

**THE RELATIVE EFFECT OF MANIPULATION AND CORE  
REHABILITATION IN THE TREATMENT OF ACUTE MECHANICAL  
LOW BACK PAIN IN SEDENTARY PATIENTS**

**By**

**Renee Joy Higgs**

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I, Renee Joy Higgs do declare that this dissertation is representative of my own  
work.

Signed: \_\_\_\_\_ Date: \_\_\_\_\_

Approved for final submission

Signed: \_\_\_\_\_ Date: \_\_\_\_\_  
**Dr C Korporaal (M Tech: Chiropractic, CCFC, CCSP, and ICCSD)**  
Supervisor

Signed: \_\_\_\_\_ Date: \_\_\_\_\_  
**Dr R White (M Tech: Chiropractic)**  
Co-Supervisor

## **Dedication**

To my Parents Derek and Noreen Prior you have both taught me the important things in life, hard work, determination, perseverance and laughter, to Raymond Lundy who will always be sorely missed and finally to Percy for always being my rock and without whom life could never be this sweet.

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## **ABSTRACT**

The aim of this research was to investigate the relative effectiveness of manipulation versus core rehabilitation in the treatment of acute mechanical low back pain in sedentary patients.

Recent research has found that dysfunction of the primary core stabiliser muscles is linked with an increasing number of the general population suffering from low back pain; this is thought to be due to the fact that people in general are living more sedentary lifestyles.

The Aims and Objectives of this study were to determine the relative effect of manipulation and core rehabilitation in sedentary patients suffering from acute mechanical low back pain in terms of subjective findings, objective findings and to determine any correlations between these findings

Thirty-two participants, with acute low back pain participated in the study. They received treatment over a period of three weeks, two treatments in the first week, two treatments in the second week and a follow up seven days later. Group A received a spinal manipulation while Group B received core rehabilitation exercises. Readings were taken at three time points, namely visit one, three and five before the treatment, they included the following readings: Numerical Pain

Rating Scale, Algometer, Roland Morris Low Back Pain and Disability Questionnaire, Biofeedback Stabiliser and the Surface EMG.

The results showed that there was no differential ( $p < 0.05$ ) treatment effect between the two Groups, and that both Groups showed a clinical improvement in their low back pain.

In conclusion, it appears that even though both these treatment protocols have very different mechanisms of action, both can be effective treatment protocols and that core rehabilitation exercises when properly performed are as effective as manipulation in the treatment of acute low back pain.

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## **DEFINITIONS**

- **Acute**

Pain this has a rapid onset and is pronounced symptoms all of which are of short duration (Magee, 2002).

- **Core Rehabilitation**

The rehabilitation and retraining of the core stabilizers of the Lumbar Spine.

- **Manipulation or Adjustment**

A manipulation or adjustment is a passive manual maneuver 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 Mrozek et al., 2005).

- **Mechanical Low Back Pain**

Pain is usually cyclical and often referred to the buttocks and thighs, while morning stiffness is commonly associated. Pain is often associated with the start of movement and flexion movements as well as when the patient returns to the erect position. Over the course of the day, pain often

becomes worse but is relieved by a change in position. Relief is obtained by lying down especially in the supine position. (Magee, 2002).

- **Mechanoreception**

A receptor that is excited by a mechanical pressure or distortion, such as touch, sound and muscle contraction (Redwood, 1997).

- **Nociception**

A receptor that is preferentially sensitive to a noxious stimulus or to a stimulus that could turn noxious if it were to be prolonged.

- **Proprioception**

Sensory perception of movement or position within the body (Magee, 2002).

- **Sedentary**

Sedentary individuals were defined as those who undertake no leisure time physical activities and those who undertake less than 30 minutes of physical activity each day. (President's Council on Physical Fitness and Sports Research, 2002).

- **Subluxation**

The temporary immobilisation of a joint in a position that it may normally occupy during any phase of movement (Redwood, 1997).

## **CHAPTER 1**

### **1.1 Introduction**

Low back pain is one of the most costly disabilities in modern society and every year thousands of working hours are lost because of it (Manga et al., 1993). Literature indicates (Goubert et al., 2004, Andersson et al., 1991; Dionne, 1999) that this is a result of the fact that between 60 and 80% of the population will at some point in their lives suffer from low back pain, while the estimated percentage of people suffering from low back pain at any point in time is in the region of 20-30% (Cassidy et al., 1992). Studies conducted in South Africa have indicated the incidence of low back pain amongst black South Africans to be 57.6% (Van der Meulen, 1997) while that amongst the Indian and Coloured population was shown to be between 70 and 80% (Docrat, 1999). It has been noted that there is a higher incidence of low back pain amongst women (Magee et al., 2002).

Although there are thousands of causes of low back pain, some of the most common causes are of a mechanical nature and include Myofascial Pain and Dysfunction Syndromes, Posterior (Lumbar) Facet Syndrome and Sacroiliac Syndrome (Kirkaldy-Willis et al., 1992).

Recent research has found that dysfunction of the primary core stabiliser muscles is linked with an increasing number of the general population suffering from low back pain (Hodges et al., 1996b). Although the cause of this finding is yet unknown, it has been hypothesized that over recent years there has been a steady decline in physical activity in the general population. This is thought to be mainly due to our fast paced lives, which has led to a steady decline in our once strong core muscle system (Jorgensen et al., 1997). The unfortunate implication of this is that those muscles which were once our strong postural muscles are now weak and ineffective, therefore making us more prone to both spinal injuries and low back pain (Hodges et al., 1996b).

Thus in more recent years there has been a move towards the use of rehabilitation and rehabilitative exercises, with regards to the core stability muscles in the treatment of mechanical low back pain and there have been some positive results produced using this method of treatment (Hides et al., 1996; O'Sullivan et al., 1997; Hides et al., 2001; Danneels et al., 2001). As a result and in conjunction with anecdotal evidence, there is a suggestion that chiropractors are moving away from a purely manipulative treatment when dealing with mechanical low back pain and are instead using a combined treatment of both manipulation and rehabilitation, which is thought to have better clinical outcomes than manipulation alone (Liebenson, 1996). Recently conducted research has indicated that a combination of manipulation combined with rehabilitation did not

in fact improve clinical outcomes (Boden, 2000) but, it has not yet been shown that core stability rehabilitation alone as a primary intervention, is effective.

Therefore the aim of this research was to investigate the relative effect of manipulation and core rehabilitation in the treatment of acute mechanical low back pain in sedentary patients.

## **1.2 Aims and Objectives**

**The relative effect of manipulation and core rehabilitation in the treatment of acute mechanical low back pain in sedentary patients.**

1.2.1 To determine the relative effect of manipulation (Group A) and core rehabilitation (Group B) in sedentary patients suffering from acute mechanical low back pain in terms of the subjective findings.

1.2.2 To determine the relative effect of manipulation (Group A) and core rehabilitation (Group B) in sedentary patients suffering from acute mechanical low back pain in terms of the objective findings.

1.2.3 To determine any correlations between the subjective and objective outcomes for Group A and Group B.

### **1.3 Hypotheses**

- 1.3.1 Patients suffering from acute mechanical low back pain should not show a clinical improvement (subjective and objective) when treated using manipulation.
- 1.3.2 Patients suffering from acute mechanical low back pain should not show a clinical improvement (subjective and objective) when treated using core stability exercises.

### **1.4 Rationale**

- 1.4.1 Health researchers have shown that low back pain is one of the most costly health problems in the world today (Manga et al., 1993) and therefore investigating means to not only treat acute mechanical low back pain but to try and prevent recurrence of the problem would be highly beneficial.
- 1.4.2 Core stability has been indicated to have an effect on mechanical lower back pain; therefore a relationship between core stability and low back pain must exist (Hodges et al., 1996).
- 1.4.3 Furthermore literature has indicated that sedentary people have a less effective core stability system than athletes, however a baseline for

sedentary participants is not available in the literature from which to make this assertion (Robertson, 2005).

1.4.4 In addition, literature (Hides et al. 1994; Danneels et al., 1994) has indicated that in a large number of cases when participants experience an episode of mechanical low back pain, the multifidus and other core stabiliser muscles may become inhibited and in some cases inactive, and even though there is resolution of that particular episode, the core stabiliser muscles (specifically the multifidus muscle) may remain inactive or in some participants weakened. As a result, it is thought that only by participation in a rehabilitation program that activation (or in this case re-activation) of the multifidus is possible and in association with endurance training and strengthening this may contribute to a lower recurrence rate in mechanical low back pain. (Evans et al., 2000).

1.4.5 A recent study compared manipulation combined with rehabilitation to manipulation alone but could not conclude that this treatment protocol was in fact more effective than manipulation alone (Boden, 2000). This was not possible because core stability rehabilitation as a primary intervention had not yet been established as an effective method of treatment in its own right.

## 1.5 **Benefits**

- 1.5.1 Once the study is complete baseline stabilizer initial readings will be available for the sedentary population, which will be able to determine whether or not they actually do have lower readings those obtained from athletes.
- 1.5.2 Completion of the study will indicate whether core stability training is in fact effective in the treatment of acute mechanical low back pain.
- 1.5.3 Completion of the study will also indicate whether or not manipulation is in fact effective in the treatment of mechanical low back pain.
- 1.5.4 The results of this study will assist in discovering a treatment protocol for mechanical low back pain that may not only treat the current symptoms but also prevent future recurrence of the current problem.
- 1.5.5 Once it can be ascertained that core stability training is / is not effective in the treatment of mechanical low back pain it can be further researched whether in fact a combination treatment of core stability training and manipulation combined is a more effective treatment protocol than manipulation alone.



## 1.6 Limitations

- 1.6.1 Due to time and financial constraints this study only had 32 participants, which may affect the statistical viability of the study. Therefore the results of this study should be viewed as a pilot investigation in order to stimulate future research in this field.
- 1.6.2 Due to limits placed on this research only patients suffering from Lumbar Facet Syndrome and Sacroiliac Syndrome were included therefore there was no indication on results pertaining to any other cause of mechanical low back pain.
- 1.6.3 All precautions were taken to ensure that the patient were in fact using the core stability muscles when performing the prescribed exercises, however there is no mechanism to prevent patients compensating for weak core muscles by using the global muscle system.
- 1.6.4 There was no stratification within the Groups with regards to whether or not the patients had a history of low back pain.

## 1.7 Conclusion

For years people around the world have been seeking a miracle cure to the devastating condition that low back pain can be, and although this is one of the most researched conditions in medical history there is still no clear answer to the problem (Liebenson et al., 1996). There are many treatment protocols that have been shown to be effective and there are many effective protocols that are commonly used but have little or no research to back them, which is why this research is so necessary and beneficial.

The following chapters will cover the literature currently available on this topic, the methodology of this study, the statistical analysis and results and lastly the conclusion and recommendations.

## **CHAPTER 2**

The following chapter reviews the relevant literature perused for this research. Information was gathered from numerous sources including journal articles, published reports, web sites and textbooks as well as anecdotal evidence.

The following aspects were reviewed:

- Anatomy of the lumbar spine and sacroiliac joints,
- Syndromes relating to low back pain,
- Manipulation
- Core Stability Rehabilitation

### **2.1 Introduction to Low Back Pain**

Approximately 80% of the world's population will develop low back pain at some point in their life (Andersson et al., 1991; Anderson et al., 1997; Dionne, 1999), and it is the fifth most common reason for a physician visits (Papageorgiou et al., 1991). In people aged 45-64 years it is the third most common reason for physical impairment (Deyo et al., 1987). Mechanical disorders of the spine are thought to be the cause in about 90% of cases, while the remaining 10% of cases are thought to be caused by systemic illness (Nachemson, 1976). Most episodes of low back pain resolve quickly and are not incapacitating (McKinnon et al. 1997; Diamond et al., 2006). Nevertheless as many as 90 % of participants with acute

back pain return to work within three months, but many experience symptom recurrence and functional limitations. (Patel et al., 2000; Goubert et al., 2004).

In the United States, approximately 50 % of people in the working population have back pain every year (Patel et al., 2000). Even though this ailment usually has a benign course, it is responsible for direct health care expenditure of more than 20 billion dollars annually and as much as 50 billion dollars annually when indirect costs are included (Deyo et al., 1991).

There are many causes of low back pain and although inroads have been made into the diagnosis and treatment of the various causes of low back pain it is agreed that it is an inexact science at best (Weinstein, 1992; Kerr et al., 2001; Feuerstein et al., 2004 ).

## **2.2 Low Back Pain**

For the purpose of this research pain was defined as pain of mechanical origin found in the region denoted anatomically as the low back. Pain is usually cyclical and often referred to the buttocks and thighs, while morning stiffness is commonly associated. Pain is often associated with the start of movement and flexion movements as well as when the patient returns to the erect position. Over the course of the day, pain often becomes worse but is relieved by a change in position. Relief is obtained by lying down especially in the supine position. (Magee, 2002)

In order to more comprehensively investigate low back pain it is important to consider the anatomical structures that are related to the development of low back pain.

### **2.3 Anatomy and Biomechanics of the Lumbar Spine**

The lumbar spine is made up of five individual vertebrae, which are easily distinguished by their massive vertebral bodies, their sturdy lamina and their lack of costal facets. (Moore et al., 1999) They increase in size from L1 to L5 as the weight which must be supported increases, therefore L5 has the largest vertebral body and transversus processes, and due to its unusual shape (it is much deeper anteriorly) it is therefore largely responsible for the sacro-vertebral angle (Grey et al., 1995). Body weight is transferred from L5 to the base of the sacrum, which is formed by the superior surface of the S1 vertebra. The shapes of the articular processes in the lumbar spine allow for the following movements; flexion, extension, lateral flexion but do not allow for rotational movement in the lumbar spine (Moore et al., 1999). Mamillary processes are found on the posterior aspect of the superior articular processes, which form the attachments of the multifidus and medial intertransversus muscles (Leitch et al., 2004).

Secondary cartilaginous joints form the joints of the vertebral bodies, which are connected by intervertebral discs and strengthened by ligaments. While the

intervertebral discs form strong joints they also form the inferior half of the anterior border of the intervertebral foramen, while concomitantly acting as shock absorbers and assisting in forming the curves of the spine (Moore et al. 1999).

### **2.3.1 Facet Joints**

These joints are also known as the zygapophyseal joints, are plane type synovial joints which are made up of the superior and inferior articulating processes of the vertebra above and below (Moore et al., 1999). Each joint has its own loose articular capsule which is attached to the margins of the articular processes (Grey et al., 1995). The shape and orientation of the facet joint allows for a gliding type movement between the surfaces and the amount of movement at each joint is determined by the size of the intervertebral disc at that region. Facet joints bear a certain amount of weight especial in the cervical and lumbar regions.

### **2.3.2 Innervation of the Facet Joints**

Innervation of the joints is mainly by articular branches which arise from the medial branch of the dorsal rami of the spinal nerves. (Moore et al., 1999). The facet joint capsule receives a rich supply of sensory innervation, which is derived from the medial branch of the posterior primary division (dorsal ramus) at the level of the joint. Each joint also receives a branch from the medial branch of the

posterior primary division of the level above and the level below. This multilevel innervation is probably one reason why pain from the facet joint frequently has a very broad referral pattern (Cramer et al., 1995; Jeffries, 1988)

### 2.3.3 Ligaments

Strong ligaments help to stabilize the spine during movement and maintain the curvatures of the spine (Moore et al. 1999). These are summarized in the [Table 2.1](#) below:

**Table 2.1 Ligaments of the Spine**

Ligament	Attachments
Anterior Longitudinal Ligament	Runs down the anterior surface of the spinal column
Posterior Longitudinal Ligament	Runs the length of the posterior aspect of the vertebral column
Interspinous and Supraspinous Ligaments	Runs between and superiorly to the spinous processes
Transversus Ligament	Runs between the transversus processes

**Table adapted from Moore et al., 1999 and Grey et al., 1995**

#### **2.3.4 Muscles of the Back**

Back muscles are generally divided into three categories, the Superficial Muscles, also known as the Extrinsic Muscles, the Intermediate group and the Deep Muscles also known as the Intrinsic Muscles. See Table 2.2 on the following page



<b>Table 2.2 Muscles of the Back</b>					
<b>Muscle</b>	<b>Muscle Subgroup</b>	<b>Origin</b>	<b>Insertion</b>	<b>Innervation</b>	<b>Function</b>
Intrinsic Muscles	These are the true back muscles which act to stabilize the spine, maintain posture and assist with movement of the spine.				
	<b>Superficial Layer of Intrinsic Muscles</b> (Splenius Muscle)	They arise from the midline spinous processes and extend superolaterally to the cervical vertebrae and skull	The Splenius Capitus attaches superolaterally to the mastoid process and the lateral 1/3 of the superior portion of the nuchal line. Splenius cervicis attaches to the posterior tubercles of the C1-C3 transversus processes.	Splenius Capitus- Lateral branches of the dorsal rami of the middle cervical spinal nerves Splenius cervicis- Lateral branches of the dorsal rami of the lower cervical spinal nerves.	When the muscles acts alone they laterally flex and rotate the head to the side of the contracting muscle. When the two muscles act together they extend the head and neck.
	<b>Intermediate Layer of Intrinsic Muscles</b> (Erector Spinae Muscles)	These muscles lie paired on either side of the vertebral column. They are the divided into three columns; the lateral column- <b>Iliocostalis</b> , the Intermediate column- <b>Longissimus</b> , and the Medial column- <b>Spinalis</b> . Each of these three columns is further divided regionally into three, depending on their superior attachments (e.g. iliocostalis lumborum, Iliocostalis thoracis etc.). These three muscles have a common tendon of origin that attaches inferiorly to the posterior part of the iliac crest, the posterior aspect of the sacrum, the sacroiliac ligament and the inferior lumbar and sacral spinous processes.	<ol style="list-style-type: none"> <li>1. Iliocostalis-insertion is to the angle of the lower ribs and cervical transversus processes</li> <li>2. Longissimus-insertion is to the ribs between the tubercles and angles, to the transeverse processes in the thoracic; and cervical regions and to the mastoid process.</li> <li>3. Spinalis-Insertion is to the spinous processes in the upper thoracic area and to the skull.</li> </ol>	Innervation is by the dorsal rami of the lower cervical, thoracic and upper lumbar spinal nerves	<p>Bilateral Activation: extension of the spine and head. During the flexion process the gradually lengthen their fibers to control movement.</p> <p>Unilateral Activation: Lateral flexion of the spine occurs.</p>

