain, whereas flexion decreases the pain. Activities that may increase the pain comprise of: sleeping on the abdomen, sitting in an upright position, rising from the seated position, lifting a load in front of the body at (or above) the waistline, and working with the hands above the head. When the symptoms become acute, sneezing and coughing may increase the pain (Gatterman, 1990).

2.3.4 THE SACRO-ILIAC JOINT (SI joint)

2.3.4.1 Anatomy

The SI joint is a synovial joint formed by the articulation between the sacrum and the ilium. The articular cartilage on the sacrum is thicker than that found on the ilium. The cartilage on the sacrum is hyaline cartilage, whereas the cartilage on the ilium is fibro-cartilage (Kirkaldy-Willis and Burton, 1992).

A number of strong ligaments and structures aid in stabilizing this atypical synovial joint (Peterson and Bergmann, 2002):

- Powerful interosseus ligament
- A strong articular capsule
- Posterior sacro-iliac ligaments
- Anterior sacro-iliac ligaments
- Iliolumbar ligament

- Sacrotuberous ligament and
- Sacrospinous ligament.

2.3.4.2 Function

The SI joint is a strong weight bearing synovial joint and differs from other synovial joints in that it possesses little mobility due to its role in transmitting most of the weight of the body to the hip bones. Due to the irregular surface of the articulating bones, the sacrum and the pelvis, and the thick interosseous and posterior sacro-iliac ligament, movement of the joint is restricted. Movement is limited to slight gliding and rotary movements. Except when a considerable force is applied, the force is transmitted through the vertebrae to the sacrum, which rotates anteriorly. The force is then transmitted to each ilium and then the lower limbs (Moore and Dalley, 1999).

2.3.4.3 Innervation

The articular branches of these joints are derived from the superior gluteal nerves bilaterally, the sacral plexus and the dorsal rami of S1 and S2 (Moore and Dalley, 1999). The posterior aspect of the sacro-iliac joint comes from branches originating from the posterior primary rami of L4-S2 spinal nerves (Kirkaldy-Willis and Burton, 1992), whereas the anterior aspect is innervated by the posterior branches from the L3-S2 nerve roots and the superior gluteal nerve L5-S2.

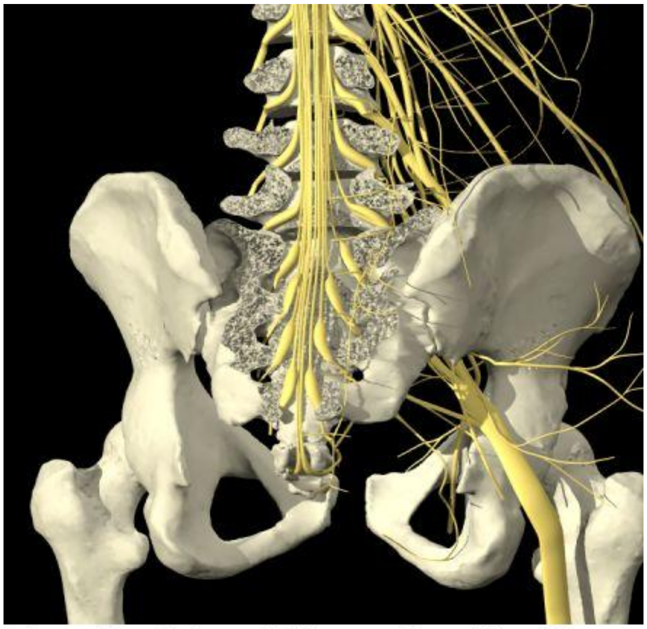


Figure 5a: Innervation of the Sacro-iliac Joint

(Permission received from Primal Pictures Ltd: Copyright Primal Pictures Ltd)

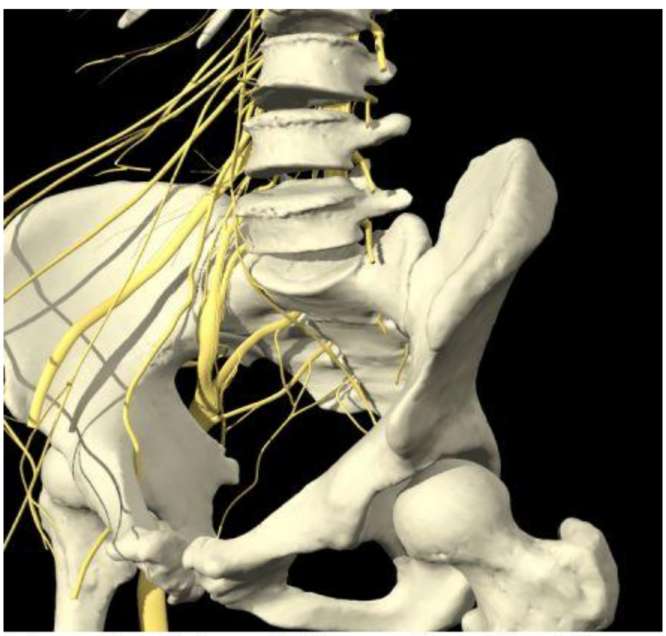


Figure 5b: Innervation of the Sacro-iliac Joint

(Permission received from Primal Pictures Ltd: Copyright Primal Pictures Ltd)

Mechanosensitive afferent units have been identified in the Sacro-iliac (SI) joint and adjacent tissues. Most of these units are nociceptive receptors (Sakamoto et al. 2001). These receptors are often the origin of LBP when stimulated by noxious stimuli and thus result in the patterns of presentation of LBP seen in patients. However, with the vast innervation that is available to the SI joint, the presentation of sacro-iliac syndrome is varied and can present in many different ways, dependant on the site of the biomechanical lesion and the resultant irritation of the receptors. The presentation of sacro-iliac syndrome will now be discussed.

2.3.5 SACRO-ILIAC SYNDROME

2.3.5.1 Presentation

Sacro-iliac syndrome presents with pain over one SI joint in the area of the posterior superior iliac spine. The pain may also radiate to the groin and anterior thigh or posteriorly down the thigh (thought to be innervation dependant). Occasionally the pain may be felt down the lateral or posterior calf to the ankle, foot and toes (Gatterman, 1990).

2.3.5.2 Associated clinical signs

There is tenderness or pressure over the SI joint and / or the buttock. Movement of the joint is usually restricted. The diagnosis may be confirmed by a successful manipulation of the SI joint or injection of the joint (Kirkaldy-Willis and Burton, 1992). The pain is also aggravated by provocation tests (McCullach and Transfeldt, 1997).

2.4 BIOMECHANICS OF THE LOWER BACK

The lumbar (posterior) facet syndrome is most commonly present due to changes in the biomechanics of the lumbo-sacral spine. However, in order to understand this, one needs to know that the lumbar and sacral spines link the lower extremities and the torso, as well as co-ordinating the transfer of power through the body in most sports via the kinetic chain (Drezner and Herring, 2001).

The main movements of the lumbar spine include flexion, extension, lateral flexion and rotation. At the lower two lumbar vertebrae, flexion and extension predominantly occur. Rotation at each lumbar vertebra is limited to a few degrees due to the vertical orientation of the facet joints. Combined flexion and rotation carry the highest injury potential (Kirkaldy-Willis and Burton, 1992).

This is compared to the sturdy SI joint that is surrounded by some of the largest, and most powerful muscles in the body, but none of these cross the joint or are known to directly influence joint movement. However, contraction of these muscles (erector spinae, psoas, quadratus lumborum, piriformis, rectus femoris and gluteus maximus, minimus and medius) will place shear and moment loads on the joint surfaces, hence influencing any movement at the SI joint (Cassidy and Mierau, 1992).

Individual risk factors that can lead to changes in the biomechanics include; (a) increasing age, (b) increase in manual labour, (c) lack of fitness, (d) poor health, (e) degeneration, (f) exposure to vibration, (g) smoking, (h) psychological problems and (i) posture, to name a few. These changes in the biomechanics often result in inflammation, due to joint irritation and pain, or the muscle spasm, or both. This pain and / or discomfort has been associated with the patient being unable to recruit all muscle fibres within muscles that cross the joint involved, thereby creating either a perceived or actual weakness in the muscle. This compromise of the muscles' functional ability perpetuates the presence of the LBP and results in a negative pathomechanical spiral in terms of the patient's presenting complaint (Kirkaldy-Willis and Burton, 1992).

2.5 ARTHROGENIC MUSCLE INHIBITION (AMI)

AMI is the inability of a muscle to recruit all motor units of a muscle group to their full extent during a maximal effort voluntary muscle contraction and is a natural response designed to protect the joint from further damage (Suter et al. 2000). It is thought that mechanoreceptor activity plays the primary role in AMI (Leach, 1994; Hopkins and Ingersoll, 2000).

AMI results from the activity of many different mechanoreceptors (Leach, 1994) located in joint capsules, ligaments and tendons (Levangie and Norkin, 2001). These include: Ruffini endings, Golgi-like endings and Pacinian corpuscles. Also pain receptors, such as free-nerve endings, are found throughout the joint tissue and are active with any joint damage. It is hypothesised that these receptors play a significant role in AMI, as these mechanoreceptors act on inhibitory interneurons synapsing on the motor neuron (MN) pool of the joint musculature, decreasing the force of any contraction stemming from that MN pool (Hopkins and Ingersoll, 2000). This suggests that exercise in patients with pain does not achieve improved clinical outcomes.

This is in contrast to the concept that spinal manipulation is thought to be able to modulate the function of the nervous system and thereby affect AMI. It has remained within the realms of philosophy (Wyke, 1985), where only the clinical outcomes of the neurophysiological effects of spinal manipulation have been observed in body segments distant from where the manipulations were performed (Suter et al. 1994; Suter et al. 1999; Suter et al. 2000; Naidoo, 2002). Based on the outcomes of the above studies and in support of the theory proposed by Wyke (1985), it is noted that articular mechanoreceptor afferent nerve fibres give off collateral branches that are distributed both intersegmentally and intra-segmentally. Therefore, manipulation of an individual joint is not only thought to affect the motor unit activity in the muscles operating over the joint being manipulated, but also in more remote muscles. This is based on the research that indicates that manipulation of a joint has been proposed to activate mechanoreceptors from structures in, and around, the manipulated joint. The altered afferent input arising from the stimulation of these receptors is thought to cause changes in the motor neuron excitability, with a subsequent decrease in arthrogenic muscle inhibition (William, 1997 and Suter et al. 2000). This suggests that manipulation in patients with pain does achieve improved clinical outcomes, which is in contrast to the outcomes for exercise.

2.6 SPINAL MANIPULATIVE THERAPY (SMT)

Spinal manipulation is a form of manual therapy which involves movement of a joint beyond its usual end range of motion, but not beyond its anatomic range of motion (Bergmann and Peterson, 2002). Spinal manipulation has been shown to have both statistically significant and clinically important results, with regards to improvements of acute LBP (Van Tulder et al. 2005).

Hypotheses for the working mechanism of spinal manipulation (Van Tulder et al. 2005) include:

a) Release of entrapped synovial folds

b) Relaxation of hypertonic muscles

c) Disruption of articular or peri-articular adhesions (Gatterman, 1990; Mense, 1991).

d) Unbuckling of motion segments that have undergone displacement

e) Reduction of disc bulge

f) Mechanical stimulation of nocioceptive joint fibres (Melzack and Wall, 1965).

g) Reduction of muscle spasm (Leach, 1994)

h) Change in neurophysiological function. (Gatterman, 1990; Mense, 1991).

Bold = principle functions which are associated with the reduction in pain.

Gattermann (1995) states that the treatment of choice for sacro-iliac syndrome is specific manipulative therapy directed at the sacro-iliac articulations. Clinical studies have shown a successful response to manipulation in more than 90% of patients ((Van Tulder et al. 2005; Gattermann, 1995). There is clear evidence to justify the use of manipulative therapy in the treatment of patients with lumbar facet syndrome and/or sacro-iliac syndrome (Di Fabio, 1992).

The core muscles, which serves as a muscular corset, works as a unit to stabilize the body and spine, thereby serving as the centre of the functional kinetic chain (Akuthota and Nadler, 2004). However, poor conditioning of the extremities places the athlete at a greater risk of low back injury (Drezner and Herring, 2001).

2.7 ANATOMY OF THE CORE MUSCLES

The core muscles serve as a “muscular corset” working as a unit to stabilize the body and spine, essentially serving as the centre of the functional kinetic chain (Akuthota and Nadler, 2004). The importance of effective core stability cannot be overestimated with regards to performance, injury prevention and recovery. Ineffective core stability causes us to place excessive stresses on the body by moving improperly. A comprehensive strengthening or facilitation of the core muscles is believed to prevent and rehabilitate various lumbar spinal and musculoskeletal disorders as a way to enhance athletic performance (Akuthota and Nadler, 2004).

2.7.1 MUSCLE SYSTEMS

Bergmark (1989) categorized the trunk muscles into local and global muscle systems based on their main mechanical roles in stabilization. These two functional muscle systems are linked to spinal stabilization (Jull and Richardson, 2000). A controlled co-operation between the local and global stability system has to be used in order to provide core stability (Stevens et al. 2006).

2.7.1.1 GLOBAL MUSCLE SYSTEM

The muscles of the global stability system consist primarily of the larger, superficial torque producing muscles, such as the Rectus Abdominus, Internal and External Oblique, Quadratus Lumborum, Erector Spinae and so on, which are found around the abdominal and lumbar region (Marshall and Murphy, 2005). These muscles are responsible for movement, controlling and balancing the forces that act on the body and general trunk stability (as they are not attached directly to the spine) (Jull and Richardson, 2000). These muscles balance external loads in a way to help reduce the resulting forces on the spine. They are the muscles that keep one upright (Richardson and Jull, 1995).

Table .1 Attachments and innervations of the main muscles of the global stability system.

MUSCLE	ORIGIN	INSERTION	INNERVATION
External Oblique	External surfaces and inferior borders of 5th-12th ribs.	Linea alba in midline, pubic tubercle and anterior half of iliac crest.	Inferior 6 thoracic nerves and subcostal nerve.
Internal Oblique	Lateral half of the inguinal ligament, the anterior two-thirds of the iliac crest and thoracolumbar fascia.	Cartilages of the 10th-12th ribs, linea alba and pubis through the conjoined tendon.	Ventral rami of inferior 6 thoracic nerves and the first lumbar nerves.
Rectus Abdominus	Pubic crest and pubic symphysis.	Costal cartilage of the 5th-7th ribs and xiphoid process.	Ventral rami of inferior 6 thoracic nerves.
Quadratus lumborum	Medial half of inferior border of 12th rib and the tips of the first 4 lumbar transverse processes.	Internal lip of iliac crest and iliolumbar ligament.	Branches of lumbar plexus arising from T12 and L1-L4 spinal nerves.

(Table compiled from Moore and Dalley, 1999; Travell and Simons, 1999).

Functions and actions of the global muscles

These anterolateral abdominal muscles form a strong support for the anterolateral abdominal wall, protect abdominal viscera, aid in respiration as well as help to move the trunk and maintain posture (Moore and Dalley, 1999). The External and Internal Oblique muscles work bilaterally, forming a muscular girdle to increase the intra-abdominal pressure and to flex the trunk. They work unilaterally to bend the trunk toward the same side and assist in trunk rotation. The Rectus Abdominus functions as a prime mover for spinal flexion and compresses abdominal viscera (Travell and Simons, 1999). It also stabilizes the pelvis during walking and when performing lower limb lifts from the supine position. It prevents tilting of the pelvis by the weight of the limbs (Moore and Dalley, 1999).

The actions of the Quadratus Lumborum are to control lateral flexion to the opposite side. Stabilization of the lumbar spine on the pelvis by the Quadratus Lumborum is important in that the complete paralysis of this muscle makes walking impossible. This muscle also aids in stabilizing the last rib for respiration. Unilaterally, with the pelvis fixed, the Quadratus Lumborum acts as a lateral flexor to the same side. With the spine fixed, unilateral contraction elevates the ipsilateral hip. Bilateral functioning of the Quadratus Lumborum extends the lumbar spine (Travell and Simons, 1999).

2.7.1.2 LOCAL MUSCLE SYSTEM

The muscles of the local stability system consist of the deep intrinsic muscles of the abdominal wall, such as the Transversus Abdominis (TrA), Multifidus, the pelvic floor muscles and the diaphragm. These muscles lie close to the spine and are responsible for sensory feedback, support and are associated with segmental stability of the lumbar spine (Marshall and Murphy, 2005). Due to their vertebrae to vertebrae attachments, they control the finer movements of the adjacent vertebrae (Stevens et al. 2006). The local system has a primary responsibility of segmental stability, with both the TrA and Multifidus being important components (Richardson and Jull, 1995).

Table.2 Attachments and innervations of the main muscles of the local stability system.

MUSCLE	**ORIGIN**	**INSERTION**	**INNERVATION**
Transversus Abdominis	Lateral third of the inguinal ligament, iliac crest, thoracolumbar fascia and the internal surfaces of 7th-12th costal cartilages.	Midline linea alba via the rectus sheath and to the pubis through the conjoined tendon.	Branches from the 8th-12th intercostals nerves innervate the TrA as well as the first lumbar nerves.
Multifidus	Base of a vertebral spinous process	Fibers cross 2-4 segments throughout the thoracic and lumbar spine and attach to a transverse process.	Dorsal primary rami of spinal nerves. The lumbar multifidi are arranged so that fibres moving a particular segment are innervated by the nerve of that segment.

(Table compiled from Moore and Dalley, 1999; Travell and Simons, 1999).

Functions and actions of the local muscles

Amongst the core stabilization muscles, the TrA and the Multifidus muscles are the main functional muscles acting as strong stabilizers. These muscles lie deep within the trunk of the body acting like a corset and reducing pressure on the spine (Davis et al. 2004). These muscles are capable of making major contributions to spinal stability. They are ideal for controlling intersegmental motion as they are closer to the centre of rotation of the spinal segments and have shorter muscle lengths (Richardson et al. 1999).

The TrA is controlled independently of other trunk muscles, allowing it to be functionally isolated from other abdominal muscles. Furthermore, it contracts with all trunk movements regardless of the initial direction of movement and it is recruited prior to all the abdominal muscles. The TrA also has a direct link to the development of intra-abdominal pressure (Richardson and Jull, 1995) by compressing and supporting the abdominal viscera (Travell and Simons, 1999). A study of the TrA found that LBP patients had reduced TrA endurance and that its protective ability was decreased (Evans and Oldreive, 2000). It was also noted that there was wasting and inhibition of the Multifidus in patients with LBP (Hides et al. 1994).

The lumbar Multifidus acting bilaterally, extends and stabilizes the vertebral column (Travell and Simons, 1999). It is shown to contribute to the control of the neutral zone and provides more than two-thirds of the stiffness increase at the L4 and L5 segments (Richardson and Jull, 1995).

These two core stabilizing muscles have been found to be related through a co-contraction pattern. The recruiting muscles that are in co-contraction are considered to provide support and joint stabilization even when contractions occur at lower levels of maximum voluntary contraction (Richardson and Jull, 1995).

2.8 THE SPINAL STABILIZATION SYSTEM

The overall stability of the spinal column is provided by itself, and the co-ordinated surrounding muscles. This is especially seen in dynamic conditions and under heavy loads (Panjabi, 2003). Spinal instability is considered to be one of the most important causes of LBP, though it is poorly defined and not well understood (Panjabi, 1992: l). Spinal instability occurs when abnormally large intervertebral motions cause either compression and/or stretching of the inflamed neural elements or abnormal deformations of ligaments, joint capsules, annular fibres, and end-plates, which are known to have a significant density of nociceptors (Panjabi, 1992: l). This may lead to LBP. Therefore, it can be said that a decrease in intervertebral motion in a patient with LBP may result in reduced pain (Panjabi, 2003).

2.8.1 SUBSYSTEMS OF THE SPINAL STABILIZATION SYSTEM

According to Richardson et al. (1999), the spinal stabilization system is made up of three sub-systems. These are essential in maintaining spinal stability (Panjabi, 1992: l):

1. The passive musculoskeletal subsystem – this includes vertebrae, facet joints, intervertebral discs, spinal ligaments, joint capsules and the passive mechanical properties of the muscles.
2. The active musculoskeletal subsystem – this includes the muscles and tendons surrounding the spinal column.
3. The neutral and feedback subsystem – this includes the various forces and motion transducers, located in the ligaments, tendons, muscles and the neutral control centre.

2.8.2 FUNCTIONING OF THE SPINAL STABILIZATION SYSTEM

The spinal stabilizing system is said to have three basic biomechanical functions, these being (White and Panjabi, 1990):

A) To permit movements between body parts;

B) To carry loads; and

C) For protection of the spinal cord and nerve roots.

In order for these functions to be performed properly, mechanical stability is necessary (White and Panjabi, 1990; Panjabi, 1992). These subsystems may be applied to the core stabilizers and their strength is essential to athletic performance. A normal function of the stabilizing system is to provide sufficient stability to the spine in order to cope with varying stability demands as a result of changes in posture, static and dynamic loads. Although these subsystems are separate, they are functionally interdependent. A dysfunction, injury or disease in any of these subsystems may lead to spinal instability (Panjabi, 1992: l). One of the key issues in the production, perpetuation and management of mechanical LBP, is the challenge to control the intersegmental relationship for normal pain free function (Richardson et al. 1999).

Two parameters of spinal stability are considered due to the multisegmental nature of the lumbar spine. The first being control of spinal orientation, relating to preservation of overall posture, and the second is associated with the control of the intersegmental relationship at a specific level regardless of the overall changes in the spine. Therefore, the integrity of both levels of support is dependant on the efficient stability of the spine (Richardson et al. 1999).

2.8.3 CLINICAL INSTABILITY

Clinical instability is defined as "a significant decrease in the capacity of the stabilizing system of the spine to maintain the intervertebral neutral zones within physiological limits so that there is no neurological dysfunction, no major deformity, and no incapacitating pain" (Panjabi, 1992: II). Within physiological ranges of spinal movements, and against normal spinal loads, these three subsystems are optimized and co-ordinated. Providing the dysfunction does not go beyond a certain limit, compensation may be provided by the system, for a dysfunction in the system. However, beyond this limit, acute or chronic problems may arise (Panjabi, 1992: I; Richardson et al. 1999).

The neutral zone is described as "that part of the range of physiological intervertebral motion, measured from the neutral position, within which the spinal motion is produced with a minimal internal resistance" (Panjabi, 1992: II). Control of intersegmental motion around the neutral zone is a major factor for maintaining spinal stability. Therefore, the sensitivity of and increase in the neutral zone is an indicator of clinical instability (Richardson et al. 1999). Dysfunction within any of the three subsystems can lead to an increase in the size of the neutral zone (Panjabi, 1992: II).

2.8.3.1 Subsystem dysfunctions:-

a) Passive system dysfunction

This may be due to mechanical injury such as, over stretching of ligaments, development of tears in the annulus, micro-fractures in the end-plates and extrusion of disc material into the vertebral bodies. These may all result in a decrease in the load bearing and stabilizing capacity of the passive subsystem (Panjabi, 1992: I).

b) Active subsystem dysfunction

Deterioration of the ability to receive and/or carry out neural commands, in order to provide accurate feedback of the muscle tension information to the neural control unit, or to produce co-ordinated and adequate muscle tensions of the active subsystem may develop as a result of disuse, degeneration, disease and injury. As a result, the stabilizing capacity of the active subsystem may be decreased (Panjabi, 1992: l).

c) Neural subsystem dysfunction

In order to achieve the required stability at every instance of time, the neural subsystem has the complex task of continually and simultaneously monitoring and adjusting the forces in each of the muscles surrounding the spinal column. A fault that may occur is that one or more muscles may fire in an undesirable manner, either too small or too large a force and/or too early or too late firing. This may result in excessive muscle tension, causing soft tissue injury and pain. In addition to damaging the active subsystem, muscle force errors might lead to overload of a passive structure i.e. disc (Panjabi, 1992: l).

2.8.3.2 Adaptations:-

Training enhances the ability to perform complex mechanical tasks. A general increase in muscle tone by training has been shown to decrease the risk for developing lower back problems (Panjabi, 1992: I). The explanation is that this causes enhanced stability of the spinal system in the form of increased capacity to generate muscle tension. The strengthening of selective muscle groups may make up for specific passive stability loss due to an injury (Panjabi, 1992: I).

2.9 CORE STABILIZATION

Core strengthening / stabilization is a term used to indicate lumbar stabilization and motor control training. Essentially, it is a description of the muscle control required around the lumbar spine in order to maintain functional stability (Akuthota and Nadler, 2004).

LBP has been linked with dysfunction of the prime core stabilizer - TrA (Hodges et al. 1996b), and the patients inability to recruit this deep postural muscle of the trunk. The factors that affect lumbar stability have been an area of extensive research. Particular attention has been paid to the core muscles as they serve as a muscular corset to stabilize the body and spine. Ineffective core stability causes one to move improperly by placing excessive stress on the body. A comprehensive strengthening or facilitation of these muscles has been advocated as a way to prevent and rehabilitate various lumbar spine and musculoskeletal disorders, thus enhancing athletic performance (Akuthota and Nadler, 2004). Hence, the goal of rehabilitation is to restore the normal lumbar spine function and promote an independent return to activity (Drezner and Herring, 2001).

It has been hypothesized that the steady reduction in physical activity and the increasing rate of sedentary lifestyles has resulted in the once strong muscle system that is responsible for maintaining our postures and movements, becoming progressively more inactive, resulting in weakened core stability in many individuals. This commonly results in an increasing incidence of LBP (Back Care, 2000). Amongst the core stabilization muscles, the TrA and the Multifidus muscles are the main functional muscles acting as the strong stabilizers. These muscles lie deep within the trunk of the body acting like a corset and reducing pressure on the spine (Davis et al. 2004). A study of the TrA found that LBP patients had reduced endurance and that its protective ability was decreased

(Evans and Oldreive, 2000). In addition, it was noted that there was wasting and inhibition of the Multifidus (Hides et al. 1994).

Achieving core stability is not only a matter of activating a few targeted muscles, but rather as a moving target. This constantly changes as a function of the three dimensional torques needed for support, anticipation of unexpected loads and to ensure sufficient stiffness in any degree of freedom of the joint which may be compromised from injury (McGill et al. 2003).

2.10 THE RELATIONSHIP BETWEEN LBP AND CORE STABILITY MUSCULATURE IN ATHLETES

The link between mechanical instability in the lumbar spine and LBP, together with the association of LBP and muscle dysfunction, sustains the notion of dynamic trunk-stability training. These dynamic stability approaches include increasing muscle strength, increasing endurance and using neuromuscular control strategies to maintain dynamic trunk-stability (Hubley-Kozey and Vezina, 2002). Having a high level of core stability can contribute to athletic performance by aiding in the “efficient transmission of force generated by the lower body through the trunk to the upper body,” whereas, an inability to stabilize this region during running may lead to poor technique and an inefficient force application (Mills et al. 2005).

Impact loading of the lumbar spine is of primary concern when dealing with runners and other sports that involve running. The forces that occur on impact of heel strike are transferred up the kinetic chain to the lumbar spine and to the related supporting structures of the trunk (Hedrick, 2000). Athletic movements, like running, create strenuous forces on the back and if the back has not been trained accordingly, can cause weakness and reduced movement which may lead to impaired athletic performance, injury and pain (Foxhoven and Plante, 1996). There is the misconception that low back injury is due to weak lumbar muscles. In most individuals, these muscles are strong and a weakness of the abdominal muscles is noted more often (Hedrick, 2000). There is a link between LBP and motor control deficits of the core muscles, namely the TrA and Multifidus muscle. These muscles show a decrease in their normal anticipatory function in patients with LBP (Jull and Richardson, 2000). Individuals demonstrated signs of delayed activation of the TrA muscle before rapid upper limb movements, when compared to healthy individuals. These individuals also have an impaired ability to consciously contract the TrA. This is improved with abdominal stabilization training (Marshall and Murphy, 2006).

Trunk strength is vital because all movements either originate, or are coupled, through the trunk and this force (or movement) is transferred through the body in a straight line. This coupling is created by a strong core, which connects movements of the lower body to those of the upper body. A well-developed core allows for improved force output, an increase in neuromuscular efficiency and a decrease in the incidence of overuse injuries. By strengthening the core, the athlete increases their ability to apply the musculature of the lower and upper body in order to perform a task, thereby greatly improving the efficiency and effectiveness of their physical performance (Hedrick, 2000).

This link between LBP and motor control deficits in muscles of the local system, mainly the TrA and lumbar Multifidus is becoming more apparent. These muscles appear to lose their normal anticipatory function in patients with LBP, thereby exhibiting delays in activation and hence a loss of their normal pre-programmed function for support (Jull and Richardson, 2000). Spinal stabilization that involves the co-contraction of the Multifidus and TrA has been shown to be an effective approach to resolving LBP (Newton, 2004).

2.11 CORE STABILITY EXERCISES

The high prevalence of LBP and the functional disability related to it has resulted in the large number of conservative treatment methods. Exercise is one of the most popular approaches to the treatment of LBP (Descarreaux et al. 2002). A well-developed core is essential, when the goal is optimal athletic performance (Stanton et al. 2004). Many training programs emphasize the extremities first, with the trunk being trained at the end of the work out. Without an adequate core strengthening, and a stability program, the athlete will not be able to properly apply extremity strength (Gambetta and Clark, 1999).

Stabilization exercises are designed to improve functioning of the core muscles that govern trunk stability. With these muscles having optimal function, they have been shown to protect the spine from trauma (Stevens et al. 2006). Retraining of the stabilizing muscles, with initial low level isometric activation and integration into functional tasks, is an essential component of back muscle rehabilitation (Koumantakis et al. 2005). Restoration of function in the motor system, and in this case specifically the core stabilizers, is becoming an essential part of LBP treatment and prevention, and is thought to contribute to the improvement of performance in athletes (Barr et al. 2005). It has been noted in recent research, with regard to athletic endeavour's, that it is muscle endurance that is of more importance than muscle strength (Akuthota and Nadler, 2004). Poor endurance of the trunk muscles may induce strain on the passive structures of the spine and eventually lead to LBP. Evidence suggests that muscle endurance is lower for people with LBP than for those without LBP (Chok et al. 1999).

2.11.1 FORMAL EXERCISE PROGRAMS

Any exercise can be a stabilization exercise, but it all depends on the manner in which it is performed. Sufficient joint stiffness is achieved by creating specific motor patterns. Stabilization exercises that are performed properly produce patterns that are practiced and these, in turn, groove motor patterns ensuring a stable spine (McGill et al. 2003). According to Hodges et al. (1996), these aberrant motor patterns in lower back patients compromise the ability of the affected person to stabilize efficiently.

Exercise involving co-contraction and holding ability of the lumbar Multifidus and TrA is sufficient to control lumbar spine position with increasing loads. A simultaneous isometric contraction of these two muscles, whilst maintaining the spine in the neutral position, should help re-educate the stabilizing role of these muscles (Richardson and Jull, 1995; Richardson et al. 1999). An exercise program should include: activating an isometric co-contraction of these core stabilizing muscles and training the patient to hold a low level tonic contraction. Specific exercises should isolate the local muscles as much as possible from the global muscles in order to ensure that the correct muscles are being reactivated (Richardson and Jull, 1995). There is no need for high loaded exercises during initial rehabilitation (Richardson and Jull, 1995). Therefore, the four point kneeling or prone lying exercises are effective (Richardson and Jull, 1995). Performance of any exercise must be preceded by conscious activation of the deep muscles by gently drawing in the abdominal muscles. The emphasis of each exercise is on control, and progression should not be too fast, as too much load too quickly may lead to compensation by the global muscles (Richardson et al. 1999).

Results from the literature indicate that endurance training of the core stabilizing muscles reduces pain and disability in patients with acute LBP, after only three weeks of exercise. This is limited after six weeks, as the improvement is masked by the improvement due to the natural history (Chok et al. 1999).

2.11.2 ARE EXERCISES HARMFUL?

The notion that active exercise can be harmful to the patient with LBP is incorrect, and a study done at the New England Baptist Hospital concluded that anticipated and induced pain with physical activities was lessened after physical therapy using exercise (Rainville et al. 2004). Guided exercise by a qualified individual is often considered the optimal treatment program for the acute and sub-acute population. The exercises that are prescribed should be those with a wide margin of safety / stability and loads under 3000N of force are considered safe for acute exercise training (Liebenson, 2004).

Recent studies in the rehabilitation and physical therapy literature have emphasized core stability exercises for acute LBP. As balance, strength and flexibility improves, the episodes and intensity of acute LBP diminish (Graves et al. 2004). The development of core strength is an important foundation for long term dynamic muscular strength training, as well as to maximize the propulsive forces developed by the power producing legs. Insufficient core strength can result in a number of errors in technique, such as, excessive movement of the head, rounded shoulders, excessive arm swing and increased lumbar lordosis (Stanton et al. 2004).

2.12 CONCLUSION

According to Bronfort (2004), spinal manipulation is the most studied form of treatment to date for LBP, with manipulation being the most effective form of treatment. The high prevalence of LBP, and the functional disability related to it, has resulted in the large number of conservative treatment methods. Exercise is one of the most popular approaches to the treatment of LBP (Descarreaux et al. 2002). There is, however, little evidence that a particular "type" of exercise is any better than another (Koumantakis, 2005).

This is especially important in view of the fact that mechanoreceptor activity plays the primary role in AMI (Hopkins and Ingersoll, 2000). AMI results from the activity of many different mechanoreceptors within a joint. These mechanoreceptors act on inhibitory interneurons synapsing on the MN pool of the joint musculature, decreasing the force of any contraction stemming from that MN pool (Hopkins and Ingersoll, 2000). This suggests that exercise in patients with pain does not achieve improved clinical outcomes.

Conversely, this is possible with manipulation as the neurophysiological effects of spinal manipulation have been observed in body segments distant from where the manipulations were performed (Suter et al. 1994; Suter et al. 1999; Suter et al. 2000; Naidoo, 2002). Wyke (1985) noted that articular mechanoreceptor afferent nerve fibres give off collateral branches that are distributed both intersegmentally and intra-segmentally. Therefore, manipulation of an individual joint not only affects the motor unit activity in the muscles operating over the joint but also in more remote muscles. The altered afferent input arising from the stimulation of these receptors is thought to cause changes in the motor neuron excitability, with a subsequent decrease in arthrogenic muscle inhibition (William, 1997; Suter et al. 2000).

From the above literature, one can determine that optimal functioning of the three subsystems as described by Panjabi (1992), is necessary in order to achieve spinal stability. Although these subsystems are separate, they are functionally interdependent. A dysfunction, injury or disease in any of these subsystems may lead to spinal instability. One of the key issues in the production, perpetuation and management of mechanical LBP, is the challenge to control the intersegmental relationship for normal pain free function (Richardson et al. 1999).

Restoration of function in the motor system, and specifically the core stabilizers, is becoming an essential part of LBP treatment and prevention and it is thought to contribute to the improvement of performance in athletes (Barr et al. 2005). The development of core strength is an important foundation for long term dynamic muscular strength training and to maximize the propulsive forces developed by the power producing legs. Insufficient core strength can result in a number of errors in technique, such as, excessive movement of the head, rounded shoulders, excessive arm swing and increased lumbar lordosis (Stanton et al. 2004). However, despite its widespread use, research in core strengthening is meagre (Akuthota and Nadler, 2004).

Therefore, this research aims to investigate whether manipulation compared with core strengthening shows statistical and clinical evidence of improvement with regards to patient healing time.

CHAPTER THREE

3. METHODOLOGY AND MATERIALS

3.1 INTRODUCTION

This chapter deals with the main methodological factors that were used in order to substantiate the basis for the data collection process and the statistical methods used to interpret the data.

3.2 STUDY DESIGN

The study design was a randomized controlled parallel group trial. A quantitative study was used, by making use of a pre – and post experimental investigation (Nansel et al. 1993; Naidoo, 2002).

This project received approval from the Institutional Review Board (FRC) of DUT and was compliant with the ethical standards of the Helsinki Declaration of 1975, in the format that it was executed and is presented here.

3.3 METHOD

3.3.1 ADVERTISING

Participants were recruited by placing flyers at the Durban University of Technology, local sport areas such as running clubs, gyms and in the local neighbourhood. Advertisements were also distributed through a distribution network, via the post office, to target the population of the greater Durban area. (Appendix A)

3.3.2 SAMPLE

3.3.2.1 Method:

Thirty participants were divided into two groups (A and B). Group A received treatment in the form of spinal manipulative therapy (SMT) and Group B received treatment in the form of core rehabilitation exercises. Each group of fifteen participants had acute mechanical LBP.

3.3.2.2 Sample size:

Thirty participants were selected as per the inclusion and exclusion criteria. Two groups each group consisting of fifteen participants.

3.3.2.3 Sample Allocation:

The participants' response to the adverts was based on a consecutive convenience sampling method (Mouton, 1996). After participants were assessed and found eligible for the study, they were then allocated to each group using a computer generated randomization table (Esterhuizen, 2006).

3.3.2.4 Sample Characteristics:

Participants were evaluated at an initial consultation. During that consultation, a diagnosis was made based on a case history (Appendix F), physical examination (Appendix G), relevant lumbar spine regional examination (Appendix H) and soape note (Appendix I). In order to establish whether they were eligible for this study, they had to meet the following inclusion and exclusion criteria:

3.3.2.4.1 Inclusion criteria:

1. Participants had to be between the ages of 18 and 45 years. Below the age of 45 was chosen to avoid and reduce the chance of sacroiliac and / or spinal ankylosis (Kirkaldy-Willis and Burton, 1992). Athletes over 19 years of age and below 45 are considered to be at their prime (Hodges, 2002). These are the years after an athlete's body has finished its developmental growth stages and before the aging process starts to slow it down (Hodges, 2002). Participants’ over the age of 18 were used to avoid parent / guardian consent. Motor Unit Potential (MUP) was also a factor in choosing participants (Buchthal, 1957).