Muscle	Muscle Subgroup	Origin	Insertion	Innervation	Function
	<b>Deep Layer of Intrinsic Muscles</b> (Transversospinal Group)	<p>1.Semispinalis-Transversus process of C4-T12</p> <p>2.Multifidus- Arises from the sacrum and ilium, transversus processes of T1-T3 and the articular processes of C4-C7</p> <p>3. Rotatores- Arises from the transversus processes of the vertebrae.</p>	<p>1. Semispinalis- attaches to the occipital bone and spinous processes in both the thoracic and cervical regions usually spanning 4-6 vertebrae</p> <p>2. Multifidus- attaches to spinous processes of the vertebra above usually spanning 2-4 vertebra.</p> <p>3.Rotatores- attach to the lamina and transversus processes of spinous process of the vertebra above, usually spanning only 1-2 vertebra</p>	Innervation is by the dorsal rami of the spinal nerves.	<p>1. Extension of the head, cervical and thoracic regions and rotation of the above regions to the opposite side</p> <p>2. Stabilisation of the spine during local movement</p> <p>3. Stabilise the individual; vertebrae and aid in localized extension and rotation. May also have a proprioceptive function.</p>
Intermediate Muscles	The <b>intermediate muscles</b> are not considered true muscles of the back but muscle of the Thoracic wall. They consist of the Serratus Posterior and the Serratus Posterior Inferior, which are both superficial respiratory muscles although they lie deep to the back muscles	<p>1.Seratus Posterior Superior- Ligamentum nuchae to the C7-T3 spinous processes</p> <p>2.Seratus Posterior Inferior- T11-L2 Spinous processes</p>	<p>1.Seratus Posterior- Ribs 2-4 at the superior border</p> <p>2.Seratus Posterior Inferior-Ribs 8-12 at the inferior border near the angle</p>	<p>1.Seratus Posterior-2<sup>nd</sup>-5<sup>th</sup> intercostals nerves</p> <p>2. Seratus Posterior Inferior- Ventral rami of thoracic spinal nerves 9-11.</p>	<p>1.Seratus Posterior-Elevation of the Ribs</p> <p>2. Serratus Posterior Inferior- Depression of the ribs</p>

<b>Muscle</b>	<b>Muscle subgroup</b>	<b>Origin</b>	<b>Insertion</b>	<b>Innervation</b>	<b>Function</b>
Extrinsic Muscles	The major <b>Extrinsic Muscles</b> consist of the Trapezius, the Latissimus Dorsi, Levator Scapulae, Rhomboid muscles and the Quadratus Lumborum.	1. <u>Trapezius</u> -medial 1/3 of the superior nuchal line, to the external occipital protuberance, the spinous processes from C7-T12	1. <u>Trapezius</u> - posterior lateral 1/3 of the clavicle, the medial acromion, the spine of the scapulae. It also attaches at the occiput via a fibrous lamina from C6-T3	1. <u>Trapezius</u> - Spinal part of the accessory nerve but also contains proprioceptive branches from the ventral rami of C3 and C4	1. <u>Trapezius</u> -Acts with other muscles to steady the shoulder during movement, the upper fibers elevate the scapula and along with serratus anterior rotates the scapula
		2. <u>Latissimus Dorsi</u> -spinous processes of T6-T12, the thoracolumbar fascia, the iliac crest and the inferior 3-4 ribs	2. <u>Latissimus Dorsi</u> -intertubercular groove of the humerus	2. <u>Latissimus Dorsi</u> -Thoracodorsal nerve (C6, C7, C8)	2. <u>Latissimus Dorsi</u> -extends, retracts and rotates the humerus medially
		3. <u>Levator Scapulae</u> -transversus process of C1-C4	3. <u>Levator Scapulae</u> -superomedial border of the scapula	3. <u>Levator Scapulae</u> - Dorsal Scapula Nerve and C3/C4 cervical nerves	3. <u>Levator Scapulae</u> -Elevates the scapula and tilts the glenoid cavity inferiorly
		4. <u>Rhomboid-Major</u> : T2-T5 spinous Processes. <u>Minor</u> : C7-T1 Spinous processes and the nuchal ligament.	4. <u>Rhomboid</u> - medial border of scapula	4. <u>Rhomboid</u> -Dorsal Scapula Nerve	4. <u>Rhomboid</u> -Rotates and retracts the scapula, also fixes the scapula to the thoracic wall
		5. <u>Quadratus Lumborum</u> -Rib 12 at the inferior border and the ends of L1-L5 transversus processes	5. <u>Quadratus Lumborum</u> -Iliolumbar ligament and the internal lip of the iliac crest	5. <u>Quadratus Lumborum</u> L1-L4 spinal nerves and the ventral branches of T12	5. <u>Quadratus Lumborum</u> - Extends and laterally flexes the lumbar spine, it also stabilizes the 12 <sup>th</sup> rib during inspiration.

**Table 2.2 compiled from the following references Moore et al., 1999, Grey et al., 1995, Travelle et al., 1999**

In summary it can be noted that the principle functions of the muscles are related to spinal support, stability and movement. A compromise of one or more of these functions could result in pain and or associated mechanical dysfunction of the spine.

### **2.3.5 The Sacroiliac Joint**

The sacroiliac (SI) joint is a synovial joint located between the articular surfaces of the sacrum and ilium. These surfaces are irregular, with elevations and depressions on their surface, which result in partial interlocking of these bones (Moore et al., 1992).

There are numerous variations in the size, shape and contour of the SI joint. The vertically oriented auricular surface lies obliquely at an angle to the saggital plane (Cassidy et al., 1992). The joints of the male have extra- and intra-articular tubercles and are built for strength, whereas the female articulation is built for mobility and parturition (Walters, 1993).

### **2.3.6 Ligaments**

The SI joint is a weight-bearing joint that is stabilized by a series of very strong ligaments (Cassidy et al., 1992).

**Table 2.3 Ligaments of the Sacroiliac Joint**

Ligament	Attachments
Interosseous Ligament	Unites the iliac and sacral tuberosities and is supported by the posterior SI ligament.
Posterior SI Ligament	Strong, short transversus fibers that join the ilium and first and second tubercles of the lateral crest of the sacrum, Long vertical fibers that the third and fourth transversus tubercles of the sacrum to the posterior iliac spines. (Moore, 1992: 251).
Anterior SI Ligament	thickening of the anterior and inferior parts of the fibrous capsule- thickest where it connects the sacrum and ilium at the third sacral segment

**Table adapted from Moore et al., 1999 and Grey et al. 1995**

The iliolumbar, sacrotuberous and sacrospinous ligaments all support the SI joint but are termed accessory ligaments and the interosseous, posterior and anterior SI ligaments are known as capsular ligaments. (Walters, 1993).

### **2.3.7 Innervation**

The SI joint can be supplied by any of the following levels from L2-S4. The L4 and L5 levels most commonly innervate the anterior aspect of the joint, whilst the posterior aspect more commonly receives its innervation from the S1 and S2 levels (Cassidy et al., 1992).

## **2.4 Neurobiology**

### **2.4.1 Introduction**

The spinal cord is like a “highway” relaying information from all parts of the body to and from the brain, whose job it is to assess the information and react upon it. The central and peripheral nervous systems act in concert to assemble, process and transmit information from all the different areas in order to allow co-ordinated movement (Hopkins et al., 2000). In order for us to better understand the proprioceptive function of many of the core muscles, we need to fully understand the anatomy involved.

### **2.4.2 The Joint**

A large amount of the information required must come from the joint itself, its position, the movement of the joint and the environment. (Levangie et al., 2001) This information is obtained from receptors within the joint, joint capsules, ligament and tendons. (Levangie et al., 2001)

Within all these structures are specialized sensory nerve endings, known as joint receptors which respond to a variety of stimuli, including mechanical, thermal and chemical stimuli. (Hopkins et al., 2000). It is ultimately a stimulation of these receptors that causes a stimulus in the form of an electrical impulse (action potential) to be sent

to the spinal cord and brain via the sensory nerve (Crossman et al., 1995). Two of the important functions that the joint receptors perform are proprioception and mechanoreception, which can be due to either noxious or non-noxious stimuli. (Hopkins et al., 2003). It had been noted that arthrogenic muscle inhibition is mainly due to the stimulation of the mechanoreceptors and to a lesser extent the free nerve endings and specialized nociceptors as well. (Hopkins et al., 2003)

### **2.4.3 Joint Receptors**

Joint receptors are divided into two groups, namely free nerve endings and mechanoreceptors. Free nerve endings are pain receptors (Darby et al., 1995), which have been described as being non-encapsulated, non-specialised and un-myelinated (Hopkins et al., 2000). They appear to play a role in the initial detection of movement. (Hopkins et al., 2000). Nociceptors are also pain receptors, but have been further grouped into the following 3 categories: Mechanical, thermal and polymodal (Kingsley et al., 1996). Each of the three groups is stimulated by a specific stimuli (i.e. The mechanoreceptors are stimulated by a mechanical stimulus, the thermal nociceptors are stimulated by a thermal stimulus). The polymodal nociceptors however can be activated by any of the following stimuli: chemical, thermal or mechanical (Jacobs et al., 1999).



Mechanoreceptors are nociceptors that respond to a mechanical stimulus in the form of pressure and / or distortion (Redwood et al., 1997). Three types of nociceptors have been recognized:

a. Ruffini Endings

Located in the joint capsule these nociceptors are thought to respond to changes in capsular pressure. They are slow adapting receptors with a low-pressure threshold, which allows for prolonged discharge.

b. Pacinian Corpuscles

Located mainly in the periosteum near the articular attachments (Jones et al., 1999). Pacinian corpuscles respond very quickly to stimulation, they are therefore stimulated by any joint movement (Hopkins et al., 2000).

c. Golgi-Like Bodies

Located in the tendons and ligaments surrounding joints (Jones et al., 1999) these nociceptors fire rapidly during the initiation of movement, they then slowly reduce their firing speed to a steady pace. These nociceptors therefore play an important role in joint position sense (Freiwald et al., 1999).

When there is stimulation of the these nociceptors, there is stimulation of the sensory nerve endings which is then transmitted via the spinal afferent nerves to the spinal cord and from there to the central nervous system where the information is assessed and a reflex action is initiated (Moore et al., 1999).

#### 2.4.4 Sensory Nerve Fibers

Sensory nerves are afferent nerves that transmit stimuli from the nociceptors to the spinal cord via the spinal nerves and from there to the central nervous system. There are numerous ways of classifying these afferent nerves but two have been selected for the purpose of the research to assist in explaining the sensory nerve functioning.

- a. Classifications according to Nerve Diameter

**Table 2.5 Classification of Sensory Nerves According to Diameter**

<b>Fiber Type</b>	<b>Fiber Dimension</b>	<b>Myelination</b>	<b>Function</b>
Type A: Beta ( $\beta$ )	5-12	Myelinated	Sensory, touch, pressure and vibration
Delta ( $\delta$ )	2-5	Thinly myelinated	Sharp localized pain, temperature and touch
Type B:	<3	Myelinated	Preganglionic, autonomic
Type C:	0,4-1,2	Unmyelinated	Deep and diffuse pain, temperature, postganglionic autonomic

(Darby et al., 1995; Snell, 1997; Jacobs et al., 1999)

- b. Classifications of nerve fibers according to their origin, function and velocity

**Table 2.6 Classification of Sensory Nerves According to Origin, Function and Velocity**

<b>Fiber Type</b>	<b>Origin</b>	<b>Function/s</b>	<b>Conduction Velocity</b>
Type I	Mechanoreceptors	Proprioception	70-120
Type II	Mechanoreceptors	Cutaneous information from the skin	30-62
Type III	Mechanoreceptors	Deep Pressure, Touch and Temperature	6-30
Type IV	Nociceptors	Crude touch, Pressure, Pain and Temperature	6-16

(Darby et al., 1995; Kingsley, 1996; Guyton et al., 1997)

#### **2.4.5 Sensation Transmission from Afferent Nerve to the Spinal Cord**

After stimulation of the appropriate receptor, the sensation is then transmitted via the appropriate afferent fiber to the dorsal root of the spinal nerve and then to the dorsal horn of the grey matter of the spinal cord where it then passes through the appropriate spinal tract to the central nervous system (Crossman et al., 1995).

#### **2.4.6 Ascending Pathways of the Spinal Cord**

Once a stimulus reaches the spinal cord it must then be transmitted to the central nervous system and this can be done via several different pathways or laminae (Darbey et al., 1995). The spinal cord is divided into 10 laminae, which are numbered from dorsal to ventral within the gray matter (Crossman et al., 1995).

The two main tracts or laminae by which information ascends to the central nervous system are known as the spinothalamic tract and the spinoreticular tract respectively, and both these tracts terminate at the reticular nuclei in the brain stem (Jacobs et al., 1999). The two tracts are thought to convey different sensations to the central nervous system, the spinothalamic tract conveys stimuli related to pain, pressure, thermal sensations and crude touch (Guyton et al., 1997), while the spinoreticular tract conveys pain sensations which are thought to relate to dull aching type pain (Crossman et al., 1995). Information that has traveled through the ascending tracts then moves through the medial lemniscus to the ventropost-lateral nucleus of the thalamus and from there to the cerebral cortex in the central nervous system.

Afferent neurons that ascend via the laminae are usually received by the substantia gelatinosa, which is made up of laminae I-III (Crossman et al., 1995). It is believed that the substantia gelatinosa therefore plays a role in the conveyance of pain sensations. Melzack et al., (1965) used this information to propose the Gate Control Theory.

#### **2.4.7 The Gate Control Theory**

The Gate Control theory was first proposed by Melzack and Wall to explain how pain transmitted through the dorsal horns of the spinal cord showing a gate type effect (Cramer et al., 1995).

Sensory neurons transmitting pain sensation from nociceptors arrive at the dorsal horn of the spinal cord and terminate at the substantia gelatinosa (Cramer et al., 1995). Within the dorsal horn are transmission cells which are neuronal projections that relay the activity in type C fibers via the spinothalamic tract (Cramer et al., 1995), meanwhile the substantia gelatinosa is mainly made up of inhibitory neurons that send generally inhibitory effects to the transmission cells. When there is high activity of the C-fibers this causes an “opening” of the “gate” to pain sensations which are then transmitted to the central nervous system. It is the high activity level in the C-fibers that causes an inhibitory effect of the substantia gelatinosa on the transmission cells thereby allowing the transmission of pain sensations to the central nervous system (Jacobs et al., 1999 and Wood, 1998)

However it must be realized that if the “gate” can be opened then there must be a mechanism by which the “gate” can be shut. The shutting of the “gate” is caused by high activity levels in the A-beta fibers, which facilitate inhibition of the substantia gelatinosa and thereby inhibits the stimulation of the transmission cells causing the pain to cease (Jacobs et al., 1999 and Wood, 1998).

#### **2.4.8 Descending Pathways of the Spinal Cord**

Information is relayed from the Central nervous system via specifically relevant tracts (Hopkins et al., 2000) to the relevant muscle in order to co-ordinate a response. There are three descending pathways, namely the corticospinal tract, the vestibulospinal tract and the rubrospinal tract, which are relevant to this study. These three pathways basically convey information along the specific pathway required, from the central nervous system back to the peripheral system (Hopkins et al., 2000).

The corticospinal tract conveys motor information from the motor and parietal cortices to the motor neurons as well as information about the strength of the muscle contraction (Porter et al., 1997) which is important in voluntary muscle contraction (Darby et al., 1995).

- a. The vestibulospinal tract conveys information from the lateral vestibular nucleus to the motor neurons (Darby et al., 1995). This information is involved in maintaining posture and the tract is activated during postural reflexes (Matkovich, 2004) as well as during the initiation of voluntary movement to cause reflex postural changes (Hopkins et al., 2000).
- b. The rubrospinal tract conveys information from the red nucleus of the midbrain to the distal motor neurons. The rubrospinal tract is involved in the innervation of musculature and has been identified as a source of inhibition for interneurons (Hopkins et al., 2000).

### 2.4.9 Motor Neurons

Motor neurons are essentially the efferent fibers which are the effector neurons in a neural element (Redwood, 1997), their function is to basically innervate a skeletal muscle (Darby et al., 1995). The motor neurons are classified according to their diameter and there are two different types as can be seen in the table below.

**Table 2.6 Classifications of Motor Neurons**

<b>Fiber Type</b>	<b>Fiber Diameter</b>	<b>Myelination</b>	<b>Function</b>
Alpha ( $\alpha$ )	12-20	Heavily Myelinated	Motor
Gamma ( $\gamma$ )	3-6	Myelinated	Motor to the Muscle Spindle

**(Darby et al., 1995; Snell, 1997; Jacobs et al., 2000)**

Each of these different types of motor neuron innervate a specific type muscle or muscle fiber, the alpha motor neurons innervate the extrafusal fibers found in skeletal muscle while the gamma motor neurons innervates the intrafusal fibers of the muscle spindle. The alpha motor neurons are the larger and the faster transmitting of the two types (Iyer et al., 1999).

The innervation of a skeletal muscle is formed when the two types of motor neurons come together to form a neuronal pool or in the case of motor neurons, a motor neuron pool. The muscle and the motor neuron pool work together to form a single efficient unit (Darby et al., 1995) and the strength of the muscle is ultimately determined by the number of motor units working on that muscle to cause a contraction (Iyer et al., 1999).

## **2.5 Arthrogenic Muscle Inhibition**

### **2.5.1 Definition**

Arthrogenic muscle inhibition is a presynaptic, ongoing reflex inhibition of musculature surrounding a joint. It is a natural response following distention or damage to structures in the joint (Hopkins et al., 2000).

### **2.5.2 What is Arthrogenic Muscle Inhibition**

Suter et al., 2000 describes arthrogenic muscle inhibition as being the inability of a muscle to recruit all its motor units during a maximum voluntary contraction (van Dieen et al., 2003), and this can be seen in the clinical setting as decreased strength of the involved muscle (Suter et al., 2000). Arthrogenic muscle inhibition occurs when joint receptors act on inhibitory neurons synapsing with the motor neuron pool and thus affecting the contraction of the muscle (Matkovich, 2004). It has been noted by Ingersoll et al., 2003, that although the free nerve endings and specialized nociceptors may have a role to play with regards to the inhibition, the main effect appears to be as a result of the mechanoreceptor activity.

The clinical importance of this observation is that a decrease in muscle strength may be due to arthrogenic inhibition of the affected muscle (Hurley et al., 1994) and what is of further interest is that the joint receptors are stimulated by pain, the stretching of



ligaments, compression of the joint capsule, effusion or joint injury (Spencer et al., 1984) which could be a possible explanation as to why the core muscles are assumed to be “weakened” in people suffering from low back pain. Suter et al. (1994) found that the decreased muscle strength hampers the rehabilitation process of the injured joint in spite of the fact that there is complete muscle integrity, while Hopkins et al., (2001) found that exercise is an important part of the healing process and can help to prevent a multifaceted injury paradigm.