2. The participants’ pain rating scale on the NRS had to be greater than 5 and less than 8, (Fejer et al. 2005).

3. All participants had acute LBP - the onset of which was 7 days or less - to avoid the natural history from improving the participants’ condition during the course of the research i.e. most individuals suffering from LBP improve within 6 weeks (Liebenson, 1996).

4. Participants signed an informed consent form to ensure that they undertook the study in full awareness of all that it entailed, and that they were given the opportunity to make any enquiries pertaining to the research. They understood that they were free to withdraw from the research at any time (Appendix J).

5. An athlete is defined by the literature as “an individual who is actively involved in at least one sport or physical activity” (www.wordnet.princeton.edu/perl/webwn, 2007). However, for the purpose of this research, participants had to be involved in running for an average of at least 10km per week and they had to have a Body Mass Index (BMI) between 18.5kg and 24.9kg (Haslett et al. 2002).

The BMI was calculated by dividing the weight of the subject (in kg) by the height (in meters) squared:

$$\text{Weight (kg)/Height}^2\text{ (m)} = \text{BMI}$$

This was in order to improve the sample homogeneity (Mouton, 1996).

6. Participants suffering from mechanical LBP that included both posterior facet syndrome in the lumbar spine (Kirkaldy-Willis and Burton 1992) and / or sacro-iliac syndrome (Cox, 1998) were included in the study.

   Signs and symptoms of posterior facet syndrome include (Kirkaldy-Willis and Burton, 1992; Plaugher, 1993):

   - Referred pain to the hip, buttock, posterior thigh and below the knee, mimicking radicular pain.
   - ill- defined sclerotomal - type pain.
   - Lower back stiffness, especially in the morning or with inactivity.
   - Local paralumbar tenderness.
   - Pain on hyperextension of the lumbar spine.
   - Absence of neurological signs and symptoms.

   For the purpose of this study, research participants had to present with at least three of the above six symptoms.

Signs and symptoms of sacro-iliac syndrome (McCulloch and Transfeldt, 1997) include:

- Pain over the SI joint.
- SI joint locally tender to palpation.
- Referred pain to the buttocks, posterior thigh, groin and occasionally lateral calf and ankle.
- Pain aggravated by provocation tests.
- Clinical evidence of increased movement or asymmetry of the SI joint.
- No other apparent cause of the patient's SI joint pain localization.
- It may mimic a herniated disc or lateral spinal stenosis.
- The lack of nerve root tension signs and absence of motor, reflex or sensory deficits help to distinguish SI syndrome from nerve root entrapment syndromes.

For the purpose of the research, participants presented with at least four out of the eight symptoms mentioned above.

3.3.2.4.2 Exclusion criteria:

1. Participants who presented with signs and symptoms of posterior facet syndrome and/or sacro-iliac syndrome had to exclude (Kirkaldy-Willis and Burton 1992; Plaugher, 1993).

    - Presence of paraesthesias.
    - Presence of neurological deficit.
    - Presence of root tension signs.
    - Presence of hip, buttock, or back pain on straight leg raising.

2. Contra-indications to spinal manipulation (Bergmann and Peterson, 2002; Kirkaldy-Willis and Burton, 1992) which includes but are not limited to:

Relative:

- Osteopenia
- Spondyloarthropathies
- Patient on anticoagulant medication
- Bleeding disorders
- Psychological overlay

Absolute:

- Destructive lesions of the spine, ribs and pelvis
- Healing fracture or dislocation
- Gross instability
- Cauda Equina Syndrome
- Large abdominal aneurysm
- Visceral referred pain
- Marked osteoporosis that was previously diagnosed
- Ankylosing Spondylitis
- The presence of fever, tumours, tuberculosis or any infectious disease
- Local inflammation, thrombosis, metal implants or a hip prosthesis
- Spinal fusion or spinal surgery

3. Contra-indications to abdominal muscle strengthening - glaucoma, hypertension, osteoporosis, spinal tumors, inflammatory diseases and impaired circulation (Harms-Ringhdal, 1993).

4. Participants who experienced extreme discomfort on contraction of the abdominal muscles were excluded. This was in order to eliminate any false negative readings and also to ensure sample homogeneity. This

muscle weakness has been attributed to arthrogenic muscle inhibition (AMI) (Suter et al. 2000).

5. Participants who were receiving manual or medicinal intervention within 48 hours prior to the onset of the study had to comply with a 3-day washout period as proposed by Poul et al. (1993) and Seth, (1999).

6. Participants who had a history of lower back surgery were excluded from the study as the source of their pain may have been related to the surgery. Richardson (1997) suggests the stabilizing function of the core musculature can be reduced when an injury to spinal structures occurs.

7. Participants who required further clinical testing to confirm the diagnosis were excluded, as budget constraints of this research did not allow for further clinical evaluation of the participants.

8. Those participants accepted into the study were asked not to change their lifestyle, daily activities, and regular medication or exercise programs in any way, therefore preventing exclusion from the study.

Those participants included into the study received a letter of information (Appendix B) and an informed consent form (Appendix J) that they signed, in line with the requirements as outlined in the ethics approval and Helsinki Declaration of 1975.

Those participants excluded from this study, were referred to other interns at the Durban University of Technology Chiropractic Day Clinic for treatment of their condition(s).

3.4 CLINICAL PROCEDURE

3.4.1. PARTICIPANT ASSESSMENT

The initial consultation took place in the Chiropractic Day Clinic at the Durban University of Technology campus. This included participant screening and establishment of their suitability for the study.

Orthopeadic tests were used to determine if the participant had a sacroiliac syndrome and/or posterior facet syndrome. Orthopeadic tests used in isolation were not considered as part of the diagnostic criteria for posterior facet syndrome and sacroiliac syndrome. However, it was possible that used as a group or set, the tests may have revealed a greater likelihood of a particular diagnosis being made.

For the purpose of this study, participants had to have a minimum of two out of the four tests listed below being positive in order to diagnose posterior facet syndrome (Kirkaldy-Willis and Burton, 1992).

A) Kemp's test

This involves a combination of lateral flexion and extension over the facet joints while the participant was in the seated position (Giles, 1990). The examiner reached around the shoulders from behind and laterally flexed, rotated and extended the participant to the right, and then the left, whilst applying an axial force (Gatterman, 1995). The test is positive if symptoms are produced (Magee, 2002).

B) Facet joint challenge

The participant was in the prone position. “Springing” the spinous process discerned the status of the facet joints. The examiner placed one thumb on the spinous process above and the other on the spinous process below. A force was applied in a horizontal direction, each towords the centre and in opposite directions to each other. A positive test would be indicated if the participant perceived pain in the area of palpation (Gatterman, 1982).

C) Palpatory tenderness

The participant was in the prone position. The examiner palpated a point in the midline, over the L4-L5 inter-space moving cephalad. The interspaces and spinous processes of the remaining lumbar vertebrae were also palpated. The examiner looked for areas of tenderness, muscle spasm and other signs of pathology. In order to palpate the lumbar facet joints, the examiner needed to move laterally 2-3 cm from the spinous processes (Magee, 2002).

D) Spinous percussion

Spinal percussion may be applied by the pisiform of the examiner’s hand or with a reflex hammer. A gentle percussive force was applied to the spinous processes. A marked, or persistent, painful response to the percussion indicated an underlying fracture or non-mechanical pathology, whereas a mild pain response indicated local irritation and dysfunction (Bergmann and Peterson, 2002).

For the purpose of this study, participants had to have two out of the four tests described below being positive, in order to diagnose sacro-iliac syndrome (Kirkaldy-Willis and Burton, 1992).

A) Gaenslen's test

The participant lay in the supine position with their upper leg (test leg) hyperextended at the hip. The participant held their lower leg flexed against their chest. The examiner stabilized the pelvis while extending the hip of the upper most leg. The other leg was tested similarly. A positive test was indicated by pain in the sacroiliac joint(s) (Magee, 2002).

B) Patrick's Faber test

The participant lay in the supine position. The examiner placed the participant's test leg so that the foot of the test leg was above the knee of the opposite straight leg. The examiner then slowly lowered / abducted the knee of the test leg towards the examination table, whilst stabilizing the opposite hemi-pelvis with the other hand. A positive test was indicated by a decrease in abduction as well as pain in the ipsilateral sacroiliac joint, therefore indicating sacro-iliac dysfunction (Magee, 2002).

C) Erichsen's test / Yeoman's test

The participant lay in the prone position. The examiner applied pressure with one hand to the affected sacro-iliac joint while the other hand lifted the participant's ipsilateral leg, while the participant's knee was flexed to 90 degrees. A positive test was indicated by pain in the sacro-iliac joint (Magee, 2002).

D) Lateral recumbent Sacroiliac compression test

The participant lay in the side lying position. The examiner's hands were placed over the upper part of the iliac crest, applying pressure towards the floor. A positive test was indicated by pain and / or an increased feeling of pressure in the sacroiliac joints (Magee, 2002).

In both Groups A and B, joint dysfunction/s were identified by motion palpation of the lumbar and sacro-iliac joints (Schaefer and Faye, 1990), and in which plane the manipulation would be given, in order to ensure the least amount of discomfort and to restore maximum joint play to the participant's spine (Schaefer and Faye, 1989).

A diagnosis was based on a minimum of two out of the four tests being positive, as well as a dysfunction, to diagnose posterior facet syndrome and / or sacro-iliac syndrome. The participants were then approved and signed for by a clinician at the Chiropractic Day Clinic.

3.4.2 INTERVENTION

Group A - the participants received treatment A (manipulation) on the fixated lumbar segment(s), and / or sacroiliac joint(s), twice a week for two weeks (Mathews, 1997). There was a subsequent follow up consultation six and a half to seven and a half days after their last treatment, when the last sets of readings were taken.

Group B - the participants received treatment B (core rehabilitation) twice a week for two weeks (Chok et al. 1999). There was a subsequent follow-up consultation six and a half to seven and a half days after their last treatment, when the last sets of readings were taken. The rehabilitation portion of the treatment consisted of four core stability exercises (Appendix K) that were taught to the participant at the initial consultation. They were then expected to perform these exercises at home and sign the attached exercise diary (Appendix E), confirming that they had in fact done the given exercises. Rehabilitation exercises included: hyper-extension exercises to strengthen para-vertebral muscles, mobilizing exercises to improve overall spinal mobility and isometric flexion exercises designed to strengthen abdominal lumbar muscles while protecting the back from excessive motion (Weinstein, 1992). Spinal stability training included introductory exercises in order to find the participants functional range, followed by low-load endurance training of stability patterns was emphasized (Liebenson, 2004). For specific lumbar segmental stabilization training, the rehabilitation programme included exercises for the TrA and Multifidus muscles. These exercises included activating an isometric co-contraction of these muscles as well as training the participant to hold a low level tonic contraction (Richardson and Jull, 1995). Training of the core muscles has been recognized as a preventative for first time episodes of LBP (Liebenson, 1997).

3.5 INTERVENTION FREQUENCY

Participants had five visits over a period of three weeks, including measurements (Kirkaldy-Wills and Burton, 1988).

3.6 MEASUREMENT TOOLS

3.6.1 Subjective data was obtained from the following:

1. Numerical Pain Rating Scale - this is an effective and reliable tool to evaluate whether pain is reduced with treatment and to what degree (Bolton and Wilkinson, 1998). Participants were asked to pick a number between 1 and 10, which best described their pain that they were feeling at that time, with 1 being the least pain and 10 the most.

2. Low Back Pain and Disability Questionnaire- this is a sensitive measure of disability in low back pain (Morris, 1983). Participants were asked to go through the questionnaire and mark off which questions were applicable to them. On completion, a score out of 24 was obtained, and then multiplied by 100, in order to obtain a percentage.

3.6.2 Objective feedback was obtained through the use of:

3. Endurance testing of TrA muscle- using the stabilizer PBU (Cairns et al. 2000). This was done utilising the prone test for TrA muscle. (Stabiliser manual Chatanooga Group Inc., 4717 Adams Road, Hixson TN 37343, USA). The PBU consists of an inelastic, three section air-filled bag which was inflated in order to fill the space between the target body area and a firm surface. There is also a pressure dial for monitoring the pressure in the bag for feedback on position (Richardson et al. 1999). The bag was inflated to an appropriate level for the purpose of this research and the pressure recorded. The movement of the body part off the bag resulted in a decrease in pressure. This was recorded as a negative value i.e. -6. The device has come into general use for all parts of the body. However, its use in assessing the abdominal drawing in action has become its most important use in relation to the treatment of problems of the local muscle system in LBP patients (Richardson et al. 1999).

   The endurance testing was done using a stopwatch. Once the participant had established the core contraction, the stopwatch was used to time the length of the contractions that the participant could hold. This was done 3 times and the readings were averaged.

   Both groups were educated on how to contract the TrA muscle by using the four point kneeling position test – (Appendix D) (Evans and Oldrieve, 2000). The patient was positioned with their hands directly under their shoulders and their knees under their hips. The examiner's hand was placed on the participant's lower abdomen and the following was asked of the participant, "As you breathe out, gently pull your navel up towards your spine and maintain this position whilst you breathe normally." This position was used as the forward drift of the abdominal contents provides a facilitatory stretch of the deep abdominal muscles and, at the same time, provides an inhibitory stretch of the superficial muscle, the Rectus Abdominus (Richardson and Jull, 1995).

The abdominal draw in test with the PBU was used to measure the participant's TrA strength and endurance in both groups. In both groups, the TrA endurance was tested in the prone position (appendix D). The stabilizer was centrally placed over the abdomen with the navel in the centre of the pressure cell and the lower edge of the pressure cell in line with the left and right anterior superior iliac spines. The stabilizer was then inflated to 70mmHg and allowed to stabilize. The participant was then instructed to gently draw in their lower abdomen off the pressure cell, without moving their spine or pelvis. A drop of 6 -10 mmHg (minimum) was obtained when the correct localized contraction was performed; this was recorded as a negative value i.e. -6. A variation of up to 2mmHg was allowed for the normal breathing pattern, but if this was exceeded i.e. the pressure increased above 66mmHg, the participant was assumed to have reached their endurance limit (Richardson et al. 1999). The endurance was measured by timing how long they could hold their abdominal contraction (Richardson et al. 1999). In order to rule out the use of the participants global muscles in obtaining the drop in the stabilizer pressure, the EMG was used. This ensured that the participants' core muscles were being activated rather than having their global muscles compensate for their core muscles (Silfies et al. 2005).

4. An algometer (the force dial algometer to assess the tenderness of the affected joints) was used to quantify response to treatment such as manipulation and provided a means of measuring participants' improvement (Fischer, 1986).

   The algometer readings were taken over the most painful area of the symptomatic sacroiliac or lumbar facet joint. The participant was requested to indicate the point of pain or discomfort by saying 'now', and the reading was taken at that point.

5. The surface EMG was used to determine the involvement of the global muscles during the core contraction. This was to ensure that the participant was learning to activate the core muscles and not compensate by recruiting the global muscles (Stevens et al. 2006).

   The surface EMG was utilized whilst the prone endurance test was being performed. For the purpose of this study, the electrodes were placed over the Quadratus Lumborum muscles, bilaterally on either side of the spine at the levels of L1 and L4, approximately 2cm from the spinous process.

3.7 MEASUREMENT FREQUENCY

All readings and testing were done prior to the interventions at consultations 1, 3 and 5 to assess for any changes.

3.8 SUMMARY OF MEASUREMENT / TREATMENT FREQUENCY

Week	**Visit**	**Group A**	**Group B**
1	1	Case history, physical, lumbar regional, clinical evaluation, readings and treatment A	Case history, physical, lumbar regional, clinical evaluation, readings and treatment B
	2	Treatment A	Treatment B
2	3	Reading 2 and treatment A	Reading 2 and treatment B
	4	Treatment A	Treatment B
3	5	Reading 3	Reading 3

Readings = Pressure biofeedback unit, Algometer and Surface EMG.

Treatment A = Core rehabilitation

Treatment B = Manipulation

3.9 STATISTICAL METHODS

Data were analysed using SPSS version 13 (SPSS Inc., Chicago, Illinois, USA) and Stata version 9.0 for Windows (StataCorp. LP, Texas, USA). A p value of <0.05 was considered statistically significant.

Baseline outcome measures and demographics were compared between treatment groups to ensure that no baseline differences existed between the groups. Quantitative outcome measures over time were compared between the two groups using repeated measures ANOVA. For EMG measurements, where a before and after reading was taken at each time point, the difference between the before and after measurement was computed and used as the outcome measure at each time point. A significant time by group interaction effect indicated a significant treatment effect. Profile plots were used to assess the trends visually. Binary outcomes over time were analysed using binary generalized linear models in Stata.

Changes in all outcome measurements over the three time points were computed and intra-group Pearson's correlation coefficients were used to assess relationships between changes in subjective and objective outcomes.

CHAPTER 4

4. RESULTS AND DISCUSSION

4.1 INTRODUCTION

The statistical findings and results obtained from the data will be presented and discussed in this chapter. The first part of this chapter contains the demographic data of all the participants and the second part contains the statistical analysis of the subjective and objective data. The participants in group A received spinal manipulation, and the participants in group B received core stabilization exercises.

The following abbreviations were used in this chapter:

PBU	- Pressure Biofeedback Unit
RMQ	- Roland Morris Questionnaire
NRS	- Numerical Pain Rating Scale
SI	- Sacro-iliac
TrA	- Transversus Abdominus

The primary data consisted of:

1. Demographic data that included – age, gender, occupation, height, weight and BMI.
2. Objective and subjective data that included – RMQ, NRS, algometer, stabilizer PBU and EMG.

The secondary data consisted of information gathered from literature found in journal articles, books and the Internet.

The following units were used in the tables:

Age	- years
Height	- metres
Weight	- kilograms
Strength and endurance	- mm/Hg and seconds
Algometer	- Kg/cm2

4.2. DEMOGRAPHICS

4.2.1 AGE, OCCUPATION, GENDER, HEIGHT, WEIGHT and BODY MASS INDEX

Thirty participants were randomized into two equal treatment groups. Their mean age was 28.5 years, with a standard deviation of 7.4 years and a range from 18 to 44 years.

4.2.1.1 Occupation

The sample had a diverse occupational profile (Table 1). The majority of participants were artisans (30%) consisting mainly of factory workers, such as fitters and welders, followed by students (26.7%). Professionals (Chiropractors and other health professionals) constituted 13.3%.

Table 1: Occupational classification of sample

	Frequency	Percent
Artisan	9	30.0
Student	8	26.7
Professional	4	13.3
Technical	3	10.0
Operator	2	6.7
Administration	1	3.3
Sales	1	3.3
Service	1	3.3
Self employed	1	3.3
Total	30	100.0

4.2.1.2 Gender

There was no significant difference between the two groups in terms of gender (p=0.215), although the core group contained a higher proportion of females than the manipulation group. The gender breakdown of each group is shown in Table 2.

Table 2: Gender by treatment group

			Gender		Total
			Male	Female	
Group	Manipulation	Count	13	2	15
		Row %	86.7%	13.3%	100%
	Core rehabilitation	Count	9	6	15
		Row %	60.0%	40.0%	100%
Total		Count	22	8	30
		Row %	73.3%	26.7%	100%

Fisher's exact p=0.215

4.2.1.3 Age

There was no significant difference in age between the two treatment groups (p=0.682), although the mean age in the core group was slightly older than the manipulation group. This is shown in Table 3.

Table 3: Comparison of mean age between the treatment groups

	Group	N	Mean	Std. Deviation	Std. Error of Mean	p value
age	Manipulation	15	27.93	6.902	1.782	0.682
	Core rehabilitation	15	29.07	8.058	2.080	

4.2.1.4 Height, weight and BMI

Table 4 shows that the two groups were similar in terms of height, weight and BMI. There were no significant differences between the groups, and the means of the two groups were very similar.

Table 4: Comparison of mean height, weight and BMI between the treatment groups

	Group	N	Mean	Std. Deviation	Std. Error of Mean	p value
Height(M)	Manipulation	15	1.7653	0.09516	0.02457	0.940
	Core rehabilitation	15	1.7627	0.09647	0.02491	
Weight	Manipulation	15	72.7333	11.37960	2.93820	0.783
	Core rehabilitation	15	71.6000	10.96618	2.83146	
BMI	Manipulation	15	23.1814	1.66298	0.42938	0.738
	Core rehabilitation	15	22.9489	2.08547	0.53847	

Remarks:

From the above information, it is evident that the groups are homogeneous in terms of age, gender, height, weight and BMI so more meaningful comparisons between these two groups can be made (Mouton, 1996). If there were any changes observed, then these changes would be due to the intervention and not the demographics.

4.3 COMPARISON OF BASELINE OUTCOMES BETWEEN THE TREATMENT GROUPS

None of the outcome measurements showed statistically significant differences between treatment groups prior to the intervention. Table 5 shows that there were no differences between the group means at baseline.

Table 5: Comparison of baseline outcome measurements between the treatment groups

	Group	N	Mean	Std. Deviation	Std. Error of Mean	p value
RMQ	Manipulation	15	3.13	1.457	.376	1.000
	Core rehabilitation	15	3.13	2.100	.542	
NRS	Manipulation	15	6.27	1.033	.267	0.386
	Core rehabilitation	15	6.57	.821	.212	
ALGOMETER	Manipulation	15	7.2533	1.29276	.33379	0.122
	Core rehabilitation	15	6.3900	1.65045	.42615	
STABILIZER PRESSURE	Manipulation	15	-5.46	1.873	.484	0.195
	Core rehabilitation	15	-6.40	1.993	.515	
TIME ON STABILIZER	Manipulation	15	34.1800	17.05692	4.40408	0.271
	Core rehabilitation	15	41.2220	17.28682	4.46344	
EMG L1	Manipulation	15	3.5087	.93005	.24014	0.550
	Core rehabilitation	15	3.3107	.86227	.22264	
EMG L4	Manipulation	15	6.791	1.3888	.3586	0.596
	Core rehabilitation	15	6.489	1.6784	.4334	
EMG ABDOMINAL	Manipulation	15	3.007	.0594	.0153	0.115
	Core rehabilitation	15	3.047	.0743	.0192	
STABILIZER ABDOMINAL	Manipulation	15	-5.47	2.066	.533	0.350
	Core rehabilitation	15	-6.13	1.767	.456	

Remarks

As described above (Mouton, 1996), the changes were likely due to the intervention. Therefore, the demographics would not be the cause of the changes between the two groups, as the participants all started at the same baseline from which the objective and subjective clinical findings were obtained.

4.4 ASSESSMENT OF THE TREATMENT EFFECT

4.4.1 SUBJECTIVE OUTCOMES

4.4.1.1 RMQ

There was no significant treatment effect according to the RMQ outcome (p=0.388). Thus, both groups showed similar progress over time. The overall time effect was highly significant (p<0.001) and Figure 1 shows that both groups experienced a sharp decrease in RMQ score over time. The rate of decrease was similar, thus for RMQ there was no evidence that the treatments showed differential effects. However, the manipulation group showed a slightly more linear decrease between the second and third time point.

Table 6: Within and between participants effects for RMQ

Effect	Statistic	P value
Time	Wilk's Lambda=0.363	<0.001
Group	F=0.03	0.959
Time*group	Wilk's Lambda=0.932	0.388

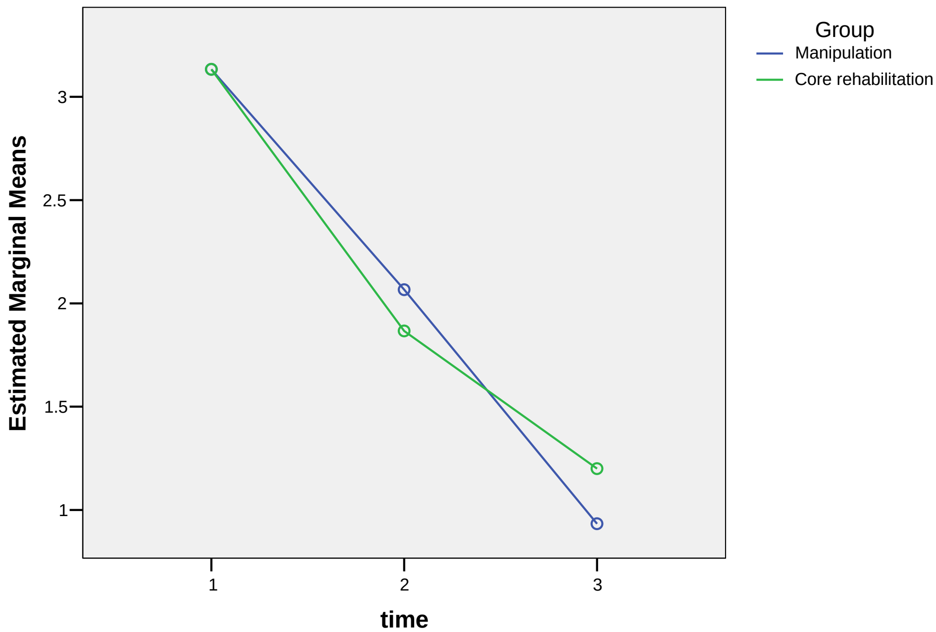


Figure 1: Profile plot of time by group for RMQ

Remarks

No difference existed between the two groups at the initial consultation. This indicates that the two groups were similarly matched regarding the severity of their LBP at the onset of the study. However, at the 2nd and 3rd reading, the manipulation group showed an elevated rate of decrease over time. These results show a reduction in the level of pain experienced by both groups over the treatment period.

Possible mechanisms for the above results include the following: In group A, the manipulation decreases pain (Van Tulder et al. 2005) and the restricted motion (Bergmann and Peterson, 2002). This allowed all the muscles to function optimally (Suter et al. 1994; Suter et al. 1999; Suter et al. 2000; Naidoo, 2002). In group B, core rehabilitation only concentrated on the participant's core (Jull and Richardson, 2000), and was not related to the clinical symptoms. This was because the purpose of rehabilitation was not to address the dysfunction. Thus, in contrast, manipulation would have had a constant reduction / improvement as opposed to the rehabilitation group, where the dysfunction was still present, limiting the functional ability. However, the above discussion is a hypothesis and requires further investigations.

4.4.1.2 NRS

Similarly, for NRS there was a significant time effect overall for NRS (p<0.001), but no evidence of a differential treatment effect (p=0.248). Figure 2 shows that the rate of improvement in pain according to NRS score was very similar in both groups (profiles of the two groups are almost parallel). However, there was a significant group effect for NRS, meaning that there were significant differences between the two groups at all time points.

Table 7: Within and between participants effects for NRS

Effect	Statistic	p value
Time	Wilk's Lambda=0.048	<0.001
Group	F=5.45	0.027
Time*group	Wilk's Lambda=0.902	0.248

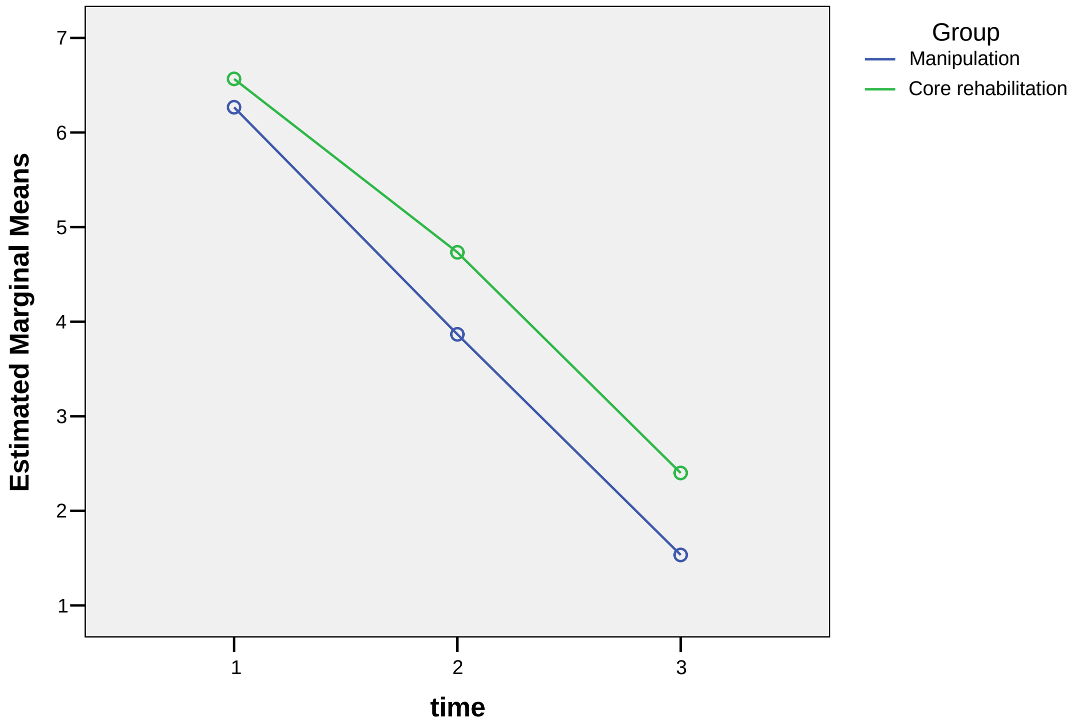


Figure 2: Profile plot of time by group for NRS

Remarks

No difference existed between the two groups at the initial consultation. This indicates that the two groups were similarly matched regarding the severity of their LBP at the onset of the study.

Both groups A and B experienced LBP / dysfunction. Group A addressed the dysfunction directly in the form of manipulation that could have resulted in the restoration of mechanical mobility (Gatterman, 1990; Bergmann and Peterson, 2002). This increased movement within the restricted joint(s) would have allowed for normal movement and stimulation of the mechanoreceptors within the now mobile joint, further decreasing the pain (Melzack and Wall, 1965).

The mechanism discussed above occured to a lesser extent in Group B, which only relied on mechanical stimulation via the muscles (Melzack and Wall, 1965). Based on this mechanical stimulation, change in both groups was therefore parallel, although at a lesser rate in Group B. This is in agreement with Chok et al. (1999); Richardson et al. (1999); Rainville et al. (2004); Liebenson, (2004) however, it is in contrast to the theories around AMI as presented in Hopkins and Ingersoll (2000).

Nevertheless, based on the results obtained, we can conclude that manipulation and core rehabilitation provided a reduction in the level of pain experienced by the participants in both groups, even though no difference was seen between the groups over the treatment period.

4.4.2 OBJECTIVE OUTCOMES

4.4.2.1 Algometer

Both groups improved significantly over time ($p<0.001$) for this outcome, but there was no difference in the rate of improvement between the groups ($p=0.825$). Thus, there was no evidence of a differential treatment effect. There was a borderline significant difference overall between the groups ($p=0.049$), but the profiles of the two groups were parallel over time (Figure 3).

Table 8: Within and between participants effects for Algometer

Effect	Statistic	p value
Time	Wilk’s Lambda=0.362	<0.001
Group	F=4.23	0.049
Time*group	Wilk’s Lambda=0.986	0.825

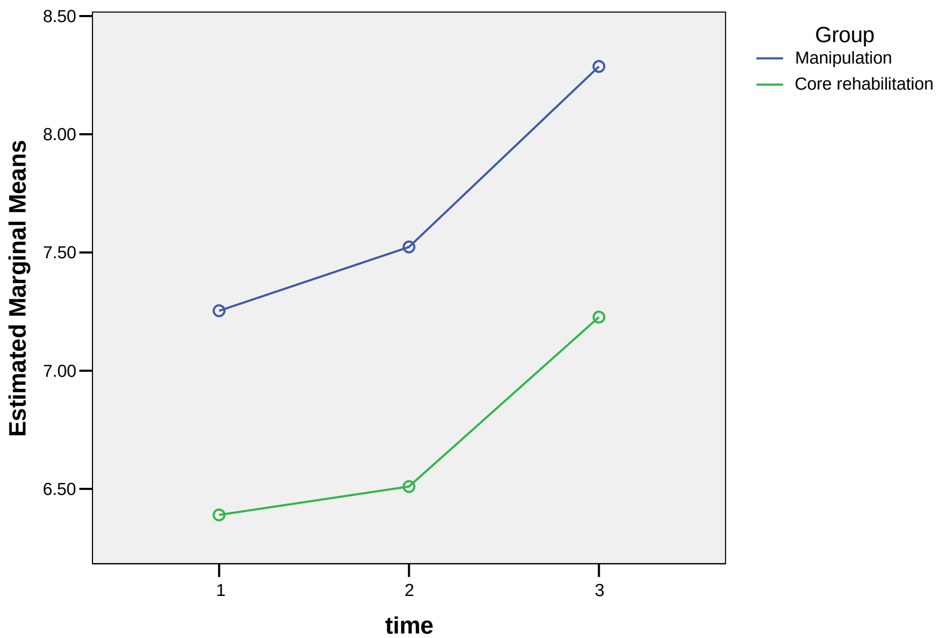


Figure 3: Profile plot of time by group for Algometer

Remarks

These results show a reduction in the level of tenderness experienced by both groups over the treatment period (Fischer, 1986). This is expected in Group A as based on the work of Korr (1978) (as cited in Leach, 1994) and supported by Melzack and Wall (1965), Gatterman (1990) and Bergmann and Peterson (2002). In Group B, tenderness was noted to decrease, which concurs with the research conducted by Richardson et al. (1999).

4.4.2.2 Stabilizer pressure

The abdominal draw-in test with the PBU was used to measure the participant's TrA strength and endurance in both groups. In groups A and B, the TrA strength and endurance was tested in the prone position (appendix D). The stabilizer was centrally placed over the abdomen with the navel in the centre of the pressure cell and the lower edge of the pressure cell in line with the left and right anterior superior iliac spines. The stabilizer was then inflated to 70mmHg and allowed to stabilize. The participant was then instructed to gently draw in their lower abdomen off the pressure cell, without moving their spine or pelvis. A drop in 6 - 10 mmHg (minimum) was obtained when the correct localized contraction was performed; this was recorded as a negative value (i.e. -6). A variation of up to 2mmHg was allowed for the normal breathing pattern, but if this was exceeded (i.e. the pressure increased above 66mmHg), the participant was assumed to have reached their endurance limit (Richardson et al. 1999)). The endurance was measured by timing the length in seconds that they could hold their abdominal contraction (Richardson et al. 1999), using a stopwatch.

For stabilizer pressure, there was a significant decrease over time for both groups ($p<0.001$), but no evidence of a differential treatment effect ($p=0.199$). Figure 4 shows that the rate of decrease was very similar over time for both groups, although there was a significant difference in stabilizer pressure between the groups regardless of time ($p=0.042$).

Table 9: Within and between participants' effects for stabilizer pressure

Effect	Statistic	p value
Time	Wilk's Lambda=0.235	<0.001
Group	F=4.539	0.042
Time*group	Wilk's Lambda=0.887	0.199

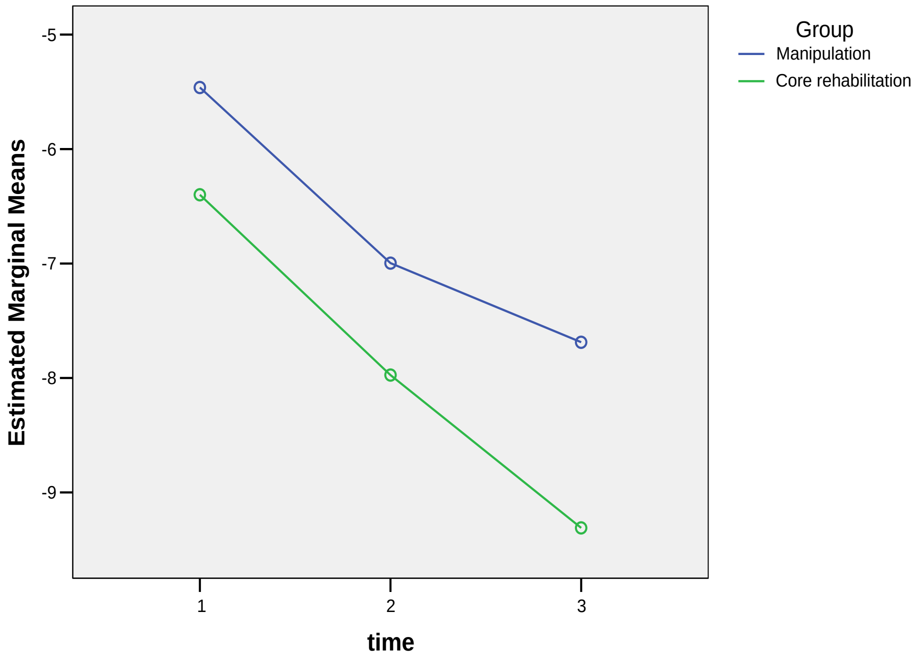


Figure 4: Profile plot of time by group for stabilizer pressure

Remarks

Both groups showed changes between readings 1, 2 and 3.

In group A, between readings 1 and 2 there was a sudden decrease in pressure - this could be due to the fact that the AMI process had been taken away as a result of the manipulation. Between 2 and 3, the improvement was not as rapid. This could be because the participants were dependant on the function of their core muscles. These had previously been compromised through the AMI process and therefore possibly atrophied with a resultant decrease in functional ability (Hides et al. 1994). Thus, without the core exercises being available to these participants, their ability to improve was hampered.

In group B, the participants were constantly improving between readings. This could be due to them constantly doing the exercises, therefore increasing their muscle ability and muscle memory (Chok et al. 1999; Richardson et al. 1999;

Rainville et al. 2004; Liebenson 2004). This is also attributed to the improvement related to an increase in mechanoreceptive stimulation (Melzack and Wall, 1965).