## **2.6 Syndromes**

Although there are numerous causes of low back pain, biomechanical, anatomical and organic in origin, these are often difficult to identify with any certainty because of the fact that any anatomical structure in the body is capable of causing pain, which often gives rise to the commonly used term of non-specific low back pain (Diamond et al., 2006). It can nevertheless, be helpful to consider back pain under the following headings, as suggested by McRae et al., 2004:

### a) Back Pain due to Clearly Defined Spinal Pathologies

Examples include, but the list is not exclusive to vertebral infections, tumours, ankylosing spondylitis, polyarthritis, Paget’s disease, osteoporotic spinal fractures, senile kyphosis, spondylolisthesis and Scheuermann’s disease.

b) Back Pain Associated with Nerve Root Pain

Examples include, but the list is not exclusive to intervertebral disc prolapse and nerve root compression.

c) Back Pain Caused by a Disturbance of the Mechanics of the Spine

Examples include, but the list is not exclusive to lumbar facet syndrome, sacroiliac syndrome, lumbar disc herniation and myofascial pain and dysfunction syndromes.

It must however be realized that in the majority of cases it is not possible to identify the exact cause of the pain. The noted diagnoses in section c) above, indicate syndromes that could be a cause of low back pain, this is however not an exhaustive list of possibilities. In addition, this is the largest group of conditions and may have formerly attracted names such as “Lumbago” and “Low Back Strain” (McRae, 2004).

As a result, a list of differential diagnoses for low back pain was developed by Deyo et al. 2001 in order to create a more exhaustive reference. This is indicated in Table 2.7.

**Table 2.7 Differential Diagnosis of Low Back Pain**

Mechanical low back or leg pain (90%)	Non-mechanical spinal conditions visceral disease	Visceral Disease
Lumbar strain, sprain Degenerative processes Herniated disc Spinal Stenosis Osteoporotic Compression Fracture Spondylolysis Traumatic Fracture Congenital Disorders Severe kyphosis Severe scoliosis Transitional vertebrae Spondylolysis Internal disc disruption Presumed instability	Neoplasia Multiple myeloma Metastatic carcinoma Lymphoma Spinal Cord Tumours Retroperitoneal tumours Primary Vertebral Tumours Infection Osteomyelitis Septic discitis Paraspinal abscess Epidural abscess Shingles Paget's disease of bone Inflammatory arthritis Ankylosing spondylitis Psoriatic spondylitis Reactive arthritis Inflammatory bowel disease Scheuermann's disease	Disease of pelvic organs Prostatitis Endometriosis Chronic Pelvic Inflammatory Disease Renal Disease Nephrolithiasis Pyelonephritis Perinephritic Abscess Vascular disease Aortic aneurysm Gastrointestinal disease Pancreatitis Cholecystitis Penetrating ulcer

(Adapted from Deyo et al., 2001)

### 2.6.1 Sacroiliac Syndrome

Sacroiliac Syndrome has been described as a subluxation of the sacroiliac joint which results in tightening of the ligaments, muscle spasm of the surrounding muscles and pain (Hendler et al., 1995). Participants often feel pain over the sacroiliac joint which is not relieved by sitting or lying supine, they do often find that they are most comfortable sitting on the unaffected buttock in the forward flexed position (Hendler et al., 1995). Participants may also experience referred pain in the buttock, groin and posterior thigh (Kirkaldy-Willis et al., 1992).

Mechanosensitive afferent units have been identified in the sacroiliac joint and adjacent tissues (Sakamoto et al., 2001). Most of these units are nociceptive receptors (Cassidy et al., 1992; Sakamoto et al., 2001). Hence their ability to stimulate type C fibers, which initiate the opening of the gate in the gate control theory and the perception of pain along with the expected inhibition of the core muscles (Cassidy et al., 1992; Sakamoto et al., 2001).

### **2.6.2 Lumbar Facet Syndrome**

Lumbar Facet Syndrome occurs when there is an episode of trauma which results in posterior joint strain and a small degree of joint subluxation. The muscles surrounding the joint support it by remaining in a state of hypertonic contraction. During this time there is a build up of metabolites in the muscles, further aggravating the pain and sustaining the muscle contraction (Cassidy et al., 1992). Lumbar Facet Syndrome is characterized by pain that is present only on one side, referred pain to the groin, greater trochanter and the posterior thigh. Pain is relieved by rest but aggravated by movement. There is commonly tenderness to pressure, usually on one side and lateral bending is abnormal and there is restriction of motion (Kirkaldy-Willis et al., 1992).

## 2.7 Manipulation

Manipulation has long since been the choice of treatment for Chiropractors (Meeker et al., 2002; Ferreira et al., 2007), it could be argued that it is the adjustment that defines chiropractic as a profession (Bergman et al., 2002). A manipulation or adjustment is a passive manual maneuver 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 et al., 1976). However Mrozek et al. (2005) took this theory one step further by indicating that “the manipulation may not necessarily happen in the para-physiological zone, but might occur at the end range of motion, which in some cases may very well be during the active or passive zones depending where the subluxation is.”

A study undertaken of the efficacy of manipulation and mobilisation in the treatment of low back pain found that it provides better pain outcomes in the short and long term when compared to placebo and other treatments such as McKenzie therapy, back school and other physical therapies and home exercises. (van Tulder et al., 1997; Ferreira et al., 2002; Meeker et al., 2002; Assendelft et al., 2003; Bronfort et al., 2004).

Notwithstanding the above it has nevertheless been noted in clinical practice and research that spinal manipulation has its limitations with respect to improved clinical outcomes (Bronfort et al., 2004). As a result more chiropractors in private practice appear to be moving away from the concept that manipulation alone is the only treatment for low back pain, with anecdotal evidence suggesting that the results obtained from using a combination therapy of both manipulation and rehabilitation in the form of core stability strengthening exercises (Hides et al., 1996; O'Sullivan et al., 1997; Hides et al., 2001; Danneels et al., 2001) is improving the healing time of participants complaining of low back pain.

The success achieved in practice has however not been replicated in clinical research, a recently conducted research study indicated that a combination of manipulation combined with rehabilitation did not in fact improve clinical outcomes (Boden, 2000) but as yet, it has not yet been shown that core stability rehabilitation alone as a primary intervention is effective. The conclusions drawn from this study (Boden, 2000) have inherent limitations, as one cannot conclusively compare outcomes without a baseline measurement with which to compare the results.

Thus the factors that affect lumbar stability have been an area of extensive research (Bergmark, 1989; Jull et al., 2000) in more recent times and the clinical application of lumbar stabilization programs (rehabilitation) have become a common treatment option for participants suffering from low back pain (Descarreaux et al., 2002). Thus it

is also increasingly used by athletes to improve performance and by the general public for optimal health and the prevention of injury. (Barr et al., 2005)

However little research has to date been completed (Akuthota et al., 2004) in this regard to quantify this anecdotal data.

## **2.8 Core Stability**

The inability to show a clinical response or a synergistic clinical response as found in the study by Boden, (2000) may be related to the fact that clinically noted that wasting and inhibition of the core stabiliser and co-contractor, multifidus, was present in participants with low back pain (Danneels et al., 1994; Hides et al. 1994). These findings re-enforce the findings of previous studies that have shown that the contraction of the transversus abdominus (TA) is significantly delayed in participants suffering from low back pain, indicating a deficit in the motor control of the TA muscle (Creswell et al., 1992, Creswell et al., 1994; Hodges et al. 1996; Hides et al., 1996). It has been hypothesized to result in ineffective core stability (Hodges et al. 1996(a)). In support of this, a study of the transversus abdominus found that low back pain participants had reduced endurance (Hultman et al. 1993) and that its protective ability was decreased (Evans et al., 2000).

This supported research that found that dysfunction of the primary core stabiliser muscles is linked with an increasing number of the general population suffering from low back pain (Hodges et al., 1996b).

Although the cause of this finding by Hodges et al. (1996b) is yet unknown, it has been hypothesized that over recent years there has been a steady decline in the amount of physical activity in the general population (Jorgensen, 1997). This is thought to be mainly due to our fast paced lives, which has led to a steady decline in our once strong core muscle system (Jorgensen, 1997) which consists of the Transversus Abdominus, the Multifidus, the Pelvic floor muscles and the Diaphragm. See Table 2.8.

The unfortunate implication of this is that those muscles which were once our strong postural muscles are now weak and ineffective, therefore making us more prone to both spinal injuries and low back pain (Hodges et al., 1996b, van Tulder et al., 2002).



Table 2.8 Core Muscles

<b>Muscle</b>	<b>Origin</b>	<b>Insertion</b>	<b>Innervation</b>	<b>Main Function</b>
Transversus Abdominus	Thoracolumbar Fascia, the anterior 2/3 of the iliac crest and the lateral half of the inguinal ligament	The inferior border of ribs 10-12, the linea alba and the pectin pubis via the conjoint tendon	The Ventral rami of the inferior 6 thoracic nerves and the 1 <sup>st</sup> lumbar nerve	Compresses and Supports the abdominal viscera
Multifidus	Arises from the sacrum and ilium, transversus processes of T1-T3 and the articular processes of C4-C7	Attaches to spinous processes of the vertebra above usually spanning 2-4 vertebrae.	Dorsal Rami of the Spinal Nerves	Stabilise the individual; vertebrae and aid in localized extension and rotation. May also have a proprioceptive function
<b>Pelvic Floor Muscles</b>				
Levator Ani (Pubococcygeus, Puborectalis and Iliococcygeus)	Attaches from the body of the pubis, the arch of the obturator fasci and the ischial spine	Attach distally to the perineal body, the coccyx, the annococcygeal ligament, the wall of the prostate or vagina, the rectum and the anal canal	Nerve to levator ani, branches of S4, inferior anal nerve and the coccygeal ligament.	Assists in supporting of the pelvic viscera. These muscles also resist an increase in intra-abdominal pressure
Coccygeus	Ischial Spine	Inferior end of the Sacrum	Branches of S4 and S5 nerves	Assists in supporting pelvic viscera and flexes the coccyx
Diaphragm	Inferior margin of the thoracic cage bilaterally. And the superior lumbar vertebrae.	Fibers converge radially on the trifoliate central aponeurotic part- the central tendon. The central tendon has no bony attachments and is divided into 3 leaves.	Motor Supply: Phrenic Nerve (C3-C5) Sensory Supply: centrally-phrenic nerve Peripherally- intercostal nerves (T5-T11) & Subcostal nerves (T12)	Main muscle of inspiration and acts to separate the thorax from the abdominal cavity.

(Adapted from Moore et al., 1999 and Grey et al., 1995)

Thus, by implication, it would seem that these important stabilizers (mentioned above) have been implicated in initiating a new trend in rehabilitation of participants who are suffering from low back pain and this is *core stability strengthening* (Hodges et al., 1996; Marshall et al., 2005)

This term core rehabilitation has come to denote lumbar stabilisation and other therapeutic exercise schemes, which have an influence on the core muscles, which stabilise the lumbar spine (Akuthota et al., 2004; Ericksen et al., 2006). The core muscles have been identified as so important because they act as a “corset” to stabilise the lumbar spine. As a result a comprehensive strengthening or facilitation of these “corset muscles” has been advocated as a way to prevent (Linton et al., 2001; van Tulder et al., 2002) and rehabilitate various lumbar spine and musculoskeletal disorders (Lahad et al., 2001; Akuthota et al., 2004). The clinical outcomes of core-strengthening programs have not been well researched and studies have been hampered by the lack of clarity (Frank et al., 1996; van Poppel et al., 1997; Lahad et al., 2001) as well as the lack of consensus of exactly what constitutes a core-strengthening program (Akuthota et al., 2004).

The research is further complicated by the theoretical possibility that core stability strengthening as a rehabilitative exercise will not assist in the treatment of low back pain if the participants are already in pain. The theoretical argument arises from asking participants to perform exercises that may cause a worsening of their clinical symptomatology (e.g. pain) because they are already in pain and they think that by

exercising that they will cause a worsening of their pain (Klenerman et al., 1995; Linton et al., 1998; Linton et al., 2000; Bauer et al., 2002; Picavet et al., 2002). In opposition to this, the gate control theory indicates that increased activity would allow for a “closing of the gate” by the alpha fiber stimulation thereby lessening the pain (Murphy et al., 1995).

This latter argument has been supported by recent evidence (Klenerman et al., 1995; Linton et al., 1998; Linton et al., 2000; Bauer et al., 2002; Picavet et al., 2002) in this complex question by investigating the pain that was anticipated before and induced by physical activity. The conclusion of the study was that anticipated and induced pain with physical activity was lessened after physical therapy using exercise in participants with low back pain (Rainville et al., 2004). Thus it would seem that even in light of research opposing the use of core stability strengthening; it nevertheless has benefits in people who have low back pain (Rainville et al., 2004; Cherkin et al., 2005)

It stands to reason that assisting in the reduction of pain prior to the application of the core stability strengthening exercises could enhance their clinical effect. In this regard manipulation is often seen as the intervention of choice in assisting pain reduction (Kirkaldy-Willis et al., 1992) and hence the combination use of these modalities often occurs.

Therefore, this research aims to investigate the relative effect of manipulation and core rehabilitation in the treatment of acute mechanical low back pain in sedentary

participants in order to ascertain the efficacy of core rehabilitation as a treatment for low back pain.

## **CHAPTER 3**

### **3.1 Introduction**

This chapter will cover the methods used in this research to collect data and the statistical methodology used in data interpretation

### **3.2 Design:**

The study design was be a randomised controlled parallel group trial.

The study Durban University of Technology Faculty of Health Sciences Research Committee approved this study, and the Ethics Review Board declared that the research conformed to the Standards set by the Helsinki Declaration of 1975.

### **3.3 Advertising and Patient Recruitment**

Flyers were placed at the Durban University of Technology, local community areas such as libraries, shopping centers and in the local neighborhood.

Advertisements (Appendix 1) were placed in local newspapers to target the population of the greater Durban area, while pamphlet drops (Appendix 1) were done at the following post offices-Berea, Overport, Chatsworth, North Beach, Avondale, Verulam, Phoenix and Durban Central.

### 3.4 Sample

**3.4.1 Method:** Thirty-two participants were divided into two groups (A and B), each group had a minimum of fifteen participants who had to have acute mechanical low back pain.

**3.4.2 Sample size:** A minimum of thirty-two participants were selected as per the inclusion and exclusion criteria (under section 3.4.5 and 3.4.6). Thus the sampling was based on a predetermined randomization list (Appendix 2) such that patients entering the study were allocated based on their initial booking at the clinic.

**3.4.3 Sample Allocation:** Two groups, each with a minimum of sixteen participants. Participants were randomly allocated into either group A or group B. Participants were allocated to each group using a computer generated randomisation table. [Appendix 2] (Esterhuizen, 2007)

**3.4.4 Sample Characteristics:** Participants initially participated in a telephonic interview where they were asked their age, how long they had had their pain and to briefly describe their pain in terms of cause, history of injury, occupation and they were also asked about their exercise habits. Once it had been determined that the patients fitted all criteria for the research, namely inclusion and exclusion criteria (3.4.5 and 3.4.6), they were then invited to make an appointment at the Durban University of Technology Chiropractic Day Clinic where further evaluation then took place to ensure that all research criteria were met for each patient. At the initial

consultation patients were asked to sign the informed consent (Appendix 3) form after having an opportunity to read the letter of information (Appendix 4) provided, discuss the research and ask questions. A diagnosis was then made based on a case history (Appendix 5), physical examination (Appendix 6), relevant lumbar spine regional (Appendix 7) examination and soap note (Appendix 8) for the following inclusion and exclusion criteria:

### **3.4.5 Inclusion**

- 3.4.5.1 Participants had to be between the ages of 18 and 45, to ensure that participants were skeletally mature. To avoid and reduce the chance of sacroiliac and / or spinal ankylosis participants could not be older than 45 years of age (Kirkaldy-Willis et al., 1992).
- 3.4.5.2 Participant's pain rating on the NRS had to be greater than 5 and less than 8 (Fejer et al., 2005), to improve the sample homogeneity (Mouton, 1996).
- 3.4.5.3 All participants had to have acute (Van Tulder et al., 2002) low back pain with an onset of 7 days or less to avoid the natural history being reflected in the results of this study, thereby nullifying any possible outcome. i.e. most individuals suffering from low back pain heal within 6 weeks, however 5-15% are unresponsive to treatment and have continued disability i.e. chronic low back pain (Liebenson, 1996).

- 3.4.5.4 Participants had to signed informed consent forms (Appendix 3).
- 3.4.5.5 For the purpose of this study sedentary individuals were defined as those who undertake no leisure time physical activities and those who undertook less than 30 minutes of physical activity each day.  
(President's Council on Physical Fitness and Sports Research, 2002)
- 3.4.5.6 Participants had to be suffering from mechanical low back pain, which included both posterior facet syndrome in the lumbar spine (Kirkaldy-Willis et al., 1992:203) and or sacro-iliac syndrome (Cox, 1998).

Signs and symptoms of posterior facet syndrome included (Plaughter, 1993, Kirkaldy-Willis et al., 1992)

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

For the purpose of this study research participants needed to present with 3 out of 6 of the above symptoms. Orthopaedic tests are not considered as part of the diagnostic criteria for posterior facet syndrome; however, it is possible that they can be used to assist in the confirmation of the



diagnosis. In order to be included in this study participants had to have a minimum of two out of the four tests listed below being positive (Kirkaldy-Willis et al., 1992).

- a) Kemp's test (Gatterman, 1982).
- b) Facet joint challenge (Bergman, 1993),
- c) Palpatory, tenderness (Magee, 1992), and
- d) Spinous percussion (Bergman, 1993)

Signs and symptoms of sacro-iliac syndrome (McCulloch et al. 1997).

- Pain over the Sacroiliac joint.
- Sacroiliac joint was locally tender to palpation.
- Referred pain was to the buttocks, posterior thigh, groin and occasionally lateral calf and ankle.
- The pain was aggravated by provocation tests.
- There was clinical evidence of increased movement or asymmetry of the Sacroiliac joint.
- There was no other apparent cause of the participant's Sacroiliac joint pain localization.
- It may have mimicked a herniated disc or lateral spinal stenosis.

- A lack of nerve root tension signs and absence of motor, reflex or sensory deficits help to distinguish Sacroiliac syndrome from nerve root entrapment syndromes.

For this research, participants needed to present with 4 out of 6 symptoms as above. Orthopaedic tests are not part of the diagnostic criteria for sacroiliac syndrome; however, they can be used to confirm the diagnosis. For the purpose of this research two out of the four tests described below had to have been positive (Kirkaldy-Willis et al., 1992).

- a) Gaenslen's test (Kirkaldy-Willis et al., 1992).
- b) Patrick Faber test (Kirkaldy-Willis et al., 1992).
- c) Erichsen's test (Schafer et al. 1989)
- d) Lateral recumbent Sacroiliac compression test (Schafer et al., 1989)

### **3.4.6 Exclusion:**

3.4.6.1 Participants who presented with signs and symptoms of posterior facet syndrome and/or sacro-iliac syndrome were not allowed to have had any of the following signs with respect to nerve root entrapment pathology, for example: (Plaugher, 1993, Kirkaldy-Willis et al., 1992).