4.4.2.3 Time on stabilizer / endurance

There was a statistically significant treatment effect for this outcome (p=0.006). In the presence of a significant interaction effect (treatment effect), the main impacts of time and group cannot be interpreted, since the effect of time is dependant on which group the subject was in. Figure 5 shows that the core rehabilitation group was able to increase their time on stabilizer at a faster rate than the manipulation group.

Table 10: Within and between participants' effects for time on stabilizer

Effect	Statistic	p value
Time	Wilk's Lambda=0.221	<0.001
Group	F=5.09	0.032
Time*group	Wilk's Lambda=0.685	0.006

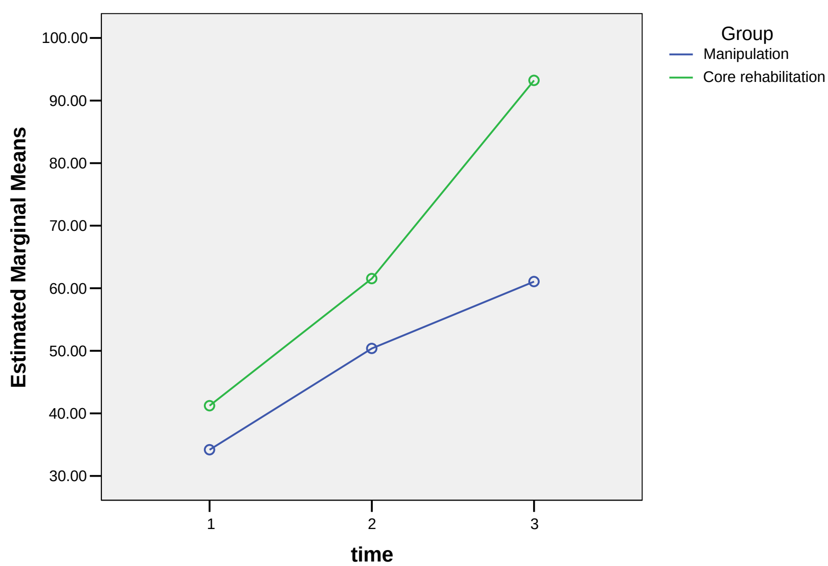


Figure 5: Profile plot of time by group for time on stabilizer

Remarks

In group A, between readings 1 and 2 there was an increase in pressure. This could be due to the fact that the AMI process had been taken away as a result of the manipulation. Between 2 and 3, the improvement was not as rapid and this could be because the participants were dependant on the function of their core muscles which had previously been compromised through the AMI process and therefore possibly atrophied with a resultant decrease in functional ability (Hides et al. 1994). Thus, without the core exercises being available to these participants, their ability to improve was hampered.

In group B, the participants were constantly improving between readings. This could be due to constantly performing the exercises, thereby increasing their muscle ability and muscle memory (Chok et al. 1999; Richardson et al. 1999; Rainville et al. 2004; Liebenson, 2004). This is also attributed to the improvement related to an increase in mechano-receptive stimulation (Melzack and Wall, 1965).

4.4.2.4 EMG at the level of L1

There was no statistical evidence of a treatment effect for this outcome (p=0.127), although Figure 6 shows that the two groups were essentially behaving differently over time, especially between time 2 and 3. The core rehabilitation group showed a decrease over this time, while the manipulation group started to increase over this time.

Table 11: Within and between participants' effects for EMG at the level of L1

Effect	Statistic	p value
Time	Wilk's Lambda=0.858	0.127
Group	F=1.27	0.270
Time*group	Wilk's Lambda=0.858	0.127

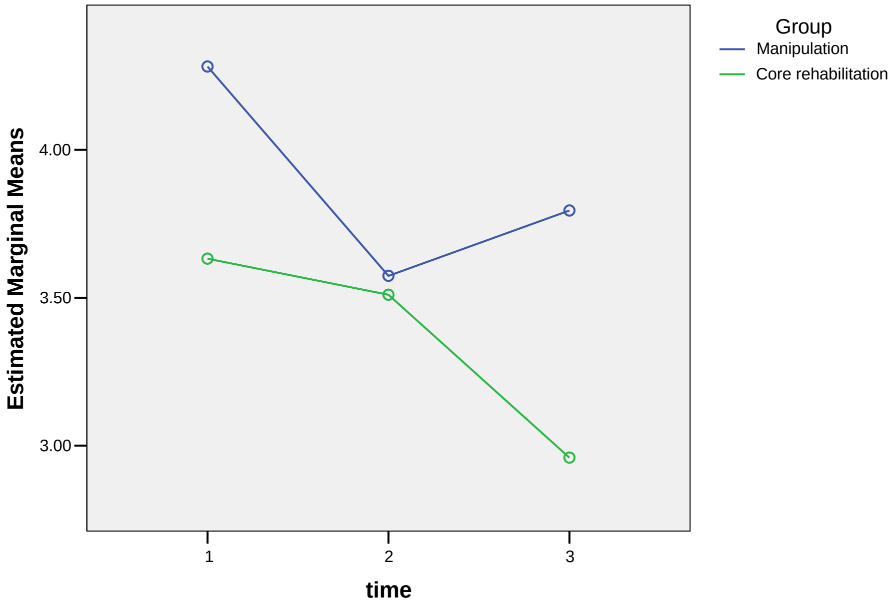


Figure 6: Profile plot of time by group for EMG at the level of L1

Remarks

In group A, the manipulation had taken away the muscle activity due to the muscle spasm (Korr, 1976), which resulted in a decrease between readings 1 and 2. At reading 2 to 3, the participants relied perhaps on their global muscles due to the fact that they have weaker core muscle (Group A) – see discussion under 4.4.2.2. With the inability of the core muscles to function appropriately, there was also a majority weight transfer through the joints (facet joints / sacro-iliac joints) thereby irritating the joint, that in turn could lead to inflammation. This could have resulted in a new muscle spasm (Dvorak, 1985; Gatterman, 1990; Mense, 1991) and hence, resulted in an increase in the EMG findings at L1.

In group B, systematically strengthening the core muscles resulted in the participants' reliance on their global muscles becoming less with each reading consistent with Richardson et al. (1999). However, the improvements noted in both groups may have been due to the natural history of acute LBP and cannot be excluded.

4.4.2.5 EMG at the level of L4

For EMG readings at L4, there was no evidence of a differential treatment effect between the groups (p=0.838). Figure 7 shows that the rate of change over time was similar between the groups. Also, although both groups showed a decrease in this measurement over time, the rate of decrease was not statistically significant for both groups combined (p=0.132).

Table 12: Within and between participants' effects for EMG at the level of L4

Effect	Statistic	p value
Time	Wilk's Lambda=0.861	0.132
Group	F=0.063	0.803
Time*group	Wilk's Lambda=987	0.838

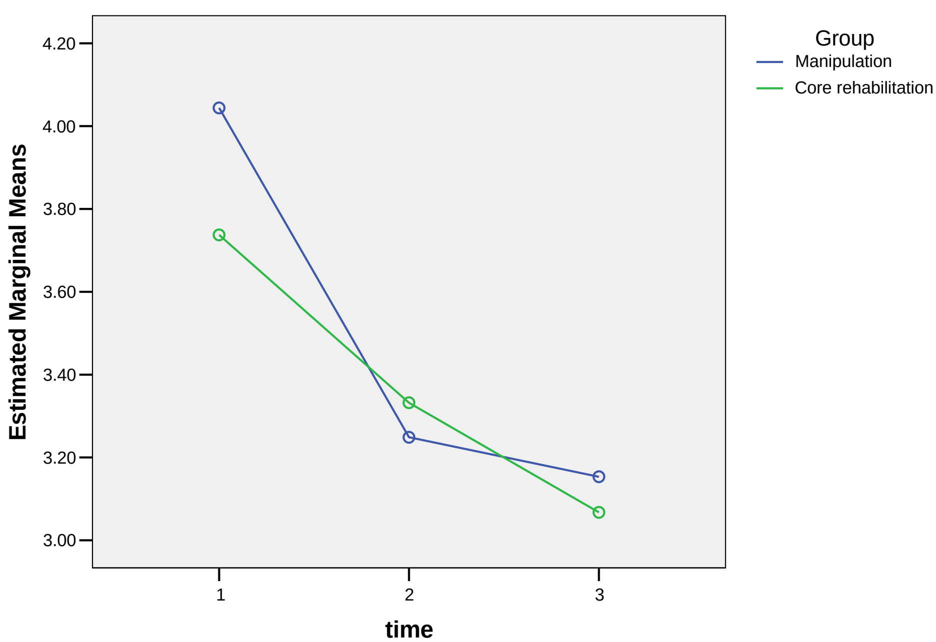


Figure 7: Profile plot of time by group for EMG at the level of L4

Remarks

In Group A, L1 has a greater propensity for movement, whereas L4 / L5 has a greater chance for joint dysfunction as it carries a greater weight (Moore and Dalley, 1999) and is bound by additional ligamentous structures (e.g. iliolumbar ligament) (Moore and Dalley, 1999). With the predisposition to dysfunction, L4 / L5 are associated with a greater degree of pain. Therefore, the result of pain reduction is greater at the L4 – L5 level as opposed to L1 level. This could thus account for the leveling off of the readings between 2 and 3.

In Group B, systematically strengthening the core muscles resulted in the participants' reliance on their global muscles becoming less with each reading - refer to discussion 4.4.2.4.

4.4.2.6 EMG Abdominal

The two groups behaved very similarly over time for this outcome. There was no evidence of a time effect (p=0.177) or a differential treatment effect (p= 0.840). This is demonstrated by the parallel profiles in Figure 8.

<u>Table 13: Within and between participants effects for EMG Abdominal</u>

Effect	Statistic	p value
Time	Wilk's Lambda=0.879	0.177
Group	F=2.056	0.163
Time*group	Wilk's Lambda=0.987	0.840

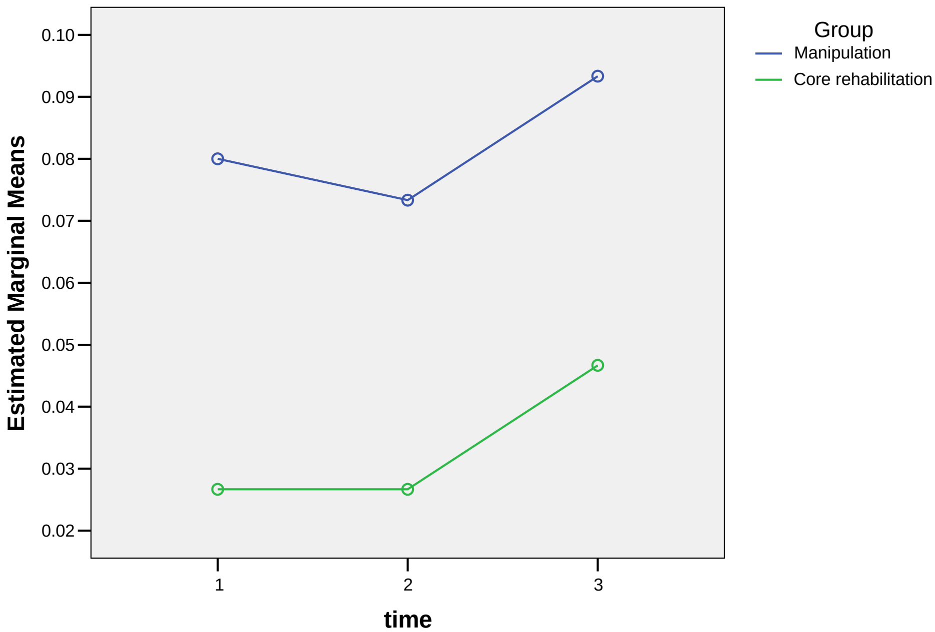


Figure 8: Profile plot of time by group for EMG abdominal

Remarks

Both groups showed similar trends here.

With increased muscle activity in the low back due to the participants' pain (Suter et al. 2000), it is possible that the abdominal muscles were also showing an increase in activity at the start of the study (Stevens et al. 2006) (i.e. above the normal level of activity).

With the application of manipulation (Gatterman, 1990; Bergmann and Peterson, 2002), there is a sharper decrease in the muscle activity as the pain is reduced and AMI is removed (Group A) - as mentioned above in 4.4.2.2., which is not evident in Group B.

However, the increase in the readings from point 2 to 3 can be due to the fact that in Group A and B, the local stability of the Multifidus is more active, either due to having removed the dysfunction via manipulation, or due to the strengthening of the core muscles via rehabilitation (Richardson et al. 1999). In both instances, the activity of global muscles cannot be excluded as the measurement tool was one of surface readings and not that of a needle EMG. Therefore, a needle EMG should be used for future studies.

It is, however, noted that these statements are hypothetical due to the miniscule differences seen in the readings.

4.4.2.7 Stabilizer abdominal

Although there was a significant decrease over time for this outcome ($p<0.001$), the decrease was shown equally in both treatment groups, leading to a conclusion of no differential treatment effect ($p=0.266$). This is shown in Table 14 and by the parallel profiles of the two groups in Figure 9.

Table 14: Within and between participants' effects for Stabilizer abdominal

Effect	Statistic	p value
Time	Wilk's Lambda=0.218	<0.001
Group	F= 3.43	0.075
Time*group	Wilk's Lambda=0.907	0.266

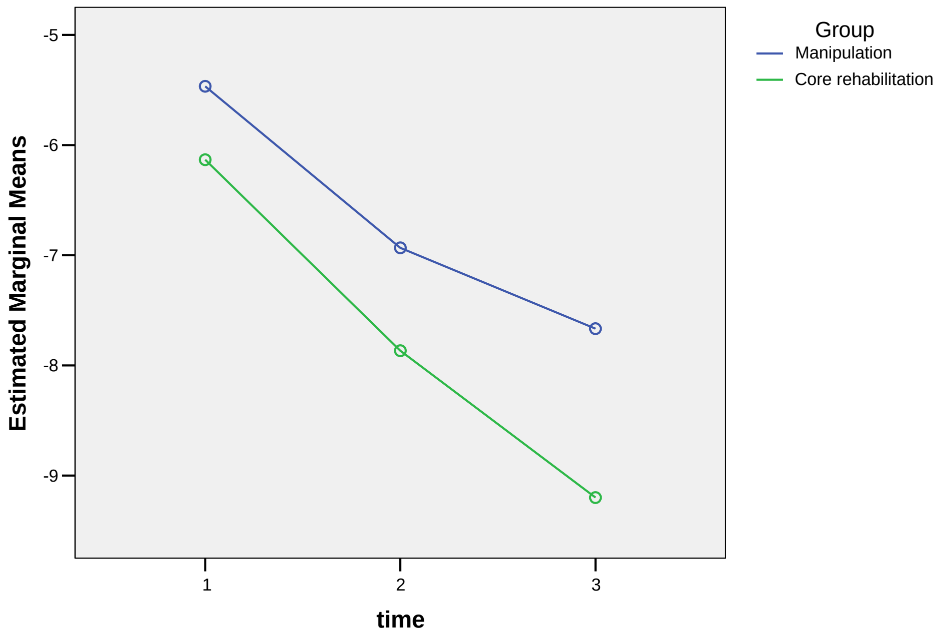


Figure 9: Profile plot of time by group for Stabilizer abdominal

Remarks

Both groups showed changes between readings 1, 2 and 3 as mentioned above in 4.4.2.2.

4.4.3 BINARY OUTCOMES

4.4.3.1 Presence of fixations on the left side

For this outcome, Table 15 shows that there were no significant effects. There was no differential treatment effect (p=0.294), thus the treatment did not affect the presence of fixation on the left side.

Table 15: Binary general linear regression model analysis for presence of fixations on the left side

Effect	Statistic	p value
Time	Risk ratio= 0.930	0.278
Group	Risk ratio= 1.04	0.663
Time*group	Risk ratio= 1.04	0.294

4.4.3.2 Presence of fixation on the right side

An analysis of fixations on the right side could not be done, since at baseline (time 1) there were no subjects with fixation on the right side in the manipulation group. This meant there was no baseline to compare the changes over time. Thus, the regression models failed continuously and no conclusion can be reached for this outcome.

4.4.3.3 Presence of fixations at L1

For this outcome, Table 16 shows that there were no significant effects. There was no differential treatment effect (p=0.739), thus the treatment did not affect the presence of fixation at L1.

Table 16: Binary general linear regression model analysis for presence of fixations at L1

Effect	Statistic	p value
Time	Risk ratio= 1.287	0.865
Group	Risk ratio= 11.99	0.297
Time*group	Risk ratio= 0.777	0.739

4.4.3.4 Presence of fixations at L2

For this outcome, Table 17 shows that there were no significant effects. There was no differential treatment effect (p=0.715), thus the treatment did not affect the presence of fixation at L2.

Table 17: Binary general linear regression model analysis for presence of fixations at L2

Effect	statistic	P value
Time	Risk Ratio = 0.850	0.826
Group	Risk Ratio = 0.792	0.865
Time*group	Risk Ratio = 0.809	0.715

4.4.3.5 Presence of fixations at L3

For this outcome, Table 18 shows that there were no significant effects. There was no differential treatment effect (p=0.179), thus the treatment did not affect the presence of fixation at L3.

Table 18: Binary general linear regression model analysis for presence of fixations at L3

Effect	Statistic	p value
Time	Risk Ratio = 1.180	0.331
Group	Risk Ratio = 1.740	0.149
Time*group	Risk Ratio = 0.863	0.179

4.4.3.6 Presence of fixations at L4

For this outcome, Table 19 shows that the time effect was significant (p=0.036), thus both groups experienced a decrease in fixations at L4 over time. However, there was no differential treatment effect (p=0.139) and hence the treatment did not affect the presence of fixation at L4.

Table 19: Binary general linear regression model analysis for presence of fixations at L4

Effect	Statistic	p value
Time	Risk Ratio = 0.720	0.036
Group	Risk Ratio = 0.800	0.524
Time*group	Risk Ratio = 1.171	0.139

4.4.3.7 Presence of fixations at L5

For this outcome, Table 20 shows that there were no significant effects. There was no differential treatment effect (p=0.583), thus the treatment did not affect the presence of fixation at L5.

Table 20: Binary general linear regression model analysis for presence of fixations at L5

Effect	Statistic	p value
Time	Risk Ratio = 1.012	0.937
Group	Risk Ratio = 1.383	0.302
Time*group	Risk Ratio = 0.946	0.583

4.4.3.8 Presence of fixations at SI

For this outcome, Table 21 shows that there were no significant effects. There was no differential treatment effect (p=0.128), thus the treatment did not affect the presence of fixation at S1.

Table 21: Binary general linear regression model analysis for presence of fixations at SI

Effect	Statistic	p value
Time	Risk Ratio = 0.807	0.128
Group	Risk Ratio = 1.008	0.978
Time*group	Risk Ratio = 1.113	0.128

Table 22: Comparison of p value at different levels

Level	p value
SI	p=0.128
L5	p=0.583
L1	p=0.739

Remarks

The bigger the joint, the more stable the joint and, therefore, the improvement over time is enhanced (Sakamoto et al. 2001). The amount of innervation of the sacro-iliac joint is greater than L1 (Moore and Dalley, 1999), thus the stimulation of the gate control is greater at the SI joint as opposed to L1, which supports Melzack and Wall (1965), as mentioned above in 4.4.2.2.