- Presence of paraesthesias

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

3.4.6.2 Patients who presented with any of the following contraindications to spinal manipulation, which could include but was not limited to these, were excluded from the study (Bergmann et al., 2002, Kirkaldy-Willis et al., 1992).

**Relative:**

- Osteopenia,
- Spondyloarthropathies,
- Participant on anticoagulant medication,
- Bleeding Disorders and / or
- 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 and / or
- Spinal fusion or spinal surgery.

3.4.6.3 Participants who presented with any of the following contraindications to abdominal muscle strengthening, which could include but was not limited to: Glaucoma, hypertension, osteoporosis, spinal tumors, inflammatory diseases and impaired circulation, were excluded from the study (Harms-Ringhdal, 1993).

3.4.6.4 Participants who experienced extreme discomfort on contraction of the Transversus Abdominus Muscle were excluded due to the fact that they would be unable to perform the necessary pre and post experimental tests.

3.4.6.5 Participants who were currently 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). For the period of participation in this study, participants were not permitted to partake of any pain or anti-inflammatory medication of any sort including homeopathic remedies to prevent

signs and symptoms being masked by medication, resulting in them being excluded from the study.

- 3.4.6.6 Participants who had low back surgery were excluded from this study as the source of their pain may have been related to the surgery and or post surgical complications. Furthermore, those participants who had had any recent abdominal surgery that may have referred pain to the back were also excluded as they may also have experienced difficulty in contracting the abdominal muscles.
- 3.4.6.7 Participants who required further clinical testing to confirm the diagnosis were excluded.
- 3.4.6.8 All participants who failed to complete the informed consent form.
- 3.4.6.9 Participants who had suffered any recent trauma involving the spine, as this could be a contraindication to manipulation.
- 3.4.6.10 Any female participants who were pregnant or who had given birth in the last 6 months, to negate the effects of the hormone Elastin produced during pregnancy.

Those participants who were 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 who were excluded i.e. those who did not meet the inclusion criteria were referred to other interns or student interns in the Durban University of Technology Chiropractic Day Clinic for treatment of their condition.

### **3.5 Intervention Method and Frequency – Group A**

Group A received treatment twice a week for two weeks (in the form of Core Stability Rehabilitation) as well as a follow up consultation seven days later (Chok et al., 1999). At consultation one, three and five readings [NRS (Appendix 9), Stabiliser, Algometer (Appendix 10) and EMG] were taken. In total there were five visits over a period of three weeks and three sets of readings were taken during this period, participant were presented with a series of rehabilitation exercises at their initial consultation and were taught these exercises as well as being taught how to contract their TA muscle. The participants then took those exercises home and performed them daily (Richardson et al., 1995) as was prescribed and they filled in a participant diary (Appendix 11) to confirm participant compliance. At each treatment the researcher then ensured that the exercises were being performed in an appropriate manner (i.e. as prescribed by the research methodology (Appendix 12) by having the participant perform the exercises during the treatment session.

### **3.6 Rehabilitation**

The rehabilitation portion of the treatment consisted of four core stability exercises (Appendix 12) which were taught to the participant at the initial consultation and they were then expected to perform these exercises at home

(Richardson et al., 1995) and sign the attached exercise diary (Appendix 11) confirming that they had actually done the given exercises.

Rehabilitation exercises included hyperextension exercises to strengthen para-vertebral muscles, mobilising exercises to improve overall spinal mobility, and isometric flexion exercises designed to strengthen abdominal lumbar muscles while protecting the back from excessive motion (Saal et al., 1989; Kellett et al., 1991; Hansen et al., 1993; Risch et al., 1993). All rehabilitation exercises were performed while the abdominal muscles remained contracted. Such exercises are thought to reduce the frequency of recurrent back pain and, in acute episodes, to reduce pain by decreasing intradiscal pressure (Weinstein et al., 1992).

### **3.7 Intervention Method and Frequency – Group B**

Group B received a spinal adjustment on the fixated lumbar segment or sacroiliac joint in the form of a lumbar roll as their treatment protocol, depending on what was found during the motion palpation twice weekly for two weeks (Mathews, 1995) and a follow up consultation seven days later at which the final set of readings was taken. Readings (NRS, Stabiliser and Algometer and EMG) were taken at treatments one, three and five. There was however no rehabilitation exercises for these participants.

### **3.8 Education**

During the initial consultation group A and B were educated on how to contract their transversus abdominal muscle by using the four point kneeling position test (Appendix 13) (Richardson et al., 1999; Richardson et al., 1995).

### **3.9 Measurement Protocol**

At the beginning of consultations one, three and five readings were taken using the biofeedback stabiliser. In order to do this, participants performed an abdominal draw in test (Jull et al., 1995; Urquhart et al., 2005)) using the biofeedback stabiliser. (Stabiliser manual Chatanooga Group Inc., 4717 Adams Road, Hixson TN 37343, USA) This was then used to measure the participant's transverse abdominal muscle strength and endurance and indicate whether the participant can activate the muscle.

Endurance was measured (Mannion, 1999) by timing how long they could hold their abdominal contraction (Richardson et al., 1999). While this test was being performed the surface EMG was used to determine the role of the global muscles in the participant's ability to contract the core muscles and this ensured that it was in fact the core muscles that were being activated rather than merely being compensated for by the global muscle system (Silfies et al., 2004). The surface



EMG Reading (Lariviere et al. 2007) was noted at all three reading points along with the stabiliser, NRS and Algometer readings.

### **3.10 Intervention Frequency**

Five visits over a period of three weeks including measurements. (Bolton et al., 1997).

### **3.11 Measurement Tools**

**Subjective data** was obtained from the following:

3.11.1 **Numerical Pain Rating Scale** is an effective and reliable tool to evaluate if pain is reduced with treatment and to what degree. (Bolton et al., 1998). Participants were asked at visit one, three and five to rate their own pain levels from zero to ten, with ten being the worst pain they had ever experienced and zero being no pain at all. This number was then noted on the Numerical Pain rating scale. (Appendix 9).

3.11.2 **Low Back Pain and Disability Questionnaire**, which is a sensitive measure of the disability of low back pain (Morris, 1983). Participants were asked to answer the questions on the questionnaire as they applied to their daily lives and answer with a

tick or cross to indicate yes or no as to whether or not the question was relevant to them. The Positive answers (ticks) were then added up to give a score out of 24, which was then noted on the questionnaire. (Appendix 14)

**Objective feedback** was obtained through the use of:

3.11.3 **Endurance Testing** of transverse abdominus muscle using the stabilizer biofeedback device (Cairns, 2000). This was done utilising the prone test for transversus abdominus and the supine position for training transverse abdominus to assess fatigability / endurance of the transversus abdominus (Stabiliser manual Chatanooga Group Inc., 4717 Adams Road, Hixson TN 37343, USA). (Appendix 10)

Endurance testing was achieved by timing the length of the contraction using a stopwatch. This was done three times and an average score was determined.

3.11.5 **An Algometer** (the force dial algometer to assess the tenderness of the affected joints). The algometer was used to quantify response to treatment such as an adjustment and provides a means of measuring (Fischer, 1986).The algometer readings were

taken over the most painful area of the symptomatic sacroiliac or lumbar facet joint. The participant was then requested to indicate the point of pain or discomfort by saying 'now', and the reading was taken at that point. (Appendix 10)

3.11.6 **Surface EMG** was used (Richardson et al., 1999) to measure the involvement and role of the global muscles during core contraction to ensure that the participant was learning to activate the core muscles and not compensate by using global muscles (Lariviere et al. 2007).

The surface EMG was performed while 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 levels of L1 and L4, approximately 2cm from the spinous process. (Appendix 10)

### **3.12 Data Collection Frequency**

Data was collected at three points in the five visit period. At visits one, three and five objective readings were taken using the Algometer, Biofeedback Stabiliser and Surface EMG. Subjective data was obtained at the same visits in the form of the Roland Morris Low Back Pain and Disability Questionnaire and NRS scale.

### 3.13 Statistics

Formal sample size calculations were not performed, since it was impossible to state *a priori* what the smallest difference of clinical/biological importance would be. Thus for the scope of the study, financial and time constraints, it was decided that thirty-two participants would be used. Participants were randomised into two equal groups of sixteen each using computer generated random number tables. One group received the standard treatment (manipulation alone) while the second group received rehabilitation. Outcomes were measured at three time points, baseline (before treatment), at treatment three (before the treatment) and after follow up (after visit five).

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**

### **4.1 Introduction**

The following chapter will look at the results of the Statistical Analysis performed and will discuss these results. Data was obtained from the participants of the research at visit one, three and five using both subjective and objective measures.

### **4.2 The Data**

#### **4.2.1 Primary Data**

##### **a) Subjective Measurements**

- Roland Morris Questionnaire
- Numerical Pain Rating Scale

##### **b) Objective Measurements**

- Algometer
- Biofeedback Stabilise
- Stabiliser Endurance Reading
- Surface EMG
- Abdominal Biofeedback Stabiliser

#### 4.2.2 Secondary Data

Secondary Data was obtained from numerous sources including, libraries, the internet, books, journal articles, lecture notes and anecdotally.

#### 4.2.3 Terminology

- **P-value** – this value is expressed as  $p < 0.05$  which indicates statistical significance (Baily, 1997). This value allows the researcher to decide whether the changes seen in the research are due to the treatment effect or due to chance (Stagg, 2006)
- **N** – Sample size.
- **Mean** – this indicates the average obtained when the sum of the observations are added together and divided to find the average.
- **Median** – This value indicates the center of the distribution. According to Bland, 1996 it would seem that half the readings must be less than or equal to the value and half the readings must be greater than or equal to the mean.
- **Standard Deviation** – this is the variance from the readings from the mean (Stagg, 2006). Should the readings vary greatly then it could be said that there is a large standard deviation, likewise if the readings are close to the mean, there would be a narrow standard deviation (Campbell et al. 1999)
- **Treatment Effect** – An effect attributable to the test treatment (Esterhuizen, 2007)

### **4.3 Statistical Methodology**

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.

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.

### **4.4 Results**

#### **4.4.1 Demographics**

Thirty-two participants were randomized into two equal treatment groups. Although the methodology indicated that a minimum of fifteen participants per group were required to conduct the study, sixteen participants were ultimately used in each group as there was no set upward limit. Their mean age was 33.3 years, with a standard deviation of 6.8 years and a range of 23 to 45 years, although it was noted that the mean age in the core group was younger than that of the treatment group. The sample had a diverse occupational profile, shown in Table 4.1. The majority of participants were professionals (28%), consisting of mainly accountants, followed by operators, specifically factory workers (22%).



**Table 4.1: Occupational classification of sample**

	Frequency	Percent
Business	1	3.1
Professional	9	28.1
Technical	2	6.3
Administration	4	12.5
Sales	4	12.5
Artisan	2	6.3
Student	2	6.3
Operator	7	21.9
Housewife	1	3.1
Total	32	100.0

There was no significant difference between the two groups in terms of gender ( $p=0.280$ ), although the core group contained a higher proportion on males than the adjustment group. The gender breakdown of each group is shown in Table 4.2.

**Table 4.2: Gender by Treatment Group**

			Gender		Total
			Male	Female	
Group	Adjustment	Count	8	8	16
		Row %	50.0%	50.0%	100.0%
	Core	Count	11	5	16
		Row %	68.8%	31.3%	100.0%
Total		Count	19	13	32
		Row %	59.4%	40.6%	100.0%

Pearson's chi square 1.166,  $p=0.280$

There was no significant difference ( $p=0.280$ ) in age between the two treatment groups ( $p=0.171$ ), this is shown in Table 4.3.

**Table 4.3: Comparison of Mean Age Between the Treatment Groups**

	Group	N	Mean	Std. Deviation	Std. Error Mean	p- value
Age	Adjustment	16	34.94	7.353	1.838	0.171
	Core	16	31.63	5.943	1.486	

Table 4.4 shows that the two groups were similar in terms of height, weight and BMI. There were no significant differences ( $p=0.171$ ) between the groups, and the two group means were very similar.

**Table 4.4: Comparison of Mean Height, Weight and BMI between the Treatment Groups**

	Group	n	Mean	Std. Deviation	Std. Error Mean	p-value
Height(M)	Adjustment	16	1.689	0.0790	0.0198	0.932
	Core	16	1.686	0.1216	0.0304	
Weight	Adjustment	16	68.09	8.707	2.177	0.775
	Core	16	69.22	12.909	3.227	
BMI	Adjustment	16	23.9187	3.00020	0.75005	0.699
	Core	16	24.4264	4.24907	1.06227	

#### **4.4.2 Comparison of Baseline Outcomes Between Treatment Groups**

None of the outcome measurements (Roland Morris Questionnaire, Algometer, Biofeedback Stabiliser and the Surface EMG) showed statistically significant differences between treatment groups prior to the intervention. Thus any improvement that is observed should be due to the intervention as the groups started off at the same baseline.

Table 4.5 shows that only Abdominal Reading 1 before approached a statistically significant difference ( $p=0.069$ ), but the absolute mean difference was small. Although there was almost statistical significance, this does not necessarily

indicate that there is a clinical significance. These values indicate that at baseline the two groups were comparable, although practically it was noticed by the researcher during the duration of the research process that clinical improvement may be influenced by the attitude of the patient (Eccleston et al., 1997), however there is little research to validate this theory.

**Table 4.5: Comparison of Baseline Outcome Measurements Between Treatment Groups**

	Group	N	Mean	Std. Deviation	Std. Error Mean	p- Value
Roland Morris Questionnaire	Adjustment	16	5.31	5.338	1.335	0.465
	Core	16	4.13	3.575	0.894	
Algometer Reading 1	Adjustment	16	4.584	1.2760	0.3190	0.775
	Core	16	4.741	1.7486	0.4372	
Stabiliser Reading 1	Adjustment	16	-3.79	2.437	0.609	0.355
	Core	16	-4.81	3.594	0.899	
Stabiliser Time 1	Adjustment	16	12.436	5.8831	1.4708	0.851
	Core	16	12.781	4.2548	1.0637	
EMG reading 1 at L1 Before	Adjustment	16	3.438	0.8578	0.2145	0.295
	Core	16	3.881	1.4256	0.3564	
EMG reading 1 at L4 before	Adjustment	16	8.175	1.9536	0.4884	0.347
	Core	16	9.200	3.8224	0.9556	
EMG Abdominal Reading 1	Adjustment	16	3.09	0.203	0.051	0.069
	Core	16	3.02	0.040	0.010	
Abdominal Stabiliser reading 1	Adjustment	16	-3.44	2.366	0.591	0.303
	Core	16	-4.63	3.862	0.966	

## 4.5 Assessment of the Treatment Effect

### 4.5.1 Subjective Outcomes

#### a) **Roland Morris Questionnaire**

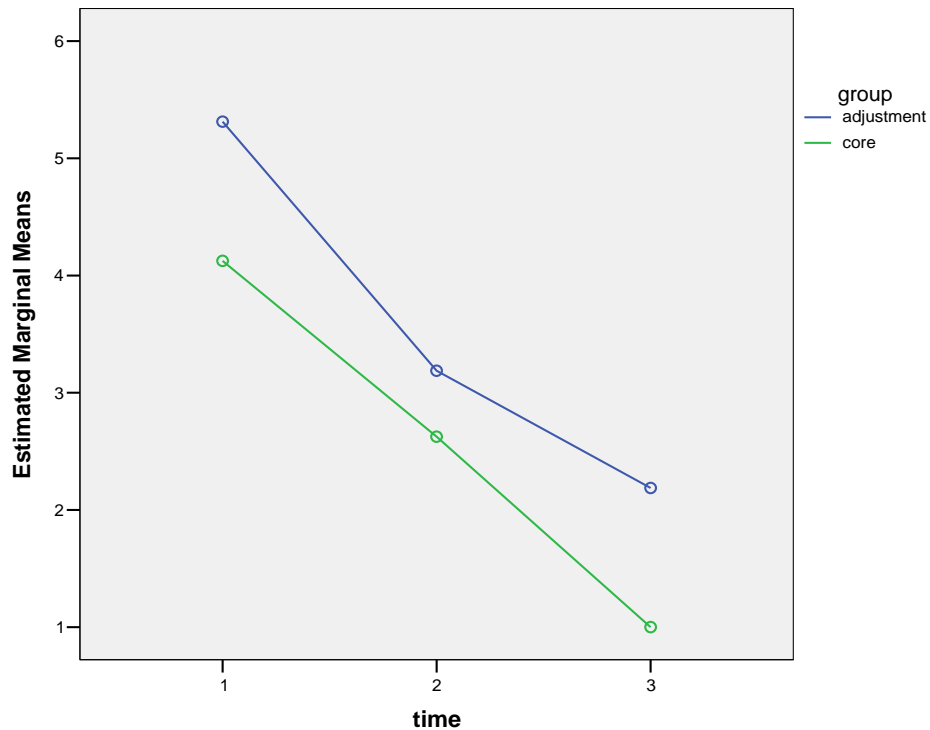
There was no significant treatment effect according to the Roland Morris Questionnaire outcome ( $p=0.394$ ). Thus both groups showed similar progress over time. The overall time effect was highly significant ( $p<0.001$ ), and Figure 4.1 shows that both groups showed a sharp decrease in Roland Morris Questionnaire score over time. The rate of decrease was similar, thus for Roland Morris Questionnaire there was no evidence that the treatments showed differential effects.

### Discussion

The results shown by statistical analysis of Roland Morris Questionnaire show that, in both treatment groups there was an improvement in daily functionality for the participants. The graph indicated that the Adjustment Group showed a steady decrease in score, however after time 2 there is a slight leveling off in the score whereas the Core Group showed a steady decrease in score throughout. In a previously conducted study similar to this, it was found that Core Rehabilitation had no or little effects as a treatment (Boden, 2002), but this is not supported by these results.

**Table 4.6: Within and Between Subjects Effects for RMQ**

Effect	Statistic	p- value
Time	Wilk's Lambda=0.535	<0.001
Group	F=0.791	0.381
Time*group	Wilk's Lambda=0.938	0.394



**Figure 4.1: Profile Plot of Time by Group for Roland Morris Questionnaire**

**b) Numerical Pain Rating Scale**

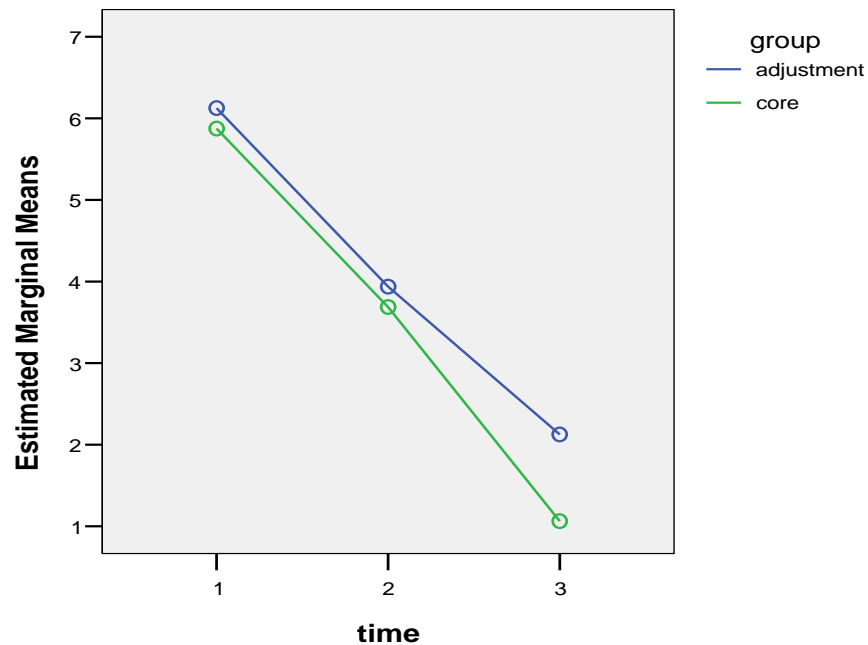
Similarly for NRS, there was a significant time effect overall ( $p < 0.001$ ), but no evidence of a differential treatment effect ( $p = 0.430$ ). Figure 4.2 shows that the rate of improvement in pain according to the NRS score was very similar in both groups. It is the time effect that is significant in this case because it shows that both Groups improve over time. Again as seen in the Roland Morris Questionnaire, the Core Group appears to improve slightly faster after time two than the Adjustment Group, which supports findings that Core Rehabilitation appears to be as effective as Manipulation. As noted in Chapter 3 reading one was taken at visit one, reading two was taken at visit three and reading three was taken at visit five.

## **Discussion**

Statistical analyses of the data obtained from the NRS readings showed that in both treatment groups there was a steady decrease in the NRS readings throughout the treatment period (Carpes et al., 2007), however it can be noted from Figure 4.2 that there was a slightly greater decrease in the Core Groups readings after time 2 when compared to the Adjustment Group. It must be noted that both of the above measurements are subjective and once again can be influenced by the patient's attitude to their back pain, and although it is not shown in current literature it could be possible that patients receiving Core Rehabilitation portray a better attitude towards their pain and thus influence the subjective findings.

**Table 4.7: Within and Between Subjects Effects for NRS**

Effect	Statistic	P value
Time	Wilk's Lambda=0.097	<0.001
Group	F=1.502	0.230
Time*group	Wilk's Lambda=0.944	0.430



**Figure 4.2: Profile plot of Time by Group for NRS**

#### **4.5.2 Objective Outcomes**

##### **a) Algometer**

There was no evidence of a significant effect for algometer as indicated by the p-value ( $p=0.280$ ). Both groups showed a significant mean increase over time ( $p<0.001$ ). Figure 4.3 shows that the adjustment group increased at a slightly faster rate than the core group between time 2 and 3, but this difference was not statistically significant. This is not a true cross over effect because it is expected that as a participant improves clinically, their Algometer readings will increase. However in this case it is that rate of improvement that causes the cross over effect, because the Manipulation Group's reading increased more than the Core Group's.

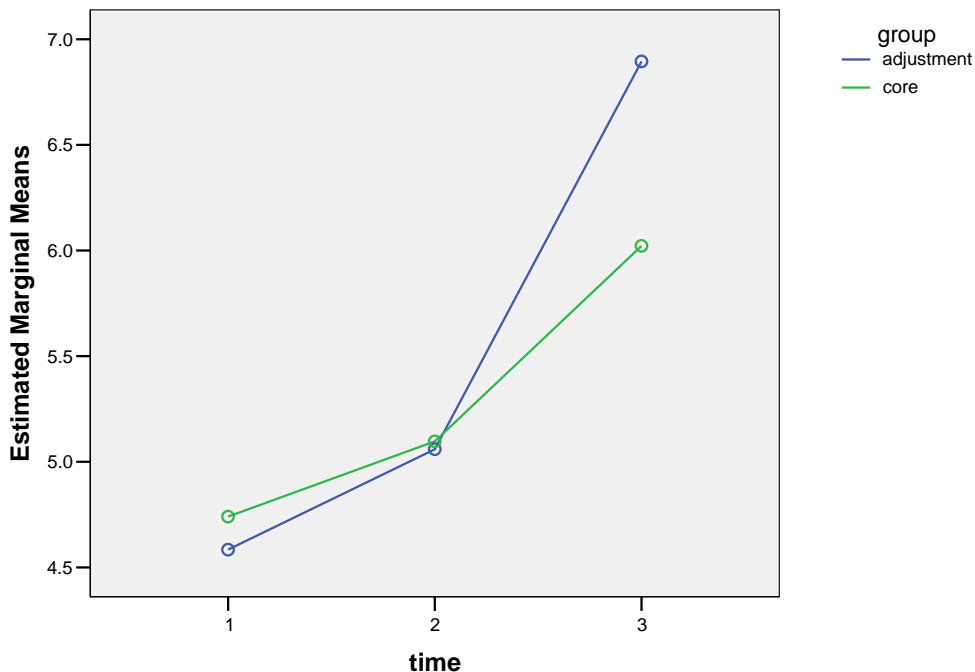
## **Discussion**

The above results could possibly be explained by the fact that the Algometer Readings were taken over the most painful Lumbar Facet Joint or Sacroiliac joint. Although technically the Algometer was designed to take a pressure reading over soft tissue, it must be realized that over every joint there is a certain amount of soft tissue. Over the Sacroiliac Joints there are Gluteal Muscles and over the Facet Joints there are erector Spinae muscles. Thus the Adjustment Group may have had either of those painful joints adjusted, thereby decreasing the subluxation and the muscle spasm which may be in the overlying area. and causing a decrease in pain in that area (Herzog et al. 1995; Lehman et al., 2001; Pickar, 2002). The Core Group would have had no manipulation of the joints which may have had an impact on their pain perception over those joints as well as the fact that unaccustomed use of these muscles may have left the participant experiencing delayed onset muscle soreness, also influencing their perception of pain.

**Table 4.8: Within and Between Subjects Effects for Algometer**

Effect	Statistic	p-value
Time	Wilk's Lambda=0.520	<0.001
Group	F=0.354	0.556
Time*group	Wilk's Lambda=0.916	0.280





**Figure 4.3: Profile Plot of Time by Group for Algometer**

**b) Stabilizer Pressure**

For stabilizer pressure, there was a significant decrease over time for both groups ( $p=0.001$ ), but no evidence of a differential treatment effect ( $p=0.392$ ) was seen. Figure 4.4 shows that the core group continued to decrease between time 2 and 3, while the adjustment group increased in this time. However, this slight trend was not statistically significant.

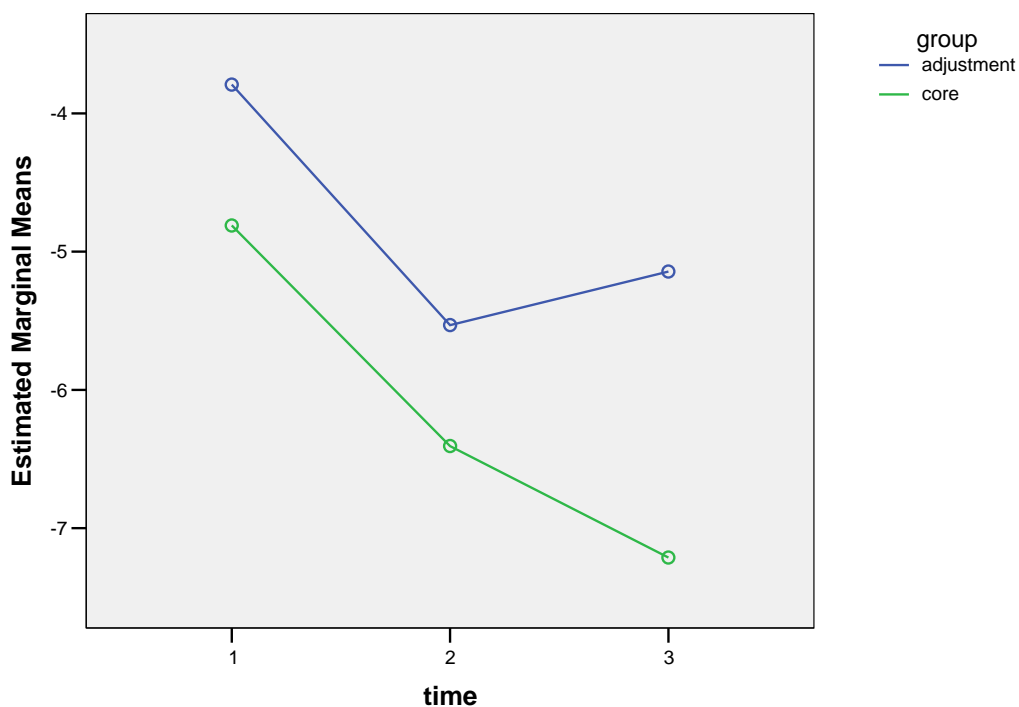
**Discussion**

Figure 4.4 shows an initial improvement in both groups up until time 2, thereafter the Adjustment groups appears to get worse while the Core Group continues to improve (Carpes *et al.* 2007). A possible explanation for this is that the Core Group are performing core-strengthening exercises which ideally should improve their core strength. The initial improvement in the Adjustment group could be

explained due to the fact that there was painful inhibition caused by their low back pain (Spencer et al. 1984; Baugher et al., 1984), but as they receive treatment and their pain decreases they are able to contract (Pickar, 2002) their core muscles to the maximum of their ability. Hides et al.1994 noted that there was atrophy of the multifidus muscle in patients who had suffered from low back pain even though they may not have low back pain at the time and this may influence the strength with which this muscle may be contracted (Biederman et al., 1991). Thus however it is not a true increase in strength, but a showing of the participants own fully functional ability to contract the core muscles. It must be noted that participants of this study were required to have acute low back pain (i.e. with an onset of seven days or less), but a history of low back pain did not exclude them from the study.

**Table 4.9: Within and Between Subjects Effects for Stabilizer Pressure**

Effect	Statistic	P value
Time	Wilk's Lambda=0.616	0.001
Group	F=1.317	0.260
Time*group	Wilk's Lambda=0.937	0.392



**Figure 4.4: Profile Plot of Time by Group for Stabilizer Pressure**

**c) Stabiliser Endurance Reading**

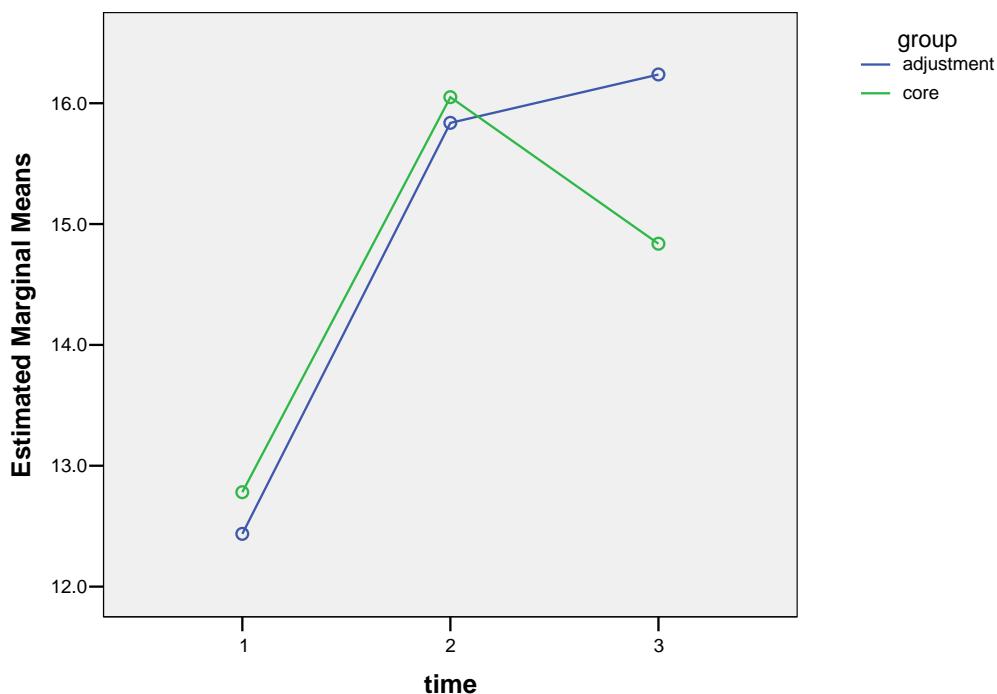
There was no significant effect for time on stabilizer between groups ( $p=0.713$ ), although both groups showed a significant increase over time ( $p=0.003$ ). Figure 4.5 shows that the core group started to decrease after time 2, while the adjustment group continued to increase. Statistically for this outcome, both groups progressed equally over time. There is a crossover effect noted in Figure 4.5 as the Manipulation Group continue to have a decrease in their stabiliser readings from time 2, while the Core Group appear to begin increasing their readings at time 2.

## **Discussion**

Figure 4.5 shows that there is an increase in the length of time that the contraction of the core muscles could be held in both treatment groups. It can be noted that at time 2 the Core Group shows a sharp decline in their progress. This could possibly be explained due to the fact that the core exercises were only performed for the first two weeks of the three-week process, while reading taken at time 3 was taken after one week of no exercise. The adjustment group received treatment throughout the same period as the Core group, but as their pain decreased (Koumantakis et al., 2005) the Adjustment group was able to hold their contraction as there was no painful inhibition or Arthrogenic Muscle Inhibition preventing the contraction of these muscles (Suter et al. 2000)

**Table 4.10: Within and Between Subjects Effects for Time on Stabilizer**

Effect	Statistic	p-value
Time	Wilk's Lambda=0.665	0.003
Group	F=0.026	0.874
Time*group	Wilk's Lambda=0.977	0.713



**Figure 4.5: Profile Plot of Time by Group for Time on Stabilizer**

**d) Surface EMG (Electromyograph) L1**

There was a borderline non-significant interaction effect for EMG L1 ( $p=0.072$ ). As noted in Chapter 3, the EMG electrodes were placed at the level of lumbar vertebra one (L1) and lumbar vertebra four (L4), two centimeters from the spinous process. Figure 6 shows a trend towards the adjustment group showing a mean decrease over time while the core group remained relatively stable over time.

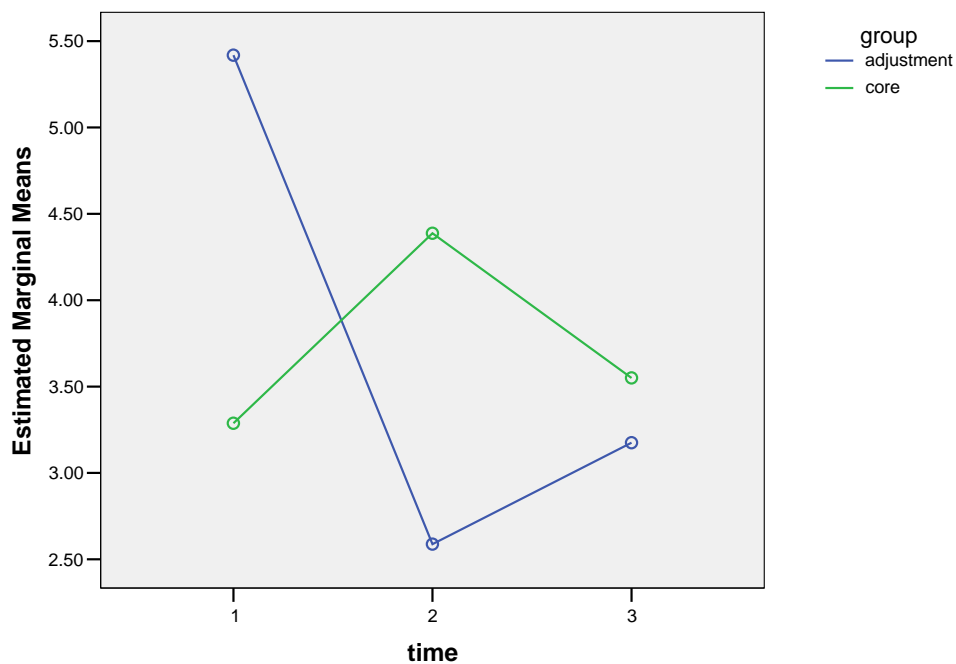
**Discussion**

The Surface EMG is placed over the belly of the Quadratus Lumborum muscle at the level of L1 to measure the contraction of the Quadratus Lumborum muscle at this point. As can be seen in Figure 4.6 the Adjustment group (Keller *et al.*, 2000) had an initially higher reading at L1 than the Core Group appears to have.

A possible explanation for this is that over the treatment period the Adjustment Group have a decrease in their pain and are thus able to contract their core muscles fully without using the QL (global muscles) muscle to “cheat” and fake a contraction of the core muscles this is however in contradiction to the findings in b and c above and in contradiction to Hides et al., (2000). The Core Group were performing exercises to strengthen the Core muscles, however as has been noted in previous chapters, there is no exact method to prevent the participant from using global muscles to “fake” a core contraction (Richardson et al., 1995). By performing the exercises repetitively (Akuthota et al., 2004) the participants learn to “cheat” and this is seen in Figure 4.6 as an increase in QL activity at L1 by time 2. During the treatment period the participants in the Core Group were, at their visit, corrected on their exercise method by the researcher to try and prevent the use of the QL muscle in the core contraction (Richardson et al., 1995). This may be a possible explanation for the decrease in QL use by time 3 (Graves et al., 1990; Lariveire et al. 2003b). Other possibilities include that a manipulation causes a decrease in pain and therefore a decrease in muscle spasm (Herzog et al., 1999; Symonds et al., 2000) or activity as measured by the surface EMG (i.e. superficial muscles), however after a while with no treatment the lack of functioning of the core (i.e. according to Hides et al. 2000 the core muscles are not affected significantly even though the low back pain has decreased). As a result the global muscles need to brace and take over the function, thus resulting in increased muscle activity. On the other hand, the core will have an initial increase in the activity as the patient learns how to contract the core (Akuthota et al., 2004), thus by “cheating” as previously mentioned as they are more able to contract the core the superficial muscle activity decreases.

**Table 4.11: Within and Between Subjects Effects for Surface EMG L1**

Effect	Statistic	p-value
Time	Wilk's Lambda= 0.913	0.269
Group	F= 0.00	0.990
Time*group	Wilk's Lambda= 0.834	0.072



**Figure 4.6: Profile Plot of Time by Group for EMG L1**

**e) Surface EMG (Electromyograph) L4**

The Surface EMG was placed over the belly of the Quadratus Lumborum muscle at the level of L4 to measure the activity in the Quadratus Lumborum muscle during core contraction. For EMG at L4, no significant time by group interaction effect was demonstrated (p=0.720). Figure 4.7 shows that both groups

decreased over time to the same extent, thus there was no differential effect of the two treatments over time.