4.5 CORRELATIONS BETWEEN CHANGES IN OUTCOMES OVER TIME

4.5.1 MANIPULATION GROUP

Change in time on the stabilizer and change in RMQ were significantly negatively correlated ($r=-0.642$, $p=0.010$). This meant that as time on the stabilizer increased, so the RMQ score decreased. The stabilizer pressure and the abdominal stabilizer pressure were positively correlated. As the one value increased, so did the other ($r=0.760$, $p=0.001$). EMG for L1 and L4 were positively correlated ($r=0.641$, $p=0.010$), as were changes in EMG at L4 and EMG abdominal ($r=0.528$, $p=0.043$).

RMQ versus the Stabilizer time:

- The increase in functionality of the participant is shown by a decrease in the RMQ - it is directly and significantly negatively correlated to the time on the stabilizer (increase). This supports 4.4.2.3.

Stabilizer pressure versus abdominal stabilizer pressure:

- Showed a positively significant relationship. This supports the discussion under 4.4.2.2 and 4.4.2.7.

EMG for L1 and L4:

- Showed a positively significant relationship which supports that L5 is more stable. This supports the discussion under 4.4.2.4 and 4.4.2.5.

EMG at L4 and EMG abdominal:

- Showed a positively significant relationship. Participants were using their global muscles more than their local muscles. This may also indicate why the change in the RMQ and the stabilizer time was so significant. This supports the discussion under 4.4.2.5 and 4.4.2.6

Table 23: Pearson's correlation between changes in outcomes for the manipulation group (n=15)

		Change in RMQ	Change in NRS	Change in algometer	Change in stabilizer pressure	Change in time on stabilizer	Change in EMG L1	Change in EMG L4	Change in EMG abdominal	Change in Stabilizer abdominal
Change in RMQ	Pearson Correlation	1	.326	-.087	.236	-.642(**)	-.324	.055	.451	.304
	Sig. (2-tailed)		.235	.758	.397	.010	.240	.847	.092	.270
	N	15	15	15	15	15	15	15	15	15
Change in NRS	Pearson Correlation	.326	1	-.231	.364	-.280	-.118	-.152	-.063	.325
	Sig. (2-tailed)	.235		.408	.182	.311	.675	.590	.822	.237
	N	15	15	15	15	15	15	15	15	15
Change in algometer	Pearson Correlation	-.087	-.231	1	.227	.500	-.219	-.378	-.504	-.208
	Sig. (2-tailed)	.758	.408		.416	.058	.433	.164	.055	.458
	N	15	15	15	15	15	15	15	15	15
Change in stabilizer pressure	Pearson Correlation	.236	.364	.227	1	-.321	.024	-.007	.015	.760(**)
	Sig. (2-tailed)	.397	.182	.416		.243	.933	.979	.958	.001
	N	15	15	15	15	15	15	15	15	15
Change in time on stabilizer	Pearson Correlation	-.642(**)	-.280	.500	-.321	1	-.191	-.237	-.476	-.395
	Sig. (2-tailed)	.010	.311	.058	.243		.494	.396	.073	.145
	N	15	15	15	15	15	15	15	15	15
Change in EMG L1	Pearson Correlation	-.324	-.118	-.219	.024	-.191	1	.641(**)	.068	-.051
	Sig. (2-tailed)	.240	.675	.433	.933	.494		.010	.809	.857
	N	15	15	15	15	15	15	15	15	15
Change in EMG L4	Pearson Correlation	.055	-.152	-.378	-.007	-.237	.641(**)	1	.528(*)	.035
	Sig. (2-tailed)	.847	.590	.164	.979	.396	.010		.043	.900
	N	15	15	15	15	15	15	15	15	15
Change in EMG abdominal	Pearson Correlation	.451	-.063	-.504	.015	-.476	.068	.528(*)	1	.319
	Sig. (2-tailed)	.092	.822	.055	.958	.073	.809	.043		.246
	N	15	15	15	15	15	15	15	15	15
Change in Stabilizer abdominal	Pearson Correlation	.304	.325	-.208	.760(**)	-.395	-.051	.035	.319	1
	Sig. (2-tailed)	.270	.237	.458	.001	.145	.857	.900	.246	
	N	15	15	15	15	15	15	15	15	15

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

4.5.2 CORE REHABILITATION GROUP

In this group, the change in stabilizer pressure and change in stabilizer abdominal were strongly positively correlated together ($r=0.792$, $p<0.001$), together with the change in EMG at L1 and L4 ($r=0.837$, $p<0.001$).

Stabilizer pressure and stabilizer abdominal:

- Showed a positively significant relationship. Participants were therefore utilizing their core muscles. This supports the discussion under 4.4.2.2 and 4.4.2. 7

EMG at L1 and L4:

- Showed a positively significant relationship. As the participants learned to utilize their core more effectively, so their need to recruit global muscles (e.g. Quadratus Lumborum) was decreased. This is evident at L1 and L4. This supports the discussion under 4.4.2.4 and 4.4.2.5

Table 24: Pearson's correlation between changes in outcomes for the core rehabilitation group (n=15)

		Change in RMQ	Change in NRS	Change in algometer	Change in stabilizer pressure	Change in time on stabilizer	Change in EMG L1	Change in EMG L4	Change in EMG abdominal	Change in Stabilizer abdominal
Change in RMQ	Pearson Correlation	1	-.139	.085	.175	.106	.284	.040	-.016	.337
	Sig. (2-tailed)		.620	.762	.533	.706	.305	.887	.954	.220
	N	15	15	15	15	15	15	15	15	15
Change in NRS	Pearson Correlation	-.139	1	-.079	-.220	-.124	-.130	-.004	-.208	-.035
	Sig. (2-tailed)	.620		.779	.431	.660	.644	.989	.457	.902
	N	15	15	15	15	15	15	15	15	15
Change in algometer	Pearson Correlation	.085	-.079	1	-.371	-.413	.156	.402	-.249	-.360
	Sig. (2-tailed)	.762	.779		.174	.126	.579	.138	.371	.187
	N	15	15	15	15	15	15	15	15	15
Change in stabilizer pressure	Pearson Correlation	.175	-.220	-.371	1	.432	-.090	-.224	.005	.792(**)
	Sig. (2-tailed)	.533	.431	.174		.108	.751	.423	.985	.000
	N	15	15	15	15	15	15	15	15	15
Change in time on stabilizer	Pearson Correlation	.106	-.124	-.413	.432	1	-.457	-.384	-.293	.365
	Sig. (2-tailed)	.706	.660	.126	.108		.087	.158	.288	.181
	N	15	15	15	15	15	15	15	15	15
Change in EMG L1	Pearson Correlation	.284	-.130	.156	-.090	-.457	1	.837(**)	-.034	-.111
	Sig. (2-tailed)	.305	.644	.579	.751	.087		.000	.906	.694
	N	15	15	15	15	15	15	15	15	15
Change in EMG L4	Pearson Correlation	.040	-.004	.402	-.224	-.384	.837(**)	1	-.271	-.289
	Sig. (2-tailed)	.887	.989	.138	.423	.158	.000		.328	.297
	N	15	15	15	15	15	15	15	15	15
Change in EMG abdominal	Pearson Correlation	-.016	-.208	-.249	.005	-.293	-.034	-.271	1	.103
	Sig. (2-tailed)	.954	.457	.371	.985	.288	.906	.328		.715
	N	15	15	15	15	15	15	15	15	15
Change in Stabilizer abdominal	Pearson Correlation	.337	-.035	-.360	.792(**)	.365	-.111	-.289	.103	1
	Sig. (2-tailed)	.220	.902	.187	.000	.181	.694	.297	.715	
	N	15	15	15	15	15	15	15	15	15

** Correlation is significant at the 0.01 level (2-tailed).

4.6 REVIEW OF THE OBJECTIVES AND ASSOCIATED HYPOTHESES

The aim was to compare the relative effect of manipulation and core rehabilitation in the treatment of acute mechanical back pain in athletes.

4.6.1 Objectives

4.6.1.1 Objective one

The first objective was to determine the relative effect of manipulation (group A) and core rehabilitation (group B) in athletes with acute mechanical lower back pain in terms of the subjective findings.

Hypothesis 1

It was hypothesized that manipulation and core rehabilitation would have no effect on the pain experienced by the athletes with acute mechanical lower back pain, and no significant difference would be found between the two groups.

Part 1:

Core & manipulation

Reject – there was an effect on pain. Refer to NRS and RMQ. 4.4.1.1 and 4.4.1.2

Part 2:

Core & manipulation

Accept - based on the lack of statistically significant differences between the groups.

4.6.1.2 Objective two

The second objective was to determine the relative effect of manipulation (group A) and core rehabilitation (group B) in athletes with acute mechanical lower back pain in terms of the objective findings.

Hypothesis 2

It was hypothesized that manipulation and core rehabilitation would have no effect on core muscle strength and endurance in athletes with acute mechanical lower back pain, and no significant difference would be found between the two groups.

Part one:

Core (Group B) and manipulation (Group A)

Reject – there was an effect on muscle strength and endurance. Refer to 4.4.2.2 and 4.4.2.3.

Part two:

Core (Group B) and manipulation (Group A)

Accept – based on the lack of statistical significant differences between the groups.

4.6.1.3 Objective three

The third objective was to determine any correlations between the subjective and objective outcomes for Group A and Group B.

Hypothesis 3

It was hypothesized that improvement of pain and core muscle strength, and/or core muscle endurance, would not correlate with the clinical indicators' -

decrease in NRS and RMQ together with surface EMG readings and an increase in algometer readings.

Manipulation

Reject this statement for

a. RMQ versus the Stabilizer time
b. Stabilizer pressure versus abdominal stabilizer pressure
c. EMG for L1 and L4
d. EMG at L4 and EMG abdominal

Accept for all other correlations that were computed.

Core

Reject this statement for

a. Stabilizer pressure and stabilizer abdominal
b. EMG at L1 and L4

Accept for all other correlations

4.7 SUMMARY

Therefore, in summary, both treatments were equally beneficial for most of the quantitative outcomes measured in this study. However, for the outcome of time on the stabilizer, the core rehabilitation group improved at a significantly faster rate than the manipulation group ($p=0.006$).

This final outcome contradicts the literature indicating that there would be muscle function compromise as a result of AMI, but it supports the literature in that it indicates beneficial effects resulting from exercise (i.e. core muscle strength) in patients with lower back pain.

Therefore, it would seem that – with respect to pain control - the theories of Wyke (1981) and Melzack and Wall (1965), are of greater significance in exercise than that of AMI. This is because increased mechanical stimulation of the region (i.e.

lower back), through either manipulation or exercise, is beneficial to the patient. In addition, the results of this study support the work of Hides et al. (1994), which indicates that muscle atrophy is a cause of LBP. As Group B improved more consistently than Group A, addressing issues around muscle weakness / atrophy is of greater benefit to the patient. Hence, the assumption in this study that patients with decreased levels of core muscle strength have increased levels of lower back pain is indirectly supported. This would require further direct research to be validated.

Furthermore, the assertion made by Back Facts (2000) as stated in Chapter 1:

“LBP may result from a steady reduction in physical activity associated with or as a result of the modern fast paced life for most individuals, is probably due to a decrease in manual labour and increase in labour-saving devices which has resulted in the once strong muscle system that is responsible for maintaining our “good’ postures and movements, becoming progressively more inactive as time has gone by resulting in weakened core stability in many individuals which commonly results in an increasing incidence of LBP”, may hold some validity.

CHAPTER FIVE

CONCLUSIONS AND RECOMMENDATIONS

5.1 INTRODUCTION

This chapter deals with the outcomes of the research and makes recommendations with regards to future studies.

5.2 CONCLUSIONS

The purpose of this study was to compare whether manipulation compared with core strengthening showed statistical and clinical evidence of improvement with regards to participants' acute mechanical lower back pain.

It was found that there was no significant difference between the manipulation and the core rehabilitation groups. Therefore, it could be stated that manipulation is equally as effective as core rehabilitation. Although both groups showed improvement with regards to their acute mechanical lower back pain, the core rehabilitation group improved at a significantly faster rate than the manipulation group with regards to endurance on the stabilizer. However, it is acknowledged that these two intervention strategies work by different mechanisms to achieve the same clinical outcomes, thus objective measures such as the PBU which measures only one clinical outcome may be biased to one intervention strategy over the other. This would be in contrast to the NRS which measured pain reduction irrespective of the intervention strategy. Thus, it is recommended that a study in which a third group be introduced (utilizing manipulation and core rehabilitation together) could allow researchers to determine whether a synergistic or antagonistic effect is possible as compared with manipulation and core rehabilitation being performed separately.

5.3 RECOMMENDATIONS

- Homogeneity - although this was addressed, the level of fitness varied between the participants (some were marathon runners whereas others were not), the distance they ran as well as their training habits should be taken into account as this may or may not have affected their core stability. It should also be noted whether they were involved in any other form of sport / physical activity.

- Also, with regards to homogeneity, participants could be further divided into groups with respect to the level of dysfunction, the side of dysfunction and whether they have posterior facet syndrome or sacroiliac syndrome.

- Lack of blinding could have resulted in researcher bias. Having a peer intern or clinician to take objective and subjective measures may result in more reliable readings.

- Larger sample size would increase the validity of the study.

- A needle EMG should be used in future studies in order to obtain more accurate readings.

- Algometer - although this is a very useful tool, unless the exact area of measurement is marked on the participant, it is very difficult to be certain that the device will be placed on the same spot at each consultation.

REFERENCES

Akuthota, V., Nadler, S.F. 2004. Core Strengthening. Archive of Physical Medical Rehabilitation. 85(1):S86-92.

Arokoski, JP., Valta, T., Airaksinen, O., Kankaanpää, M. 2001. Back and Abdominal Muscle Function During Stabilization Exercises. Archives of Physical Medical Rehabilitation. 82: 1089-1098

Back Facts [online]. 2000. Available: www.backcare.com, [Accessed 6/02/2006].

Barr, K.P., Griggs, M., Cadby, T. 2005. Lumbar Stabilisation: Core Concepts and Current Literature, part 1. American Journal of Physical Medical Rehabilitation. 84(6): 473-80.

Bergmann, T., Peterson, D.H. 2002. Chiropractic Technique - Principles and Procedures. 2nd Edition. New York: Mosby, Inc. ISBN 0-323-02016-X.

Biering-Sorensen F. 1984. Physical measurements as risk indicators for low back trouble over a one-year period. Spine. 9: 106-119.

Bergmark, A. 1989. Stability of the Lumbar Spine. A Study in Mechanical Engineering. Actavia Orthopoedic Scandinavia Suppl. 230:1-54.

Bigos, S., Bowyer, O., Braen, G. 2003. Acute Low Back Problems in Adults – Spinal Manipulation. [online] Available: http://www.spineuniverse.com [Accessed 15/05/2006]

Boden, N.L. 2002. The Effectiveness of Spinal Manipulation Versus Spinal Manipulation in Conjunction with Core Stabilization Exercises in the Treatment of

Mechanical Low Back Pain Master's Degree in Technology: Dissertation. Technikon Natal, Berea, Durban, South Africa.

Bono, C.M. 2004. Low Back Pain in Athletes. Journal of Bone and Joint Surgery (American volume). 86(2): 382-397.

Bolton, J.E., Wilkinson, R. 1998. Responsiveness of pain scales: A comparison of three pain intensity measures in Chiropractic patients. Journal of Manipulative and Physiological Therapeutics, 21(1): 1-7.

Bronfort, G., Haas, M., Evans, R.L., Bouter, L.M. 2004. Efficacy of Spinal Manipulation and Mobilisation for Low Back Pain and Neck Pain: A Systematic Review and Best Evidence Synthesis. The Spine Journal 4 (2004)335-356.

Buchthal, H. 1957. Miniature Painting in the Latin Kingdom of Jerusalem, Oxford.

Cairns, M., Harrison, K., Wright, C. 2000. Pressure bio-feedback: a useful tool in the measurement of abdominal muscular dysfunction? Physiotherapy. 86(3):127-138.

Cassidy, D.J., Mierau, D.R. 1992. Pathophysiology of the sacroiliac joint. In: Haldeman, S. (ed). Principles and practice of chiropractic. 2nd Edition. Connecticut: Appleton and Lange.

Chok, B., Lee, R., Latimer, J., Tan, S.B. 1999. Endurance Training of the Trunk Extensor Muscles in People with Sub-acute Low Back Pain. Physical Therapy. 79 (11):1032-1042.

Cox, J.M. 1998. Low Back Pain Mechanism, Diagnosis and Treatment. 6th Edition. ISBN: 0-683-30358-9.

Davis, C., Laskowski, E. 1998-2004. Mayo Foundation for Medical Education and Research [online]. Available at http://www.mayofoundation.com [Accessed 15 March 2006]

Descarreaux, M., Normand, M.C., Laurencelle, L., Dugas, C. 2002. Evaluation of a Specific Home Exercise Program for Low Back Pain. Journal of Manipulative and Physiological Therapeutics. 25(8):497-503.

Di Fabio, R.P. 1992. Efficacy of Manual Therapy. Physical Therapy. 72(12):853-864.

Docrat, A. 1999. A comparison of the epidemiology of low back pain in Indian and Coloured communities in South Africa. Masters Degree in Technology: Chiropractic.Dissertation. Durban Institute of Technology, South Africa.

Drezner, J.A., Herring, S.A. 2001. Managing Low Back Pain; Steps to Optimize Function and Hasten Return to Activity. The Physician and Sports Medicine. 29(8): 37.

Dvorak, J. 1985. Neurological and biomechanical aspects of pain. In Buerger, A.A., Greenman, P.E. eds. Approaches to the validation of spinal manipulation. Springfield,ill. Charles C Thomas. 241 – 266.

Evans. C., Oldreive, W. 2000. A study to investigate whether golfers with a history of low back pain shows a reduced endurance of transverse abdominus. The journal of Manual and Manipulative Therapeutics. 8(4): 162-174.

Fejer, R., Jordan, A., Hartvigsen 2005. Categorising the severity of neck pain: Establishment of cut-points for use in clinical and epidemiological research. Pain. Volume 119, Issue 1-3, 15 December 2005, pages 176-182.

Fischer, A.A. 1986. Pressure Threshold Meter. Its use for quantification of tender spots. Archive of Physical and Medical Rehabilitation. 67: 835-838.

Foxhoven, B., Plante, P. 1996. Athletes with Back Pain - a Trunk Stabilization Program. Strength and Conditioning Journal. 18:69-73.

Gambetta, V., Clark, M. 1999. Hard core training. Training and Conditioning Journal. 10(4):34-40.

Gatterman, M.I. 1995. Foundations of Chiropractic Subluxation. 2nd Edition. Elsevier, Mosby. ISBN 0-323-02468-6.

Gatterman, M.I. 1990. Chiropractic Management of Spine Related Disorders. Baltimore: Williams and Wilkins. ISBN 0-683-03438-3.