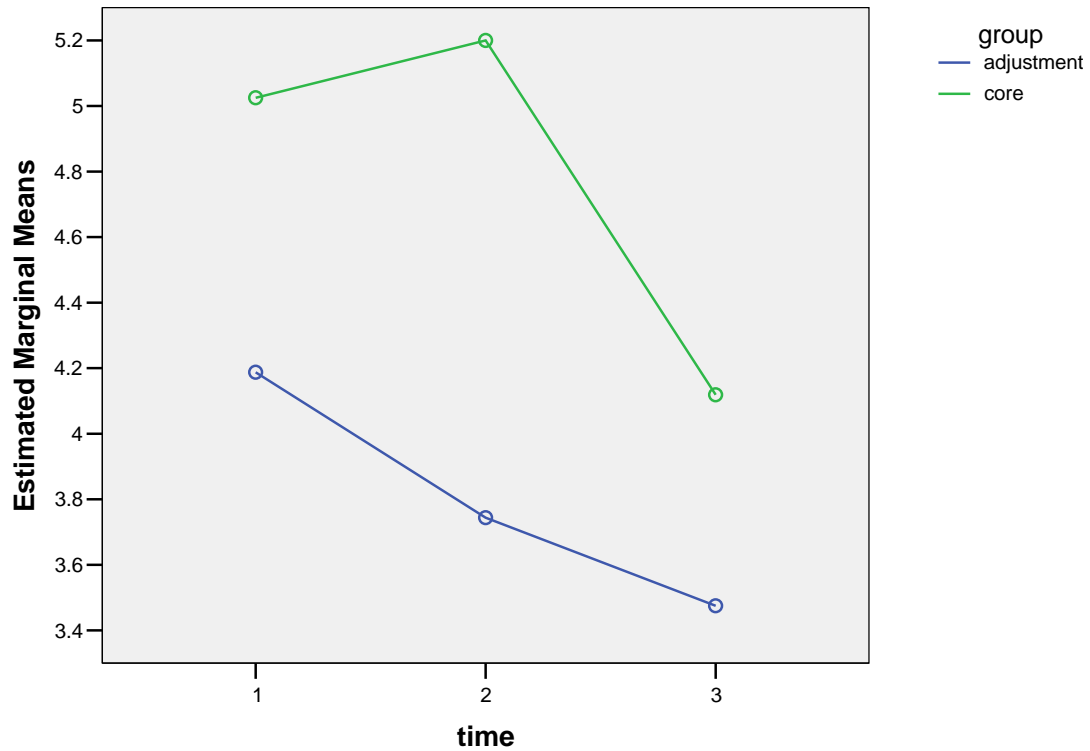
### **Discussion**

In figure 4.7 it can be noted that the Core Group initially used the Quadratus Lumborum muscle at L4 more than at L1 as was seen in Figure 4.6. It appears that as the Core Group progressed in their treatment protocol they did learn to use the correct muscles (i.e. the core muscles) rather than “cheating” using the Quadratus Lumborum muscle. In both Groups the initially high readings (Arendt-Nielsen et al., 1996; Radehold et al., 2000, Hodges et al., 2003a,b) were hypothesized to be caused by muscle spasm in the Quadratus Lumborum muscle. There was a steady decrease in the readings obtained from the Manipulation Group which could in part be cause by the hypothesized decrease in muscle spasm, but could also be due to the fact that in order to obtain these readings, all participants were taught to contract their core muscle and inevitably the Manipulation Group will learn to contract the core muscle more efficiently every time they are asked to do so.

**Table 4.12: Within and Between Subjects Effects for EMG L4**

Effect	Statistic	p- value
Time	Wilk’s Lambda= 0.947	0.415
Group	F= 1.067	0.310
Time*group	Wilk’s Lambda= 0.978	0.720





**Figure 4.7: Profile Plot of Time by Group for EMG L4**

f) **Surface EMG (Electromyograph) Abdominal**

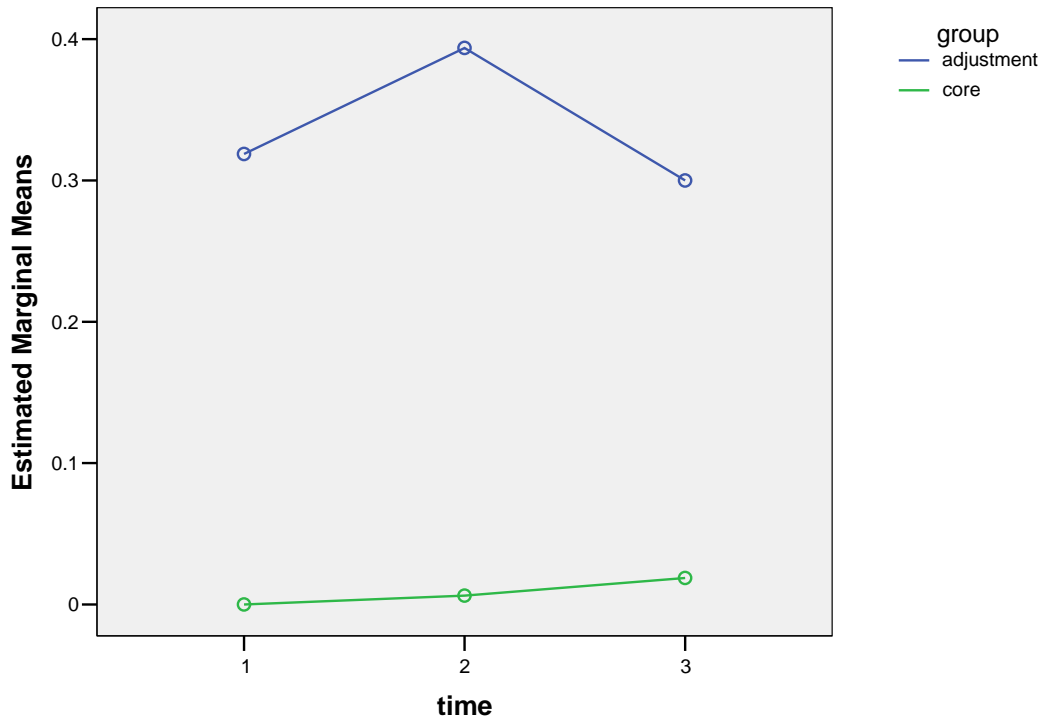
The two groups behaved very similarly over time for this outcome. There was no evidence of a time effect ( $p=0.226$ ) or a differential treatment effect ( $p= 0.549$ ). This is demonstrated by the parallel profiles in Figure 4.8.

## **Discussion**

In the core group there was a very slight but steady improvement in the core contraction, which is positively explained by the treatment protocol where they were performing exercises to increase to the core muscle strength. The adjustment group appears to begin by using their core muscles but as their treatment progresses and their pain decreases they appear to revert to using the Quadratus Lumborum muscles (global muscles) along with the multifidus as has been noted in the above figures 4.5 and 4.6.

**Table 4.13: Within and Between Subjects Effects for EMG Abdominal**

Effect	Statistic	P value
Time	Wilk's Lambda= 0.903	0.226
Group	F= 0.961	0.335
Time*group	Wilk's Lambda= 0.960	0.549



**Figure 4.8: Profile Plot of Time by Group for EMG Abdominal**

**g) Abdominal Stabiliser**

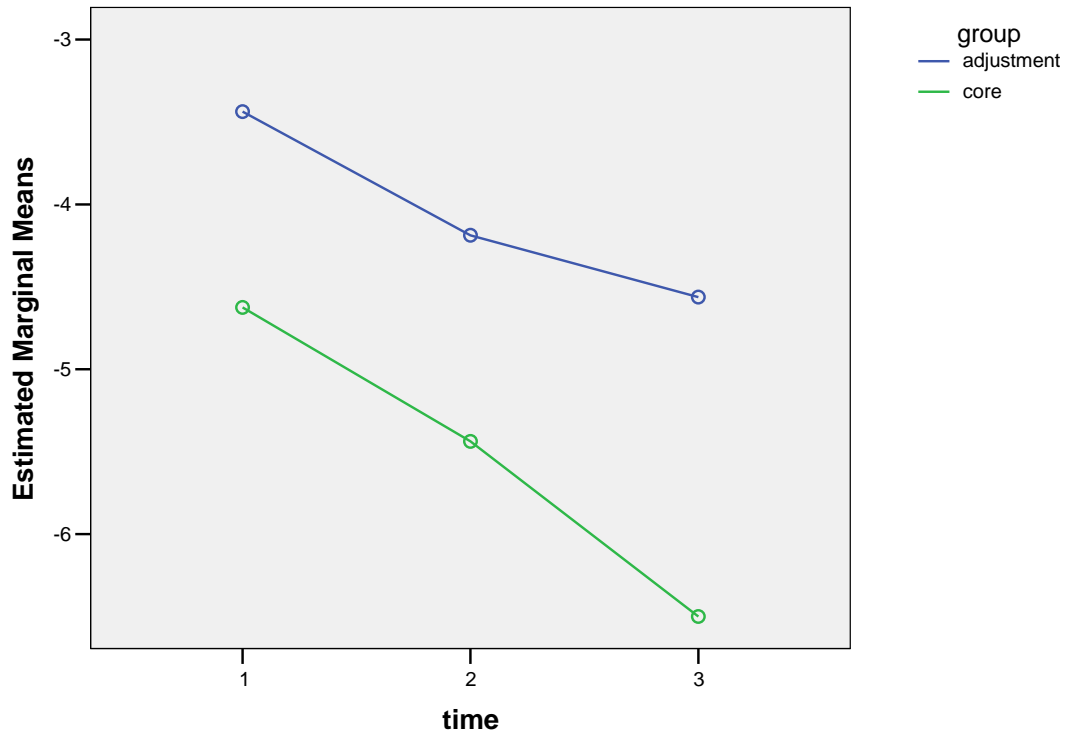
Although there was a significant decrease over time for this outcome ( $p=0.003$ ), the decrease was shown equally in both treatment Groups, leading to a conclusion of no differential treatment effect ( $p=0.589$ ). This is shown in Table 14 and by the parallel profiles of the two groups in Figure 9.

## **Discussion**

In Figure 4.9 it can be seen that both Groups appear to have a decrease in the pressure reading taken using the Biofeedback Stabiliser, indicating that there is an increase in the strength of the Transversus Abdominus contraction, however the Core Group does appear to experience a larger drop in their pressure readings. This could indicate that the core rehabilitation exercises could have an affect on core strengthening, which supports the available literature (Akuthota et al., 2004; Ericksen et al., 2006) on the subject.

**Table 4.14: Within and Between Subjects Effects for Stabilizer Abdominal**

Effect	Statistic	p- value
Time	Wilk's Lambda= 0.669	0.003
Group	F= 1.356	0.253
Time*group	Wilk's Lambda= 0.964	0.589



**Figure 4.9: Profile Plot of Time by Group for Stabilizer Abdominal**

## **4.6 Binary Outcomes**

### **4.6.1 Presence of Fixations on the Left Side**

For this outcome, Table 4.15 shows that there were no significant effects. There was no differential treatment effect ( $p=0.392$ ), thus the treatment did not affect the presence of fixation on the left side.

**Table 4.15: Binary General Linear Regression Model Analysis for Presence of Fixations on the Left Side**

Effect	Statistic	p-value
Time	Risk ratio= 0.959	0.784
Group	Risk ratio= 0.718	0.279
Time*group	Risk ratio= 0.907	0.392

#### **4.6.2 Presence of Fixation on the Right Side**

There was a significant time effect for presence of fixations in the right side ( $p=0.040$ ), indicating that there was a decrease in the risk as time progressed. This decline in risk was the same in both treatment groups, as there was no evidence of a differential treatment effect ( $p=0.252$ ). It can be noted that the researcher is right handed and therefore adjustments performed by the researcher using her right hand may have been more effective than those performed using the left hand, this may have influenced the results.

**Table 4.16: Binary General Linear Regression Model Analysis for Presence of Fixations on the Right Side**

Effect	Statistic	p-value
Time	Risk ratio= 0.321	0.040
Group	Risk ratio= 0.103	0.161
Time*group	Risk ratio= 1.476	0.252

#### **4.6.3 Presence of Fixations at L1**

For this outcome, Table 4.17 shows that there were no significant effects. There was no differential treatment effect ( $p=0.490$ ), thus the treatment did not affect the presence of fixation at L1.

**Table 4.17: Binary General Linear Regression Model Analysis for Presence of Fixations at L1**

Effect	Statistic	p- value
Time	Risk ratio= 0.939	0.865
Group	Risk ratio= 1.564	0.486
Time*group	Risk ratio= 0.854	0.490

#### **4.6.3 Presence of Fixations at L2**

For this outcome, Table 4.18 shows that there were no significant effects. There was no differential treatment effect ( $p=0.273$ ), thus the treatment did not affect the presence of fixation at L2.

**Table 4.18: Binary General Linear Regression Model Analysis for Presence of Fixations at L2**

Effect	Statistic	p- value
Time	Risk Ratio = 0.488	0.103
Group	Risk Ratio = 0.884	0.830
Time*group	Risk Ratio = 1.299	0.273

#### **4.6.4 Presence of Fixations at L3**

For this outcome, Table 4.19 shows that only the time effect was significant ( $p<0.001$ ), meaning that there was a significant decrease in risk of fixations at L3 over time in both groups. There was no differential treatment effect ( $p=0.453$ ), thus the treatment did not affect the presence of fixation at L3. There are however arguments that L3 being the apex of the lumbar lordosis and therefore

the point of most movement in the lumbar spine it may therefore bear the brunt if there were any mechanical dysfunction. It may be possible that patients who “cheat” by using global muscles may place increased pressure on the L3 vertebra causing a mechanical dysfunction and therefore increasing the risk of subluxation at this point. This could explain the lack of findings at the other lumbar levels.

**Table 4.19: Binary General Linear Regression Model Analysis for Presence of Fixations at L3**

Effect	Statistic	p-value
Time	Risk Ratio = 0.405	<0.001
Group	Risk Ratio = 3.092	0.298
Time*group	Risk Ratio = 0.860	0.453

#### **4.6.5 Presence of Fixations at L4**

For this outcome, Table 4.20 shows that there were no significant effects. There was no differential treatment effect ( $p=0.359$ ), thus the treatment did not affect the presence of fixation at L4.

**Table 4.20: Binary General Linear Regression Model Analysis for Presence of Fixations at L4**

Effect	Statistic	p-value
Time	Risk Ratio = 0.984	0.968
Group	Risk Ratio = 0.914	0.904
Time*group	Risk Ratio = 0.761	0.359



#### **4.6.7 Presence of Fixations at L5**

For this outcome, Table 4.21 shows that there were no significant effects. There was no differential treatment effect ( $p=0.432$ ), thus the treatment did not affect the presence of fixation at L5.

**Table 4.21: Binary General Linear Regression Model Analysis for Presence of Fixations at L5**

Effect	Statistic	p-value
Time	Risk Ratio = 0.990	0.978
Group	Risk Ratio = 0.588	0.525
Time*group	Risk Ratio = 0.811	0.432

#### **4.6.8 Presence of Fixations at S1**

For this outcome, Table 4.22 shows that there were no significant effects. There was no differential treatment effect ( $p=0.480$ ), thus the treatment did not affect the presence of fixation at S1.

**Table 4.22: Binary General Linear Regression Model Analysis for Presence of Fixations at S1**

Effect	Statistic	p-value
Time	Risk Ratio = 0.913	0.277
Group	Risk Ratio = 0.689	0.268
Time*group	Risk Ratio = 1.047	0.480

## **4.7 Correlations Between Changes in Outcomes Over Time**

### **4.7.1 Adjustment Group**

Table 4.23 shows that changes in NRS and algometer measurements were significantly negatively correlated ( $r=-0.659$ ,  $p=0.006$ ), indicating that as NRS decreased, algometer measurements increased. Change in NRS was also negatively correlated with changes in EMG abdominal ( $r=-0.534$ ,  $p=0.033$ ), although this was a relatively weak correlation. There was an expected positive correlation between changes in EMG for L1 and L4 ( $r=0.577$ ,  $p=0.019$ ), and between changes in stabilizer pressure and stabilizer abdominal ( $r= 0.561$ ,  $p=0.024$ ).

### **4.7.2 Core Group**

Table 4.24 shows that the core group experienced negative correlations between change in stabilizer pressure and change in EMG for L1 ( $r= -0.620$ ,  $p=0.010$ ) and for L4 ( $r= -0.556$ ,  $p=0.025$ ). NRS was also negatively correlated with changes in abdominal EMG ( $r= -0.520$ ,  $p=0.039$ ), and algometer changes were negatively correlated with changes in abdominal stabilizer ( $r= -0.512$ ,  $p=0.042$ ). There was a strong positive correlation between change in EMG for L1 and L4 ( $r=0.805$ ,  $p<0.001$ ). Change in stabilizer pressure and abdominal stabilizer were also positively correlated ( $r= 0.699$ ,  $p=0.003$ ).

**Table 4.23: Pearson's Correlation Between Changes in Outcomes for the Adjustment Group (n=16)**

		Change in RMQ	Change in NRS	Change in Algometer	Change in stabilizer	Change in time on stabilizer	Change in EMG for L1	Change in EMG for L4	Change in EMG Abdo	Change in stabilizer for Abdo
Change in RMQ	Pearson's r	1	0.297	0.115	-0.036	0.149	-0.070	0.088	-0.389	-0.142
	P value		0.264	0.673	0.896	0.582	0.798	0.747	0.136	0.600
Change in NRS	Pearson's r	0.297	1	-0.659(**)	-0.081	-0.144	0.179	0.075	-0.534(*)	0.129
	P value	0.264		0.006	0.767	0.596	0.507	0.782	0.033	0.633
Change in Algometer	Pearson's r	0.115	-0.659(**)	1	-0.052	0.467	-0.236	0.131	0.168	0.110
	P value	0.673	0.006		0.849	0.068	0.378	0.627	0.534	0.685
Change in stabilizer	Pearson's r	-0.036	-0.081	-0.052	1	-0.052	-0.363	-0.358	0.120	0.561(*)
	P value	0.896	0.767	0.849		0.847	0.167	0.174	0.657	0.024
Change in time on stabilizer	Pearson's r	0.149	-0.144	0.467	-0.052	1	0.082	0.303	-0.259	0.145
	P value	0.582	0.596	0.068	0.847		0.762	0.254	0.334	0.593
Change in EMG for L1	Pearson's r	-0.070	0.179	-0.236	-0.363	0.082	1	0.577(*)	-0.304	-0.149
	P value	0.798	0.507	0.378	0.167	0.762		0.019	0.252	0.583
Change in EMG for L4	Pearson's r	0.088	0.075	0.131	-0.358	0.303	0.577(*)	1	-0.229	-0.170
	P value	0.747	0.782	0.627	0.174	0.254	0.019		0.394	0.528
Change in EMG Abdo	Pearson's r	-0.389	-0.534(*)	0.168	0.120	-0.259	-0.304	-0.229	1	0.139
	P value	0.136	0.033	0.534	0.657	0.334	0.252	0.394		0.607
Change in stabilizer for Abdo	Pearson's r	-0.142	0.129	0.110	0.561(*)	0.145	-0.149	-0.170	0.139	1
	P value	0.600	0.633	0.685	0.024	0.593	0.583	0.528	0.607	

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

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

### Discussion

Positive	EMG L4	EMG L1	The changes with respect to the EMG were reflected at both the L1 and the L4 taking an overall reading into account. However the mechanism by which each of these readings were arrived at where different and therefore this correlation does not add any further value to the discussion (as seen under sections 4.5.2 d) and 4.5.2 e).
	Stabiliser Abdo	Stabiliser	Section 4.5.2. b) and g) act as reference and as expected both these values decreased with time, thereby resulting in a positive correlation .
Negative	Algometer	NRS	As discussed under sections 4.5.2. a) and 4.5.1. b) respectively the NRS (Bolton <i>et al.</i> , 1998) was expected to decrease with a reciprocal increase in the value of the algometer (Fischer , 1986).
	EMG Abdo	NRS	As discussed under sections 4.5.2. g) and 4.5.1 b) the NRS decreased over time (Bolton <i>et al.</i> , 1998), but there was no change in the overall EMG readings hence the correlation reading obtained here is not reflective of the overall clinical result as discussed in each of these sections.