Giles, L.G.F and Singer, K.P. 1997. Sacroiliac joint. Clinical Anatomy and Management of Low Back Pain. Volume 1. London: The Bath Press.

Graves, N., Krepcho, M., Mayo, H. 2004. The Journal of Family Practice. 53(8).

Harms-Ringhdal, I. 1993. Muscle strength. International perspectives in Physiotherapy. Churchill-Livingston. 1993. ISBN 0-443-04336-1.

Haslett, C., Chilvers, E.R., Boon, N.A., Colledge, N.R., Hunter, J.A.A. 2002. Davidson's Principals and Practice of Medicine. 19th Edition. ISBN 0-443-07035-0.

Hedrick, A. 2000. Training the Trunk for Improved Athletic Performance. Strength and Conditioning Journal. 22(3): 50-61.

Hides, J.A., Stokes, M.J., Saide, M., Jull, G.A., Cooper, D.H. 1994. Evidence of lumbar multifidus muscle wasting ispilateral to symptoms in patients with acute/ subacute low back pain. Spine. 19(11): 165-172.

Hills, E.C. 2005. Mechanical low back pain [online] Available: http://www.emedicine [Accessed 5/5/2006]

Hodges, P., Richardson, C. 1996. Insufficient muscle stabilization of the lumbar spine associated with low back pain. Spine. 21(22): Pg 2640-50.

Hopkins, J.T., Ingersoll, C.D. 2000. AMI the limiting factor. Journal of Sport Rehabilitation. 9(2):135-159. [Online] Available from: http://www.cast.ilstu.edu/hopkins/ami_the_limiting_factor.htm [Accessed 15/6/2006]

Hubley-Kozey, C.L., Vezina, M.J. 2002. Muscle Activation During Exercises to Improve Trunk Stability in Men With Low Back Pain. Archive of Physical Medical Rehabilitation. 83:1100-1108.

Jull, G.A., Richardson, C.A. 2000. Motor control problems in patients with spinal pain: A new direction for therapeutic exercise. Journal of Manipulative and Physiological Therapeutics. 23(2): 115-117.

Kirkaldy-Willis, W.H., Burton, C.V. 1988. Managing low back pain. 2nd Edition. New York USA: Churchill Livingstone Inc. Pg 249.

Kirkaldy-Willis, W.H., Burton, C.V. 1992. Managing low back pain. 3rd Edition. New York USA: Churchill Livingstone Inc.

Korr, I.M. 1976. The spinal cord as organizer of disease processes: some preliminary perspectives. Journal Am Osteopathic Association. 76: 89 – 99.

Koumantakis, G.A., Watson, P.J., Oldham, J.A. 2005. Trunk Muscle Stabilization Training Plus General Exercise Versus General Exercise Only: Randomised Controlled Trial of Patients With Recurrent Low Back Pain. Physical Therapy. 85(3):209-225.

Leach, R.A. 1994. The Chiropractic Theories – Principals and Clinical Applications. 3rd Edition. Williams and Wilkins. ISBN:0-683-04904-6.

Levangie, P.K., Norkin, C.C. 2001. Joint structure and function. 3rd Edition. Philadelphia: F.A. Davies Company. Pg 71.

Liebenson, C. 2004. Spinal Stabilization-an update. Part 3-training. Journal of Bodywork and Movement Therapies. 2004; 8 Pg 278-285.

Liebenson, C. 1997. Spinal Stabilization training. The therapeutic alternative to weight training. Journal of Bodywork and Movement Therapies. 1997; **1**(2): 87-90.

Liebenson, C. 1996. Rehabilitation of the Spine: A Practitioners Manual. 1996. ISBN0-683-05032-x.

Magee, D.J. 2002. Orthopaedic Physical Assessment. Philadelphia, USA: 2002. ISBN 0-7216-9352-0.

Manga, P., Angus, D.E., Papadopoilis, C., and Swan, W.R. 1993. A study to examine the effectiveness and cost-effectiveness of Chiropractic Management of Low Back Pain. University of Ottawa, Ontario, Canada.

Marshall, P.W.M., Murphy, B.A. 2006. Evaluation of Functional and Neuromuscular Changes After Exercise Rehabilitation for Low Back Pain Using a

Swiss Ball: A Pilot Study. Journal of Manipulative and Physical Therapies. 29(7):550-560.

Marshall, P.W., Murphy, B.A. 2005. Core Stability Exercises On and Off a Swiss Ball. Archive of Physical Medical Rehabilitation. 86:242-249.

Mathews, R. 1997. The approaches of frequent and infrequent application of chiropractic treatment: a study of their relative efficacy in the management of mechanical low back pain. Masters Degree in Technology: Chiropractic. Dissertation. Durban Institute of Technology, South Africa.

McCullach, J., Transfeldt, E. 1997. Macnab's Backache. 3rd Edition. Baltimore. Williams and Wilkins. ISBN 0-683-05797-9.

McGill, S.M., Grenier, S., Kavcic, N., Cholewicki, J. 2003. Co-Ordination of Muscle Activity to Assure Stability of the Lumbar Spine. Journal of Electromyography and Kinesiology. 13(4): 353-359.

McGill, S.M. 2001. Low back stability: from formal description to issues for performance and rehabilitation. Exercises and Sport Sciences Reviews. 29(1):26-31.

Melzack R., Wall, P.D. 1965. Pain mechanisms: A New Theory. Science, 150: 971-979.

Mense, S. 1991. Considerations concerning the neurological basis of muscle pain. Canadian Journal Physiological Pharmacology. 69: 610 – 616.

Mills, J.D., Taunton, J.E., Mills, W.A. 2005. The Effect of a 10-week Training Regimen on Lumbo-pelvic Stability and Athletic Performance in Female Athletes: A Randomized-Controlled Trial. Physical Therapy in Sport. 6(2): 60-66.

Moore, K.L., Dalley, A.F. 1999. Clinically Orientated Anatomy. 4th Edition. Baltimore. Williams and Wilkins. ISBN 0-683-06141-0.

Morris, M.R. 1983. A study of the natural history of back pain: Part I: Development of a reliable and sensitive measure of disability in low back pain. 1983; 8:141-144.

Mouton, J. 1996. Understanding Social Research. Pretoria: Van Schaik Publishers. Pg 132.

Naidoo, T.P. 2002. The effect of Segmental manipulation of the Cervical spine on the grip strength in patients with mechanical cervical spine dysfunction. Masters Degree in Technology: Chiropractic. Dissertation. Durban Institute of Technology, South Africa.

Nansel, D.D., Waldorf, T., Cooperstein, R. 1993. Effect of Cervical Spinal Adjustments on Lumbar Paraspinal muscle tone: Evidence for Facilitation of intersegmental tonic neck reflexes. Journal of Manipulative and Physiological Therapeutics. **16**(2): 91-95.

Newton, A. 2004. Core stabilization, Core Coordination. {online} Available: http://www.corestabilization,corecoordination.

Panjabi, M.M. 2003. Clinical Spinal Instability and Low Back Pain. Journal of Electromyography and Kinesiology. 13:371-379.

Panjabi, M.M. 1992. The Stabilizing System of the Spine. Part I. Function, Dysfunction, Adaption, and Enhancement. Journal of Spinal Disorders. 5(4):383-389.

Panjabi, M.M. 1992. The Stabilizing System of the Spine. Part II. Neutral Zone and Instability Hypothesis. Journal of Spinal Disorders. 5(4):390-397.

Peterson, D.H. and Bergmann, T.F. 2002. Chiropractic Technique: Principles and Procedures. 2nd Edition. Missouri.Mosby. ISBN 0-323-02016-X.

Plaugher, G. 1993. Textbook of Clinical Chiropractic – A specific Biomechanical approach. Baltimore. Williams and Wilkins. ISBN 0-683-06897-0.

Poul, J., West, J., Buchannan, N., Grahame, R. 1993. Local action transcutaneous Flurbiprofen in treatment of soft tissue rheumatism. British Journal of Rheumatology. 32: Pg 1000-1003.

Rainville, J., Hartigan, C., Jouve, C., Martinez, E. 2004. The Influence of Intense Exercise-based physical therapy program on back pain anticipated before and induced by physical activities. The Spine Journal 4. (2004) 176-183.

Richardson, C.A., Hides, J., Hodges, P. 2004. Principles of the “segmental stabilization” exercise model. In : therapeutic exercise for lumbopelvic stabilization: A motor control approach for the treatment and the prevention of low back pain. 2nd Edition. Sydney : Churchill Livingston. Pg 175 - 183.

Richardson, C.A., Jull, G.A., Hodges, P., Hides, J. 1999. Therapeutic Exercise For Spinal Segmental Stabilization in Low Back Pain – Scientific Basis and Clinical approach. Edinburgh: Churchill Livingstone.

Richarson, C.A., Jull, G.A. 1995. Muscle Control – Pain Control. What Exercises would you Prescribe? Manual Therapy. 1:2-10.

Roland, M., Morris, R. 1983. A study of the natural history of back pain: Part I: Development of a reliable and sensitive measure of disability in low back pain. 8:141-144.

Sakamoto, N., Yamashita, T., Takebayashi, T., Sekine, M., Ishii, S. 2001. An Electrophysiological Study of mechanoreceptors in the sacroiliac joint and adjacent tissues. Spine, Oct 26(20) E468-471. [Online] Available from: http://ipsapp006.lwwonline.com/content/getfile/1140/142/8/fulltext.htm [Accessed 10/6/2006].

Sandoz, R.W. 1978. Some Reflex Phenomena Associated with Spinal Derangements and Adjustments. Annuals of the Swiss Chiropractic Association. 45 – 65.

Sandoz, R. 1976. Some Physical Mechanisms and effects of Spinal adjustments. Annuals of the Swiss Chiropractic Association. 6: 91-142.

Schaefer, R.C., Faye, L.J. 1990. Motion palpation and Chiropractic technique: Principles of dynamic Chiropractic. 2nd Edition. California, USA: The motion palpation institute. ISBN 0924889-00-4.

Schaefer, R.C., Faye, L.J. 1989. Motion palpation and Chiropractic technique: Principles of dynamic Chiropractic. 1st Edition. California, USA: The motion palpation institute.

Seth, S.D. 1999. Textbook of Pharmacology. 2nd ed. Churchill Livingstone. Pg 174.

Silfies, S.P., Squillante, D., Maurer, P., Westcott, S., Karduna, A. R. 2005. Trunk muscle recruitment patterns in specific chronic low back pain populations. Clinical Biomechanics, Volume 20, Issue 5, June 2005, Pages 465-473.

Stabilizer – Pressure Biofeedback Instruction Manual. (Chatanooga Group Inc., 4717 Adams Road, Hixson TN 37343, USA.)

Stanton, R., Reaburn, P.R., Humphries, B. 2004. The Effect of Short-Term Swiss Ball Training on Core Stability and Running Economy. The Journal of Strength and Conditioning Research. 18(3): 522-528.

Stevens, V.K., Coorevits, P.L., Bouche, K.G., Mahieu, N.N., Vanderstraeten, G.G., Danneels, L.A. 2006. The influence of specific training on trunk muscle recruitment patterns in healthy subjects during stabilization exercises. Manual Therapy. 1(4):1-9. .

Suter, E., McMorland, G., Herzog, W., Bray, R. 2000. Conservative lower back treatment reduces inhibition in knee-extensor muscles: A randomized controlled trial. Journal of Manipulative and Physiological Therapeutics, 23(2): 76-80.

Suter, E., McMorland, G., Herzog, W., Bray, R. 1999. Decrease in quadriceps inhibition after sacroiliac manipulation in patients with anterior knee pain. Journal of Manipulative and Physiological Therapeutics, 22(3): 149-153.

Suter, E., Herzog, W., Conway, P.J., Zang, Y.T. 1994. Reflex response associated with manipulative treatment in the thoracic spine. Journal of the Neuromuscularskeletal system, 2(3): 123-130.

Taylor, J., O'Sullivan, P. 2000. Lumbar segmental instability: pathology, diagnosis, and conservative care. Physical therapy of the low back. 3rd Edition. New York : Churchill Livingston. Pg 201 – 247.

Travell J.G., Simons, D.G., Simons, L.S. 1999. Myofascial Pain and Dysfunction: The Trigger Point Manual – Volume 1. Upper Half of the Body. 2nd Edition. Philidelphia: Lippencott Williams and Wilkins. ISBN: 0-683-08366-X.

Travell, J.G., Simons, D.G., Simons, L.S. 1999. Myofascial Pain and Dysfunction: The Trigger Point Manual – Volume 2. The Lower Extremities. Philidelphia: Lippencott Williams and Wilkins. ISBN: 0-683-08367-8.

Van der Meulen, A.G. 1997. An epidemiological investigation of low back pain in a formal Black South African township. Master's Degree in Technology: Dissertation. Technikon Natal, Berea, Durban, South Africa.

Van Tulder, M.W., Furnan, A.D., Gagnier, J.J. 2005. Complementary and Alternative Therapies for Low Back Pain. Best Practice and Research Clinical Rheumatology. 19(4): 639-654.

Van Tulder, M.W., Koes, B.W. and Bouter, L.M. 1997. Conservative Treatment of Acute and Chronic Non-specific Low Back Pain. A Systemic Review of Randomised Controlled Trials of the Most Common Interventions. Spine, 22(18): 2128-2156.

Vernon, H., Mrozek, J. 2005. A revised definition of Manipulation. Journal of Manipulative and Physiological Therapeutics. 28(1): 68-72.

Wang, S.S., Whitney, S.L., Burdett, R.G., Janosky, J.E. 1993. Lower Extremity Muscular Flexibility in Long Distance Runners. Journal of Orthopedic and Sports Physical Therapy. 17(2): 102-107.

Weinstein, J.N. 1992. Clinical Efficacy and Outcomes in the Diagnosis and Treatment of Low Back Pain. ISBN0-88167-841-4.

White, A.A., Panjabi, M.M. 1990. Clinical biomechanics of the spine. 2nd Edition. Philadelphia, JB Lippincott.

William, E. (ed). 1997. Rehabilitation techniques in sports medicine. 2nd Edition. Pg. 144.

WordNet, a lexical database for the English language. 2007. Cognitive Science Laboratory, Princeton University, 221 Nassau St., Princeton, NJ 08542. [Online] Available from: www.wordnet.princeton.edu/perl/webwn. [Accessed 07/02/2006]

Wyke, B.D. 1981. The neurology of joints: a review of general principals. Clinical Rheumatological Disorders. 7: 223 – 239.

Wyke, B.D. 1985. Articular Neurology and Manipulative Therapy. In Glasgow, E. F., Tworney, L.T., Scull, E.R., Kleynhans, A.M. Aspects of Manipulative Therapy. New York: Churchill Livingstone. Pg72 -77.

APPENDICES:

APPENDIX A

Are you between 18 – 45 years old and run 10Km a week?

DO YOU SUFFER FROM

LOW BACK PAIN

Research is currently being carried out at the Durban University of Technology

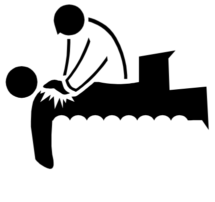
Chiropractic Day Clinic

FREE TREATMENT

Is available to those who qualify to take part in this study

For more information contact Jennifer on

031 2042205 / 2512 / 082 887 5534



APPENDIX B

LETTER OF INFORMATION:

Dear Participant.
Welcome to my research project.

Title of the research:
The relative effect of manipulation and core rehabilitation in the treatment of acute mechanical low back pain in athletes.

Name of Research student:
Jennifer Campbell
Contact number: 031 2042205 / 082 8875534

Name of Research Supervisor:
Dr. C Korporaal (M Tech Chiropractic, CCFC, CCSP, ICCSD)
Contact number: 031 2042611

Name of Research Co-supervisor:
Dr. R White (M Tech: Chiropractic)
Contact number: 033 3422649 / 0845138721

Institution: Durban University of Technology (DUT)
You have been selected to take part in a research study which is looking at the relative effect of manipulation and rehabilitation in the treatment of athletic patients suffering from acute mechanical lower back pain.

Thirty participants will be required to complete this study. Each participant will have a standard clinical treatment, which include either manipulation or core rehabilitation of the core stability muscles for the purposes of this study.

Research process:
The first consultation will take place at the DUT Chiropractic Day Clinic. Here participants will be screened for suitability for this study, which will be determined by a case history, physical examination and a lumbar spine regional examination, and specific measurements of your low back pain and your core stability will be measured.

All treatments will be performed, under the supervision of a qualified chiropractor, by the research student and will be free of charge.

Risk / discomfort:
The research study is safe, although participants may experience transient tenderness and stiffness that is common post interventions used in this study; it is unlikely to cause any adverse side effects.

Remuneration and costs:
All treatments will be free of charge and participants taking part in the study will not be offered any other form of remuneration for taking part in the study.
On completion of your participation in this study you are eligible for two free treatments at the Durban Institute of Technology Chiropractic Day Clinic.

All patient information is confidential and the results of the study will be made available in the Durban Institute of Technology library in the form of a mini-dissertation.

Implications for withdrawal from the research:
You are free to withdraw at any stage of the research project.

Benefits of the study:
Your participation and co-operation will assist the Chiropractic profession in expanding its knowledge and the treatment protocol for mechanical lower back pain, and thus making future rehabilitation of patients suffering from this condition more successful.

Confidentiality:
All participant information is confidential and the results will be used for research purposes only. It will be stored in the Chiropractic Day Clinic for 5 years, after which it will be shredded. Supervisors and senior clinic staff may however be required to inspect the records.

Persons to contact with problems or questions:
Should you have any further queries and you would like them answered by an independent source, you can contact my supervisor on the number above or alternatively you could contact the Faculty of Health Sciences Research and Ethics Committee as per Mr. Vikesh Singh at (031) 2042701.

Thank you for your participation.
Yours sincerely,

____________________________ ____________________________

Jennifer Campbell (Research Student) **Dr. C. Korporaal (Supervisor)**
(M Tech Chiropractic, CCFC, CCSP, ICCSD)

APPENDIX C

NRS Pain Rating Scale

Patient Name:

Date:
Pain Severity Scale:
Rate your usual level of pain today by checking one box on the following scale:

0	1	2	3	4	5	6	7	8	9	10

No pain **Excruciating**

Date:
Pain Severity Scale:
Rate your usual level of pain today by checking one box on the following scale:

0	1	2	3	4	5	6	7	8	9	10

No pain **Excruciating**

Date:
Pain Severity Scale:
Rate your usual level of pain today by checking one box on the following scale:

0	1	2	3	4	5	6	7	8	9	10

No pain **Excruciating**

As adapted from (Bolton and Wilkinson, 1998).

APPENDIX D

Core stability assessment techniques using the Stabiliser Biofeedback Device

The prone test for transverses abdominis and internal oblique

- Place 3-chamber pressure cell under the abdomen and inflate to baseline of 70 mmHg.
- Draw abdominal wall up and in without moving the spine or pelvis.
- Pressure should decrease 6-10 mmHg.
- Patient must attempt to maintain this contraction for as long as the participant is able.
- Measurement of time at which the patient can no longer hold the contraction at the baseline level (70mmmHg – 6 to 10 mmHg), within the set time period for the test.
- Measurement of the change in mmHg from the baseline level (70mmHg – 6 to 10mmHg) up to the end of the contraction.

Four Point Kneeling Position

- Hips are over the knees and the shoulders are directly over the hands.
- Elbows must be relaxed, and not forced into extension.
- Spine in the neutral position.

Procedure:

- Patient relaxes abdominal wall (which examiner can feel by gently palpating abdomen).
- Patient is asked to breathe in and out, and then without breathing in, to draw in the abdominal wall towards the spine and up towards the ribs.
- Examiner can help the co-ordination by sweeping the palpating hand in the direction required.
- Patient is now asked to perform the movement without respiration.
- Contraction should take place in a controlled and slow manner.
- Once the contraction is achieved, slow shallow breathing can commence and the contraction is held for 10 seconds.

APPENDIX E

Patient Diary:

Patient Name:

Week 1:

	Date	Sign	Date	Sign	Date	Sign
Exercise 1						
Exercise 2						
Exercise 3						
Exercise 4						

Week 2:

	Date	Sign	Date	Sign	Date	Sign
Exercise 1						
Exercise 2						
Exercise 3						
Exercise 4						

APPENDIX F

DURBAN INSTITUTE OF TECHNOLOGY
CHIROPRACTIC DAY CLINIC
CASE HISTORY

Patient: ______________________ Date: ________

File #: ________ Age: ________
Sex: ________ Occupation: ______________________

Intern: ______________________ Signature: ______________________

FOR CLINICIANS USE ONLY:
Initial visit
Clinician: Signature:

Case History:

Examination: Previous: Current:

X-Ray Studies: Previous: Current:

Clinical Path. lab: Previous: Current:

CASE STATUS:

PTT: Signature: Date:

CONDITIONAL:
Reason for Conditional:

Signature: Date:

Conditions met in Visit No:	Signed into PTT:	Date:
Case Summary signed off:		Date:

<u>Intern's Case History:</u>

1. Source of History:

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

3. Present Illness:

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

4. Other Complaints:

5. past Medical History:

< General Health Status

< Childhood Illnesses

< Adult Illnesses

< Psychiatric Illnesses

< Accidents/Injuries

< Surgery

< Hospitalizations

6. Current health status and life-style:

< Allergies

< Immunizations

< Screening Tests incl. x-rays

< Environmental Hazards (Home, School, Work)

< Exercise and Leisure

< Sleep Patterns

< Diet

< Current Medication
Analgesics/week:

< Tobacco

< Alcohol

< Social Drugs

7. Immediate Family Medical History:

< Age

< Health

< Cause of Death

< DM

< Heart Disease

< TB

< Stroke

< Kidney Disease

< CA

< Arthritis

< Anaemia

< Headaches

< Thyroid Disease

< Epilepsy

< Mental Illness

< Alcoholism

< Drug Addiction

< Other

8. Psychosocial history:

< Home Situation and 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 G

Durban Institute of Technology
PHYSICAL EXAMINATION: SENIOR

Patient Name : ______________________________ **File no :** ____________ **Date :** __________
Student : **Signature :**

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

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

SYSTEM SPECIFIC EXAMINATION:
CARDIOVASCULAR EXAMINATION
RESPIRATORY EXAMINATION
ABDOMINAL EXAMINATION
NEUROLOGICAL EXAMINATION
COMMENTS

Clinician: **Signature :**

APPENDIX H

REGIONAL EXAMINATION - LUMBAR SPINE AND PELVIS

Patient:______________________File#:________________ Date:_________

Intern\Resident:_____________________________ Clinician:________________________________

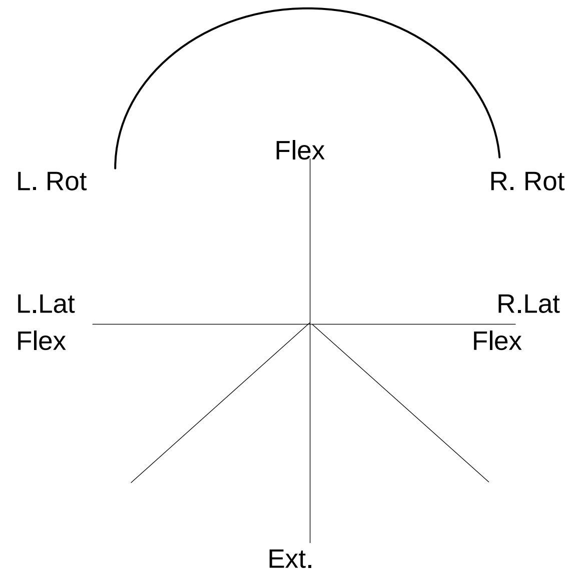
STANDING:

Posture– scoliosis, antalgia, kyphosis
Body Type
Skin
Scars
Discolouration

Minor’s Sign
Muscle Tone
Spinous Percussion
Scober’s Test (6cm)
Bony and Soft Tissue Contours

GAIT:

Normal walking
Toe walking
Heel walking
Half squat



ROM:

Forward Flexion = 40-60° (15 cm from floor)
Extension = 20-35°
L/R Rotation = 3-18°
L/R Lateral Flexion = 15-20°

Which movt. reproduces the pain or is the worst?

- Location of pain
- Supported Adams: Relief? (SI)
  Aggravates? (disc, muscle strain)

SUPINE:

Observe abdomen (hair, skin, nails)
Palpate abdomen\groin
Pulses - abdominal
- lower extremity
Abdominal reflexes

		Degree	**LBP?**	**Location**	**Leg pain**	**Buttock**	**Thigh**	**Calf**	**Heal**	**Foot**	**Braggard**
SLR	**L**										
	R										

	L	**R**
Bowstring		
Sciatic notch		
Circumference (thigh and calf)		
Leg length: actual -		
apparent -		
Patrick FABERE: pos\neg – location of pain?		
Gaenslen’s Test		
Gluteus max stretch		
Piriformis test (hypertonicity?)		
Thomas test: hip \ psoas? \ rectus femoris?		
Psoas Test		

<u>SITTING:</u>
Spinous Percussion
Valsalva
Lhermitte

		Degree	LBP?	Location	Leg pain	Buttock	Thigh	Calf	Heal	Foot	Braggard
tripod	**L**										
	R										

Slump 7 test	**L**										
	R										

LATERAL RECUMBENT:	L	R
Ober's		
Femoral n. stretch		
SI Compression		

PRONE:	**L**	**R**
Gluteal skyline		
Skin rolling		
Iliac crest compression		
Facet joint challenge		
SI tenderness		
SI compression		
Erichson's		
Pheasant's		

MF tp's	**Latent**	**Active**	**Radiation**
QL			
Paraspinal			
Glut Max			
Glut Med			
Glut Min			
Piriformis			
Hamstring			
TFL			
Iliopsoas			
Rectus Abdominis			
Ext/Int Oblique muscles			

<u>NON ORGANIC SIGNS:</u>
Pin point pain
Axial compression
Trunk rotation
Burn's Bench test

Flip Test
Hoover's test
Ankle dorsiflexion test
Repeat Pin point test

NEUROLOGICAL EXAMINATION

Fasciculations

Plantar reflex

level	Tender?	Dermatomes		DTR		
		L	R		L	R
T12				Patellar		
L1				Achilles		
L2						
L3				Proproception		
L4						
L5						
S1						
S2						
S3						

Action	Muscles	L	R	
Lateral Flexion spine	Muscle QL			
Hip flexion	Psoas, Rectus femoris			5+ Full strength
Hip extension	Hamstring, glutes			4+ Weakness
Hip internal rotat	Glutmed, min;TFL, adductors			3+ Weak against grav
Hip external rotat	Gluteus max, Piriformis			2+ Weak w\o gravity
Hip abduction	TFL, Glut med and minimus			1+ Fascic w\o gross movt
Hip adduction	Adductors			0 No movement
Knee flexion	Hamstring,			
Knee extension	Quad			W - wasting
Ankle plantarflex	Gastroc, soleus			
Ankle dorsiflexion	Tibialis anterior			
Inversion	Tibialis anterior			
Eversion	Peroneus longus			
Great toe extens	EHL			

BASIC THORACIC EXAM

History
Passive ROM
Orthopedic

BASIC HIP EXAM

History ROM: Active
Passive : Medial rotation : A) Supine (neutral) If reduced - hard \ soft end feel
B) Supine (hip flexed): - Trochanteric bursa

APPENDIX I

DURBAN INSTITUTE OF TECHNOLOGY

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

Date: ***Visit:*** ***Intern:***
Attending Clinician: ***Signature:***

S: ***Numerical Pain Rating Scale (Patient)*** ***Intern Rating*** ***A:***
Least 0 1 2 3 4 5 6 7 8 9 10 Worst

0: ***P:***

E:

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

Date: ***Visit:*** ***Intern:***
Attending Clinician: ***Signature:***

S: ***Numerical Pain Rating Scale (Patient)*** ***Intern Rating*** ***A:***
Least 0 1 2 3 4 5 6 7 8 9 10 Worst

O: ***P:***

E:

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

Date: ***Visit:*** ***Intern:***
Attending Clinician: ***Signature***

S: ***Numerical Pain Rating Scale (Patient)*** ***Intern Rating*** ***A:***
Least 0 1 2 3 4 5 6 7 8 9 10 Worst

O: ***P:***

E:

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

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

Date: ***Visit:*** ***Intern:***
Attending Clinician: ***Signature:***

S: ***Numerical Pain Rating Scale (Patient)*** ***Intern Rating*** ***A:***
Least 0 1 2 3 4 5 6 7 8 9 10 Worst

0: ***P:***

E:

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

Date: ***Visit:*** ***Intern:***
Attending Clinician: ***Signature:***

S: ***Numerical Pain Rating Scale (Patient)*** ***Intern Rating*** ***A:***
Least 0 1 2 3 4 5 6 7 8 9 10 Worst

O: ***P:***

E:

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

Date: ***Visit:*** ***Intern:***
Attending Clinician: ***Signature***

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

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

O: ***P:***

E:

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

APPENDIX J

INFORMED CONSENT FORM
(To be completed by patient / subject)

Date:

Title of research project:
The relative effect of manipulation and core rehabilitation in the treatment of acute mechanical low back pain in athletes.

Name of supervisor: Dr C. Korporaal (M Tech Chiropractic, CCFC, CCSP, ICCSD)
Tel: 031-2042611 (Work) 0832463562 (Cell)

Name of research student: Jennifer Campbell
Tel: 031 2042205 (work) 082 887 5534 (cell)

Please circle the appropriate answer	**YES /NO**	
1. Have you read the research information sheet?	Yes	No
2. Have you had an opportunity to ask questions regarding this study?	Yes	No
3. Have you received satisfactory answers to your questions?	Yes	No
4. Have you had an opportunity to discuss this study?	Yes	No
5. Have you received enough information about this study?	Yes	No
6. Do you understand the implications of your involvement in this study?	Yes	No
7. Do you understand that you are free to withdraw from this study	Yes	No
at any time without having to give any a reason for withdrawing, and without affecting your future health care?	Yes	No
8. Do you agree to voluntarily participate in this study?	Yes	No
9. Who have you spoken to?		

____

Please ensure that the researcher completes each section with you
If you have answered NO to any of the above, please obtain the necessary information before signing.

Please print in block letters:

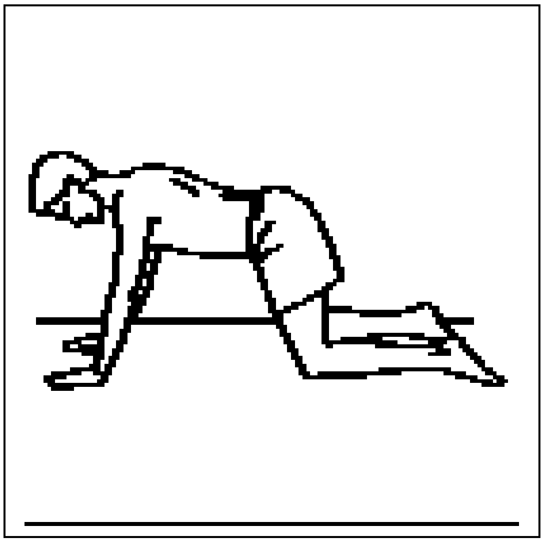
Patient /Subject Name:______________________Signature: ________________

Witness Name:_____________________________Signature:_________________

Research Student Name:_____________________Signature:_________________

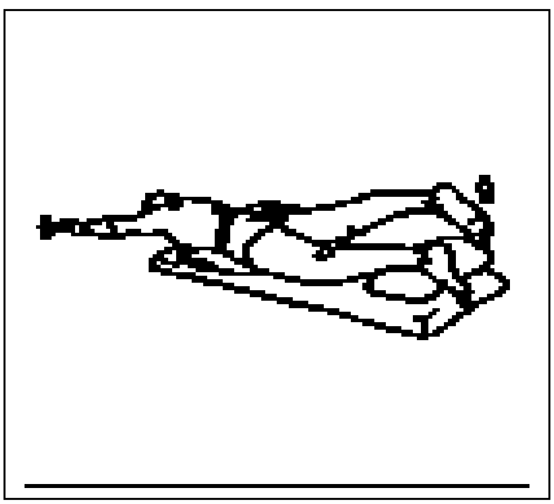
APPENDIX K

Exercise 1:



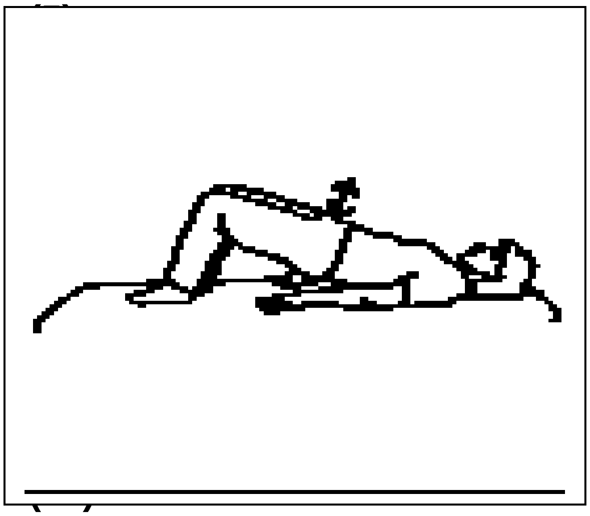
- Crawling position.
- Pull your stomach in.
- Hold for 5 seconds.
- Repeat 10 times. (Mornings and evenings)

Exercise 2:



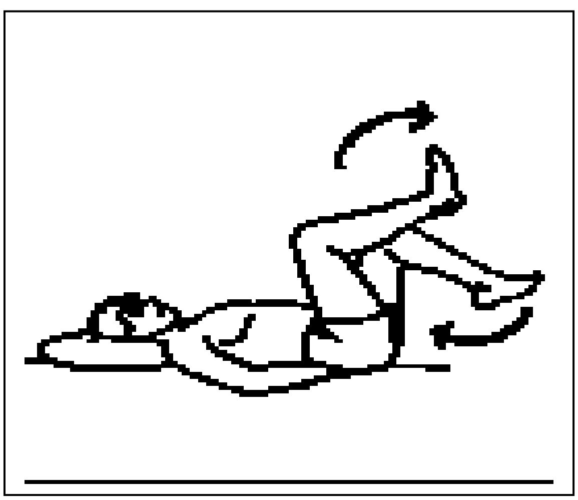
- Lying face down with your arms above your head on the floor (you can have a pillow under your stomach and one under your ankles)
- Lift opposite arm and leg approximately 20cm off the floor and stretch.
- Hold the stretching for approximately 5 seconds – relax, and repeat with other side.
- Repeat 10 times. (Mornings and evenings)

Exercise 3:



- Lying on your back with your knees bent and feet on the floor.
- Lift your pelvis and lower back (gradually vertebra by vertebra) off the floor.
- Hold the position. Lower down slowly returning to the starting position.
- Hold for 5 seconds.
- Repeat 10 times. (Mornings and evenings)

Exercise 4:



- Lying on your back with hands supporting your pelvis.
- Make a cycling movement with one leg 10 times.
- Repeat with the other leg.

- Lying on your back with hands supporting your pelvis.
- Make a cycling movement with both your legs for 1 minute.
- (Mornings and evenings)

(Physio Tools Ltd)

APPENDIX L

Patient name:

Algometer Readings:

	Date	Location	Readings		Av
Reading 1					
Reading 2					
Reading 3					

Stabiliser Readings:

	Date	Reading / Time						Av Reading / Time	
		Stab	**Tim**	**Stab**	**Tim**	**Stab**	**Tim**	**Stab**	**Time**
Reading 1									
Reading 2									
Reading 3									

Surface EMG:

	Date	Location	Readings						Av	
			Bef	**Aft**	**Bef**	**Aft**	**Bef**	**Aft**	**Bef**	**Aft**
Reading 1		**L1**								
		L4								
Reading 2		**L1**								
		L4								
Reading 3		**L1**								
		L4								

	Date		Bef	Aft	Stab	Time
Reading 1		**Abdo**				
Reading 2		**Abdo**				
Reading 3		**Abdo**				

APPENDIX M

LOW BACK PAIN AND DISABILITY QUESTIONNAIRE

NAME: ________________________________ DATE: _________ AGE:_____ SCORE: ________

When your back hurts, you may find it difficult to do some of the things you normally do. Mark only the sentences that describe you today by circling the corresponding number:

1. I stay at home most of the time because of my back.
2. I change position frequently to try and get my back comfortable.
3. I walk more slowly than usual because of my back.
4. Because of my back, I am not doing any jobs that I usually do around the house.
5. Because of my back, I use a handrail to get up stairs.
6. Because of my back, I lie down to rest more often.
7. Because of my back, I have to hold onto something to get out of an easy chair.
8. Because of my back, I try to get other people to do things for me.
9. I get dressed more slowly than usual because of my back.
10. I stand up for only short periods of time because of my back.
11. Because of my back, I try not to bend or kneel down.
12. I find it difficult to get out of a chair because of my back.
13. My back is painful almost all the time.
14. I find it difficult to turn over in bed because of my back.
15. My appetite is not very good because of my back.
16. I have trouble putting on my socks (or stockings) because of pain in my back.
17. I walk only short distances because of my back.
18. I sleep less well because of my back.
19. Because of back pain, I get dressed with help from someone else.
20. I sit down for most of the day because of my back.
21. I avoid heavy jobs around the house because of my back.
22. Because of my back I am more irritable and bad tempered with people than usual.
23. Because of my back, I go up stairs more slowly than usual.
24. I stay in bed most of the time because of my back.

From Roland M, Morris R. A study of the natural history of back pain: Part I: Development of a reliable and sensitive measure of disability in low back pain. 1983; 8:141-144.
The original 24 item Roland-Morris Questionnaire is displayed. The RM-18 deletes 2, 15, 17, 19, 20 and 24 without affecting it quality.