**Table 4.24: Pearson's Correlation Between Changes in Outcomes for the Core Group (n=16)**

		Change in RMQ	Change in NRS	Change in Algometer	Change in stabilizer	Change in time on stabilizer	Change in EMG for L1	Change in EMG for L4	Change in EMG Abdo	Change in stabilizer for Abdo
Change in RMQ	Pearson's r	1	0.006	-0.338	0.257	-0.031	0.109	-0.280	0.102	0.462
	P value		0.982	0.201	0.337	0.909	0.689	0.294	0.707	0.072
Change in NRS	Pearson's r	0.006	1	-0.263	0.140	-0.270	-0.068	0.105	-0.520(*)	0.173
	P value	0.982		0.326	0.606	0.312	0.804	0.700	0.039	0.523
Change in Algometer	Pearson's r	-0.338	-0.263	1	-0.172	0.003	0.381	0.308	0.195	-0.512(*)
	P value	0.201	0.326		0.524	0.992	0.145	0.246	0.468	0.042
Change in stabilizer	Pearson's r	0.257	0.140	-0.172	1	0.076	-0.620(*)	-0.556(*)	0.115	0.699(**)
	P value	0.337	0.606	0.524		0.779	0.010	0.025	0.673	0.003
Change in time on stabilizer	Pearson's r	-0.031	-0.270	0.003	0.076	1	0.090	0.134	0.077	-0.034
	P value	0.909	0.312	0.992	0.779		0.740	0.620	0.778	0.900
Change in EMG for L1	Pearson's r	0.109	-0.068	0.381	-0.620(*)	0.090	1	0.805(**)	-0.101	-0.320
	P value	0.689	0.804	0.145	0.010	0.740		0.000	0.709	0.227
Change in EMG for L4	Pearson's r	-0.280	0.105	0.308	-0.556(*)	0.134	0.805(**)	1	-0.232	-0.443
	P value	0.294	0.700	0.246	0.025	0.620	0.000		0.388	0.086
Change in EMG Abdo	Pearson's r	0.102	-0.520(*)	0.195	0.115	0.077	-0.101	-0.232	1	0.207
	P value	0.707	0.039	0.468	0.673	0.778	0.709	0.388		0.442
Change in stabilizer for Abdo	Pearson's r	0.462	0.173	-0.512(*)	0.699(**)	-0.034	-0.320	-0.443	0.207	1
	P value	0.072	0.523	0.042	0.003	0.900	0.227	0.086	0.442	

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

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

## Discussion

Positive	Stabiliser abdo	Stabiliser	As seen in sections 4.5.2 b) and 4.5.2 g), both these measures decreased with time resulting in a positive correlation. This result supports the hypothesis made at the outset of this study.
	EMG L1	EMG L4	The changes with respect to the EMG were reflected at both the L1 and the L4 taking an overall reading into account. However the mechanism by which each of these readings were arrived at where different and therefore this correlation does not add any further value to the discussion (as seen under sections 4.5.2 d) and 4.5.2 e).
Negative	EMG abdo	NRS	With the use of the transversus abdominis it is expected that the EMG abdo reading increases whilst at the same time there is a decrease in the NRS (Bolton et al. 1998) as discussed in sections 4.5.1 b) and 4.5.2 f).
	Stabiliser abdo	Algometer	With the increased negativity of the stabiliser reading, there is decreased pressure on the biofeedback stabiliser resulting in a more negative reading, whereas the Algometer shows a steady increase as the tenderness over the points decreases (Fischer, 1986) – see sections 4.5.2 a) and 4.5.2 g).
	Stabiliser	EMG L1	With intervention (core stabilization) there was a decrease in the stabiliser readings (as per rationale in negative correlation above), as compared to the marginal increase in the readings for the EMG L1 , thus resulting in a negative correlation, which supports the discussion in sections 4.5.2 b) and 4.5.2 d).
	Stabiliser	EMG L4	4.5.2 b) and 4.5.2 e)

## Summary of Results and Discussion

The objectives of this study as laid out in chapter one were defined as follows, with their respective hypotheses:

- 4.8.1 To determine the relative effect of manipulation (Group A) and core rehabilitation (Group B) in sedentary patients suffering from acute mechanical low back pain in terms of the subjective findings
- 4.8.2 To determine the relative effect of manipulation (Group A) and core rehabilitation (Group B) in sedentary patients suffering from acute mechanical low back pain in terms of the objective findings.

4.8.3 To determine any correlations between the subjective and objective outcomes for Group A and Group B.

#### **4.9 Hypotheses**

4.9.1 Patients suffering from acute mechanical low back pain should not show a clinical improvement (subjective and objective) when treated using manipulation.

4.9.2 Patients suffering from acute mechanical low back pain should not show a clinical improvement (subjective and objective) when treated using core stability exercises.

#### **4.10 Rejection / Acceptance of the Hypotheses**

Hypothesis one indicated that patients suffering from acute mechanical low back pain should not show a clinical improvement (subjective and objective) when treated using manipulation.

This hypothesis is rejected based on the results obtained in this study as there was clinical improvement in the patients as noted in NRS, Algometer and Stabiliser.

Hypothesis two indicated that patients suffering from acute mechanical low back pain should not show a clinical improvement (subjective and objective) when treated using core stability exercises.

This hypothesis is rejected based on the results obtained in this study as there was clinical improvement in the patients as noted in NRS, Algometer and Stabiliser.

Notwithstanding the above results this research was limited in explaining the mechanisms by which patients obtained readings as the measures utilized only assessed clinical parameters and not direct measures (e.g. a needle electrode EMG may have yielded more mechanism specific information as opposed to a surface EMG).

## **Chapter 5**

### **5.1 Introduction**

This chapter will look at the conclusions and recommendations that were made during the period of this study. The study aimed to investigate the relative effect of manipulation and core rehabilitation in the treatment of acute mechanical low back pain in sedentary patients. The sample consisted of 32 male and female participants between the ages of 18 and 45.

### **5.2 Conclusion**

According to statistical analyses the outcome of this study lacked statistical significance however, the trends show that both treatment Groups showed an improvement with regards to pain, daily functionality and movement. Trends also indicate that there was an improvement in the Core Rehabilitation Group with regards to core strength and endurance. The final conclusion drawn by the researcher is that core rehabilitation properly performed and instituted can be an effective treatment protocol when compared with manipulation. Although, according to the literature, these two interventions have very different mechanisms of action they appear to be comparable in their outcomes.



### 5.3 Recommendations

a) Sample Size

A larger sample size would increase the validity of the results and possibly prevent a type 1 error from being incurred.

b) Length of Treatment

Although the treatment period for this study was 3 weeks, it was found by the researcher to be too long as there was a high percentage of drop-outs (15 participants dropped out, out of the 47 participants who began the study) due to the fact that patients were getting better before the 3 weeks of treatment were up and did not want the hassle of coming into the Chiropractic Day Clinic for treatments. Therefore a shorter follow-up period is recommended, even though literature indicated that a longer treatment period was recommended (Carpes et al., 2007).

c) Use of the Needle EMG

This study used the surface EMG to take readings of the quadratus lumborum muscle, however the surface EMG may not be entirely accurate (Carman et al., 1972; Strohl et al., 1981; Goldman et al., 1987; De Troyer et al., 1990) especially in participants who have excess body weight. For future research the researcher recommends the use of needle EMG to improve the accuracy of the readings, possibly needle EMG guided by

Ultrasound, to ensure specific placement of the needle within a specific muscle.

d) Treatment Protocol

This research looked specifically at two separate treatment protocols, however, further research may consider whether a combined treatment of manipulation and rehabilitation of the core musculature would prove to be an even more efficient treatment

e) Stratification of Sample

In this research no distinction was made between those patients who had a history of low back pain and those who did not. For future research it is recommended that the groups are either stratified in this regard or research is restricted to either those who do have a history of low back pain or those who do not.

## REFERENCES

- Akuthota, V., and Nadler, S.F. 2004. Core Strengthening. Arch of Phys Med Rehabil, vol 85:86-92.
- Andersson, G.B.J. The epidemiology of spinal disorders. 1997. In Frymoyer JW (ed.). The Adult Spine: Principles and Practice. 2nd edn. New York: Raven Press, p. 93.
- Andersson, G.B.J., Pope, M.H., Frymoyer, J.W., Snook, S. Epidemiology and Cost. In Pope, M.H., Andersson, G.B.J., Frymoyer, J.W., Chaffin, D.B. Ed. Occupational low back pain: Assessment, treatment and prevention. St. Louis, MO: Mosby 1991
- Arendt-Nielsen, L., Graven-Nielsen, T., Svarrer, H., Svensson, P. 1996. The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. Pain. 64:231-40
- Assendelft, W.J., Morton, P.C., Yu E.I. 2003 Spinal manipulative therapy for low back pain: a meta-analysis of effectiveness relative to other therapies. Annals of Internal Medicine. 12:135-41
- Bailey, D.M. 1997. Research for the Health Professional. A Practical Guide. 2<sup>nd</sup> edition. F.A. Davis Company. USA. p. 278. ISBN: 0803601514.
- Bland, M. 1996. An Introduction to Medical Statistics. 2<sup>nd</sup> edition. Oxford University Press. Great Britain. p. 396. ISBN: 0192624288
- Barr, K.P, Griggs, M, Cadby, T. 2005. Lumbar Stabilisation: Core Concepts and Current Literature, part 1. Am J Phys Med Rehabil, 84: 473-80.

Baughner, W.M., Warren, R.S., Marshall, J.L., Joseph, A. 1984. Quadriceps atrophy in anterior cruciate deficient knee. The American Journal of Sports Medicine. 16:113-122

Bergmann, T., Peterson, D.H., Lawrence, D. 1993. Chiropractic Technique-Principles and Procedures. New York: Churchill Livingstone Inc. ISBN 0-443-08752-0.

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

Bergmark, A. 1989. Stability of the lumbar spine. A study in mechanical engineering. Acta Orthop Scand Suppl. 230:20-40

Biederman, H.J., Shanks, G.L., Forrest, W.J., Inglis, J. 1991. Power spectrum analyses of electromyographic activity: discriminators in differential assessment of patients with chronic low back pain. Spine. 16:1179-1184.

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.

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

Bolton, J.E., Wilkinson, R. 1997. Clinical Anatomy and Management of Low Back Pain pg 353.

Bronfort, G., Haas, M., Evans, R.L., and 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 4:335-356.

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

Carman, D.J., Blanton, P.L., Biggs, N.L. 1972. Electromyographic study of the anterolateral abdominal musculature using indwelling electrodes. American Journal of Physical Medicine. 51:113-29

Carpes, F.P., Reinehr, F.B., Mota, C.B. 2007. Effects of a program for trunk strength and stability on pain, low back and pelvis kinematics, and body balance: A pilot study. Journal of Bodywork and Movement Therapies.

Campbell, M.J, Machin, D. 1999. Medical Statistics. A Commonsense Approach. 3<sup>rd</sup> edition. John Wiley & Sons Ltd. Great Britain. p. 203. ISBN: 0471987212.

Cassidy, J.D., Burton, C.V. 1992. Economics, epidemiology and risk factors. In: Kirkaldy-Willis, W.H. Managing Low Back Pain. 3<sup>rd</sup> ed. New York: Churchill Livingstone. 418p.

Cherkin, D.C., Deyo, R.A., Wheeler, K., Ciol, M.A. 1995. Physicians view about treating low back pain: the results of a national survey. Spine. 20:1-10

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

Cox, J.M. 1998. Low Back Pain Mechanism, Diagnosis and Treatment. 6<sup>th</sup> edition. p 225-227. ISBN: 0-683-30358-9.

Cramer, G.D., Darby, S.A. 1995. Basic and Clinical Anatomy of the Spine, Spinal Cord and ANS. Mosby. Year Book Inc., St Louis, Missouri, USA. ISBN 0-8016-6467-5.

Cresswell, A.G, Grundstrom, A., Thorstensson, A. 1992. Observations on intra-abdominal pressure and patterns of abdominal intra-muscular activity in Man. Acta Physiological Scandinavica. 144:409-418.

Cresswell, A.G., Oddsson, L., Thorstensson, A. 1994. The influence of sudden perturbances on trunk muscle activity and Intra-abdominal pressure while standing. Experimental Brain Research. 48:336-341.

Crossman, A.R. and Neary, D. 1995. Neuroanatomy: An illustrated colour text. Churchill Livingstone. ISBN 0-443-04479-1.

Danneels, L.A., Vanderstraeten, G.G., Cambier, D.C., Witvrouw, E.E., De Cuyper, H.J. 2000. CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. Eur Spine J. 9:266-72

Danneels, L.A., Coorevits, P.L., Cools, A.M., Vanderstraeten, G.G., Cambier, D.C., Witvrouw, E.E., De Cuyper, H.J. 2002. Differences in electromyographic activity in the multifidus muscle and the iliocostalis lumborum between healthy subjects and patients with sub-acute and chronic low back pain. European Spine Journal. 11:13-19

Darby, S.A. and Daley, D.L. 1995. Neuroanatomy of the Spinal Cord. In: Cramer, G.D. and Darby, S.A. (eds). Basic and Clinical Anatomy of the Spine, Spinal cord and ANS. Mosby. ISBN 0-8016-6467-5

- Descarreaux, M., Normand, M.C., Laurencelle, L. and Dugas, C. 2002. Evaluation of a specific home exercise program for low back pain. Journal of Manipulative and Physical Therapy. 25:497-503.
- De Troyer, A., Estenne, M., Ninane, V., Van Gansbeke, D., Gorini, M. 1990. Transversus abdominis muscle function in humans. Journal of Applied Physiology. 68:1010-6
- Deyo, R.A., and Weinstein, J.N. 2001. Primary care: low back pain. The New England Journal of Medicine 344:363-370.
- Deyo, R.A., Cherkin, D., Conrad, D. and Volinn, E. 1991. Cost, controversy, crisis: low back pain and the health of the public. Annual Review Public Health 12:141-56.
- Diamond, S. and Borenstein, D., 2006. Chronic Low Back Pain in a Working Age Adult. Best Practice and Research Clinical Rheumatology. 20:707-720.
- Dionne, C.E. Low Back Pain. In: Crombie, I.K, Ed. Epidemiology of Pain. Seattle: IASP Press; 1999. p283-97
- Docrat, A. 1999. A comparison of the epidemiology of low back pain in Indian and Coloured communities in South Africa. Masters Degree in Technology: Durban Institute of Technology, Berea, Durban, South Africa.
- Eccleston, C., Crombez, G. Pain demands attention: a cognitive-affective model of the interruptive function of pain. Psychol Bull 1999. 125:356-66
- Evans. C. and 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. 8:162-174.

Ericksen, J.J., Bean, J.F., Klely, D.K., Hicks, G.E., Levelle, S.G. 2006. Does Gynecologic surgery contribute to low back pain problems later in life? An analysis of the women's health and aging study. Archives of Physical Medicine and Rehabilitation. 87:172-176

Esterhuizen, T. 2007. Personal Communication.

Fejer, R., Jordan, A. and Hartvigsen C. 2005. Categorising the severity of neck pain: Establishment of cut-points for use in clinical and epidemiological research. Pain. 119: 176-182.

Ferreira, M., Ferreira, P.H., Latimer, J. 2002. Does spinal manipulative therapy help people with chronic low back pain? Australian Journal of Physiotherapy. 48:277-84

Ferreira, M.L., Ferreira, P.H., Hodges, P.W. 2007. Changes in postural activity of the trunk muscles following spinal manipulative therapy. Journal of Manual Therapy. 12:240-248

Feuerstein, M., Marcus, S.C., Huang, G.D. 2004. National trends in nonoperative care for nonspecific back pain. Spine. 4:56-63

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

Frank, J.W., Kerr, M.S., Brøker, A.S. Disability resulting from occupational low back pain. Part 1. What do we know about primary prevention? 1996. Spine. 21:2908-2917

Friedwald, J., Reuter, I. and Engelhard, M. 1999. Neuromuscular and Motor System Alterations After Knee trauma and Surgery. In: Lehman et al. (ed)



Overload Performance Incompetence and Regeneration in Sport. New York: Kluwer Academic/ Plenum Publishers pp 81-100. Online: [http://www.FB3/sport/bewegungslehre/freiwald/dokumente/neuromusc\\_motorsystem.pdf](http://www.FB3/sport/bewegungslehre/freiwald/dokumente/neuromusc_motorsystem.pdf). (accessed 3/8/2003).

Gatterman, M.I. 1982. Chiropractic Management of Spine Related Disorders. Baltimore: Williams and Wilkins. ISBN 0-683-03438-3.

Goldman, J.M., Lehr, R.P., Millar, A.B., Silver, J.R. 1987. An electromyographic study of the abdominal muscles during postural and respiratory maneuvers. Journal of Neurology, Neurosurgery and Psychiatry. 50:866-9

Goubert, L., Crombez, G., Bourdeaudhuij, I. 2004. Low back pain, disability and back pain myths in a community sample: prevalence and interrelationships. European Journal of Pain. 8:385-94

Graves, J.E., Pollock, M.L., Carpenter, D.M., Leggett, S.H., Jones, A., MacMillan, M., Fulton, M. 1981. Quantitative assessment of full range-of-motion isometric lumbar extension strength. Spine. 15:289-94

Guyton, A.C., Hall, J.E. 1997. Human Physiology and Mechanisms of Disease. 6<sup>th</sup> Edition. W.B. Saunders Company. ISBN 0-7216-3299-8.

Hansen, F., Bendix, T., Skov, P., Jensen, C., Kristensen, J., Krohn, L., Schioeler, H. 1993. Intensive, dynamic back-muscle exercises, conventional physiotherapy, or placebo-control treatment of low back pain. A randomized observer-blind trial. Spine. 18:98-108.

Harms-Ringhdal, I. 1993. Muscle strength. International perspectives in Physiotherapy. Churchill-Livingston. p243. ISBN 0-443-04336-1.

Hendler, N., Kozikowski, J.G., Morrison, C. and Sethuraman, G. 1995. Diagnosis and Management of Sacroiliac Joint Disease. Journal of the Neuromusculoskeletal System 3:169-174.

Herzog, W., Scheele, D., Conway, P.J. 1999. Electromyographic responses of back and limb muscles associated with spinal manipulative therapy. Spine. 24:146-152

Herzog, W., Conway, P.J., Zhang, Y.T., Gal, J., Guimaraes, A.C. Reflex responses associated with manipulative treatments on the thoracic spine: A pilot study. Journal of Manipulative Therapy and Physiological Therapeutics. 18:233-236

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

Hides, J.A., Richardson, C.A., Jull, G.A. 1996. Multifidus muscle recovery is not automatic following acute first episode low back pain. Spine. 21:2763-9

Hides, J.A., Richardson, C.A., Jull, G.A. 2001. Long term effects of specific stabilising exercises for first –episode low back pain. Spine. 26:243-248

Hodges, P., Richardson, C. 1996. Inefficient muscle stabilisation of the lumbar spine associated with low back pain: a motor control evaluation of transeverse abdominis. Spine. 21:2640-50

Hodges, P.W., Richardson, C.A., Jull, G. 1996. Evaluation of the relationship between laboratory and clinical tests of transverse abdominal function. Physiotherapy Research International, 1:30-40.

Hodges, P., Kaigle Holm, A., Holm, S., Ekstrom, L., Cresswell, A., Hansson, T. 2003a. Intervertebral stiffness of the spine is increased by evoked contraction of the transversus abdominis and the diaphragm: in vivo porcine studies. Spine. 28:2594-601

Hodges, P.W., Moseley, G.L., Gabrielsson, A.H., Gandevia, S.C. 2003b. Acute experimental pain changes postural recruitment of the trunk muscle in pain free humans. Experimental Brain Research, 151:262-71

Hopkins, J.T., Ingersoll, C.D., Edwards, J.E., and Cordova, M.L. 2000. Changes in Soleus Motorneuron Pool Excitability after Artificial Knee Joint Effusion. Archive of Physical Medicine Rehabilitation. 81:1199-1203.

Hopkins, J.T. and Ingersoll, C.D. 2000. AMI the Limiting Factor. Journal of Sport Rehabilitation, 9:135-159.

Hultman, G., Nordin, M., Saraste, H., Ohlson H. 1993. Body composition, endurance, strength, cross-sectional area, and density of MM erector spinae in men with and without low back pain. Journal of Spinal Disorders. 6:114-23

Hurley, M.V., Jones, D.W. and Newham, D.J. 1994. Arthrogenic Quadriceps Inhibition and Rehabilitation of Patients with Extensive Traumatic Knee injuries. Clinical Science, 86:305-310

Ingersoll, C.D., Palmieri, R.M. and Hopkins, J.T. 2003. A joint Dilemma (on Line) Los Angeles: The Interdisciplinary Journal of Rehabilitation.  
<http://www.rehabpub.com/features/1022003/6.asp> (Accessed 31/7/2003)

Iyer, M.B., Mitz, A.R. and Winstein, C. 1999. Motor 1: Lower Centers. In: Cohen, H. (Ed). Neuroscience for Rehabilitation 2<sup>nd</sup> Edition. Philadelphia: Lippincott, Williams and Wilkins. ISBN 0-397-55465-6.

Jacobs, S.E. and Lowe, D.L. 1999. The Measurement of Clinical Pain Intensity. A comparison of six methods. Pain. 27:117-126.

Jacobs, S.E. and Lowe, D.L. 1999. Somatic Senses 1: The Anterolateral System. In: Cohen, H. (ed). Neuroscience for Rehabilitation. 2<sup>nd</sup> ed. Philadelphia: Lippincott Williams and Wilkins. ISBN 0-397-55465-6.

Jeffries, B. 1988. Facet joint injections. Spine : State of the art reviews. 2:409-417

Jones, L.A. 1999. Somatic Senses 3: Proprioception. In: Cohen, H. (ed) Neuroscience for Rehabilitation. 2<sup>nd</sup> Edition, Philadelphia: Lippincott, Williams and Wilkins. ISBN 0-0683-55465-6.

Jorgensen, K. 1997. Human trunk extensor muscles physiology and ergonomics. Acta Physiol Scand Suppl 637:1-58. August Krogh Institute, University of Copenhagen.

Keller, T.S., Colloca, C.J. 2000. Mechanical force spinal manipulation increases trunk muscle strength assessed by electromyography: a comparative clinical trial. Journal of Manipulative and Physiological Therapeutics. 23:585-95

Kellett, K., Kellett, D., Nordholm, L. 1991. Effects of an exercise program on sick leave due to back pain. Journal of Physical Therapy. 71:283-293

Kerr, S., Frank, J.W., Shannon, H.S., Norman, R.W.K., Wells, R.P., Neumann, W.P., Bombardier, C., and the OUBPS Group. 2001. Biomechanical and psychosocial risks factors for low back pain at work. American Journal of Public Health. 91:1069-1075

Kingsley, R.E. 1996. Concise Text of Neuroscience. Baltimore: Williams and Wilkens. ISBN 0-683-18119-X.

Kirkaldy-Willis, W.H. and Burton, C.V. 1992. Managing low back pain. 3<sup>rd</sup> Edition. New York USA: Churchill Livingstone Inc. p195, 243, 291.

Klenerman, L., Slade, P.D, Stanley, M., Pennie, B., Reilly, J.P., Atchison, L.E, Troup, J.D.G., Rose, M.J. The prediction of chronicity of patients with an acute attack of low back pain in general practice setting. 1995. Spine. 20:478-84

Koumantakis, G.A., Watson, P.J., Oldham, J.A. 2005. Trunk muscle stabilisation training plus general exercise versus general exercise only. Randomised controlled trial of patients with recurrent low back pain. Journal of Physical Therapy. 85:209-225

Lahad, A., Malter, A.D., Berg, A.O., Deyo, R.A. The effectiveness of four interventions for the prevention of low back pain. 1994. Journal of the American Medical Association. 272:1286-1291

Lariviere, C., Gravel, D., Gagnon, D., Arsenault, A.B., Loisel, P., Lepage, Y. 2003. Back strength cannot be predicted accurately from anthropometric measures in subjects with and without chronic low back pain. Clin Biomech. 18:473-9

Lariviere, C., Gagnon, D., Gravel, D., Arsenault, A.B. 2007. The assessment of back muscle capacity using intermittent static contractions. Part 1-Validity and reliability of electromyographic indices of fatigue. Journal of Electromyography and Kinesiology.

Lehman, G.J., McGill, S.M. 2001. Spinal manipulation causes variable spine kinematic and trunk muscle electromyographic responses. Clinical Biomechanics. 16:293-9

Levangie, P.K. and Norkin, C.C. 2001. Joint Structure and Function. 3<sup>rd</sup> Edition. Philadelphia: F. A. Davies Company.

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

Linton, S.J., Buer, N., Vlaeyen, J.W.S., Hellsing, A. Are fear avoidance beliefs related to the inception of an episode of back pain? A prospective study. 2000. Psychol Health. 14:1051-9

Linton, S.J., Hallden, K. 1998. Can we screen for problematic back pain? A screening questionnaire for predicting outcome in acute and subacute back pain. Clin J Pain. 14:209-15

Linton, S.J., van Tulder, M.W. 2001. Prevention interventions for back and neck pain problems: what is the evidence? Spine. 26:2103-2113

McKinnon, M.E., Vickers, M.R., Ruddock, V.N., Townsend, J., Meade, T.W. 1997. Community studies of the health service implications of low back pain. Spine. 22:2161-6

Mannion, A.F. 1999. Fiber type characteristics and function of the human paraspinal muscles: normal values and changes in association with low back pain. Journal of Electromyography and Kinesiology. 9:363-377.

Magee, D.J. 1992. Orthopaedic Physical Assessment. Philadelphia, USA: ISBN 0-7216-9352-0.

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

Manga, P., Angus, D.E., Papadopoulos, 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., Murphey, B.A. 2005. Core stability exercises on and off a Swiss ball. Archives of Physical Medicine and Rehabilitation. 86:242-249

Mathews, R. 1995. 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. Technikon Natal, Berea, Durban, South Africa.

Matkovich, G. 2004. The Immediate Effect of Sacroiliac Manipulation on Hip Strength in Patients Suffering from Chronic Sacroiliac Syndrome. Masters Degree in Chiropractic. Durban Institute of Technology, South Africa.

McCullach, J. and Transfeldt, E. 1997. Macnab's Backache. 3<sup>rd</sup> Edition. Baltimore. Williams and Wilkins. p180-181. ISBN 0-683-05797-9.

McRae, R. 2004. Clinical Orthopaedic Examination. 5<sup>th</sup> Edition. Churchill Livingstone, Elsevier Ltd. ISBN 0 443 07408 9

Meeker, W.C., Haldeman, S. 2002. Chiropractic: a profession at the crossroads of mainstream and alternative medicine. Annals of Internal Medicine. 136:216-227

Melzack and Wall 1965 – Pain Mechanisms-A New Theory.

Moore, K. L. 1992. Clinically Oriented Anatomy. 3<sup>rd</sup> Edition. Lippincott: Williams and Wilkens. ISBN 0-683-06141-0

Moore, K.L. 1999. Clinically Oriented Anatomy 4<sup>th</sup> Edition Lippincott: Williams and Wilkens. 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. 8:141-144.

Mouton, J. 1996. Understanding Social Research. 3<sup>rd</sup> Edition Pretoria, Van Schaik Publishers. ISBN 0 627 02163 8.

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

Nachemson, A. 1976. The lumbar spine: an orthopedic challenge. Spine 1: 59-76.

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



Nansel, D.D., Waldorf, T. and 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: 91-95.

O'Sullivan, P.B., Twomey, L.T., Allison, G.T. 1997. Evaluation of specific stabilising exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylosis or spondylolisthesis. Spine. 22:2259-2967

Papageorgiou, A.C., Rigby, A.S. 1991. Review of UK data on the rheumatic diseases-7. Low back pain. British Journal of Rheumatology. 30:208-10

Picavet, H.S.J., Schouten, J.S.A.G. Musculoskeletal pain in the Netherlands: Prevalences, consequences and risk groups, the DMC study. 2003. Pain. 102:167-78

Patel, A.T. and Ogle, A.A. 2000. American Family Phycician. 61(6).

Pickar, J.G. 2002. Neurophysiological effects of spinal manipulation. Spine. 2:357-71

Plaughter, G. 1993. Textbook of Clinical Chiropractic – A specific Biomechanical approach. Baltimore. Williams and Wilkins. p 525. ISBN 0-683-06897-0.

Porter, J.L. and Wilkinson, A. 1997. Lumbar-Hip Flexion Motion. Spine, 22(13):1508-1514.

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

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No. 16 March 2002

Radebold, A., Cholewicki, J., Panjabi, M.M., Patel, T.C. 2000. Muscle response patten to sudden trunk loading in healthy individuals and in patients with chronic low back pain. Spine. **25**:947-54

Rainville, J., Hartigan, C., Jouve, C. and Martinez, E. 2003. The Influence of Intense Exercise-based physical therapy program on back pain anticipated before and induced by physical activities. The Spine Journal. 176-183.

Redwood, D. 1997. Contemporary Chiropractic. Churchill Livingston, New York. ISBN 0443078092.

Richardson, C.A., Jull, G.A., Hodges, P.W., Hides, J.A. 1999. Therapeutic Exercise for Spinal Segmental Stabilisation and Low Back Pain: Scientific basis and clinical approach. Edinburgh: Churchill Livingstone.

Richardson, C.A., Jull, G.A. 1995. Muscle control-pain control. What exercises would you prescribe? Manual Therapy. 1:2-10

Risch, S.V., Norvell, N.K., Pollock, M.L., Risch, E.D., Langer, H., Fulton, M., Graves, J.E., Leggett, S.H. 1993. Lumbar strengthening in chronic low back pain patients. Spine. **18**:232-238

Robertson, N. 2005. A Cross-sectional cohort study of core stability muscle activation and endurance in elite male athletes and its link with mechanical lower

back pain. Master's Degree in Technology: Chiropractic Dissertation. Technikon Natal, Berea, Durban, South Africa.

Saal, J.A., Saal, J.S. 1989. Non operative treatment of herniated lumbar disc with radiculopathy: An outcome study. Spine. 14:431-437

Sakomoto, N., Yamashita, T., Takebayashi, T., Sekine, M. and Ishii, S. 2001. An Electrophysiologic Study of Mechanoreceptors in the Sacroiliac Joint and Adjacent Tissues. Spine, 26:E468-E471.

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

Schaefer, R.C. and Faye, L.J. 1989. Motion palpation and Chiropractic technique: Principles of dynamic Chiropractic. 1<sup>st</sup> Edition. California, USA: The Motion Palpation Institute. p211-216, 256-259,270.

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

Snell, R.S. 1997. Clinical Neuroanatomy for Medical Students. 4<sup>th</sup> Ed. Baltimore: Lippincott Williams and Wilkins. ISBN 0-316-80103-8.

Spencer, J.D., Hayes, K.C. and Alexander, I.J. 1984. Knee Joint Effusion and Quadriceps Reflex Inhibition in Man. Archives of Physical Medicine and Medicine Rehabilitation. 65:171-177.

SPSS Inc. Chicago, Ill, USA. Version 13.

Stabiliser Manual Chatanooga Group Inc, 4717 Adams Road Hixon TN 37343, USA.

Stata. StatCorp.LP, Texas, USA. Version . 9.0 for Windows.

Stagg, K. 2006. A pragmatic clinical trial investigating the efficacy of low-level laser therapy as a part of the palliative management of the hand symptoms of rheumatoid arthritis. Master's Degree in Technology: Chiropractic Dissertation. Durban University of Technology, Berea, Durban, South Africa.

Strohl, K.P., Mead, J., Banzett, R.B., Loring, S.H., Kosch, P.C. 1981. Regional differences in abdominal muscle activity during various maneuvers in humans. Journal of Applied Physiology. 51:1471-6.

Symmonds, B.P., Herzog, W., Leonard, T., Nguyen, H. 2000. Reflex responses associated with the activator treatment. Journal of Manipulative and Physical Therapy. 23:155-159

Suter, E., Herzog, W., Conway, P.J. and Zang, Y.T. 1994. Reflex Response Associated with Manipulative Treatment in the Thoracic Spine. Journal of the Neuromusculoskeletal System. 2:123-130.

Travell, J.G., Simons, D.G. and Simons, L.S., 1999. Myofascial Pain and Dysfunction. 2<sup>nd</sup> Edition ISBN 0-683-08363-5.

Urquhart, D.M., Hodges, P.W., Allen, T.J., Story, I.H. 2005. Abdominal muscle recruitment during a range of voluntary exercises. Journal of Manual Therapy. 10:144-153

Van Dieen, J.H., Selen, L.P., Cholewicki, J. Trunk muscle activation in low back pain patients: an analysis of the literature. Journal of Electromyography and Kinesiology. 13:333-51

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 Poppel, M.N.M., Koes, B.W., Smid, T., Bouter, L.M. 1997. A systemic review of controlled clinical trials on the prevention of low back pain in industry. Occupational and Environmental Medicine. 54:842-847

van Tulder, M., Koes, B., Bombardier, C. Low Back Pain. 2002. Best Practice and Research Clinical Rheumatology. 5:761-775

van Tulder, M, Koes, B.W., Benson, H., Wright, A. Conservative treatment of acute and chronic non-specific low back pain-a systemic review of randomised controlled trials of the most common interventions. Spine. 22:2128-56

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

Wood, L. 1998. Physiology of Pain. In: Kitchen, S., Bazin, S. (ed) 1996. Clayton's Electrotherapy. 10<sup>th</sup> Edition. London: W.B Saunders. ISBN 0-7020-1762-0.

**APPENDIX 1**

**Advertisement**

**Do You Exercise Less Than  
30 Minutes a day?**

**AND  
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 Renee on  
031-2042205 / 2512 / 083 6390917**



## **APPENDIX 2**

### **Randomisation Table**



### Randomisation Table

1. a
2. a
3. b
4. a
5. b
6. b
7. a
8. b
9. a
10. a
11. b
12. b
13. a
14. b
15. a
16. b
17. b
18. a
19. b
20. a
21. b
22. a
23. b
24. b
25. a
26. b
27. a
28. a
29. b
30. a
31. a
32. b

**APPENDIX 3**

**Letter of Informed Consent**

# INFORMED CONSENT FORM

(To be completed by participant / subject )

Date \_\_\_\_\_ :

---

## Title of research project

The relative effect of manipulation and core rehabilitation in the treatment of acute mechanical low back pain in sedentary participants

---

Name of supervisor: Dr C Korporaal(M Tech Chiropractic, CCFC, CCSP, ICCSD)  
Tel: 031-2042102

---

Name of research student: Renee Higgs  
Tel: 031-2042205 Cell:0836390917

---

### 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?<br>at any time<br>without having to give any a reason for withdrawing, and<br>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:

Participant /Subject Name: \_\_\_\_\_ Signature: \_\_\_\_\_

Witness Name: \_\_\_\_\_ Signature: \_\_\_\_\_

Research Student Name: \_\_\_\_\_ Signature: \_\_\_\_\_

**APPENDIX 4**

**Letter of Information**

## 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 sedentary participants.

### Name of Research student:

Renee Higgs

Contact number: 031 2042205 / 083 6390917

### Name of Research Supervisor:

Dr. C Korporaal

Contact number: 031 2042611

(M Tech: Chiropractic, CCFC, CCSP, and ICCSD)

### Name of Research Co-supervisor:

Dr. R White

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 new treatment approaches for participants who perform little or no daily exercise and who are suffering from acute low back pain.

Thirty people will be required to complete this study. Each individual will have a standard clinical treatment, which will include either an adjustment or 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. 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. Specific measurements will be taken regarding your low back pain and your core stability will be assessed.

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 adjustment, it is unlikely to cause any adverse side effects. There may also be some slight discomfort as a result of the rehabilitation exercises in the form of mild muscle stiffness.

### Remuneration and costs:

- All treatments will be free of charge and subjects taking part in the study will not be offered any other form of remuneration for taking part in the study.
- All participant 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 participants 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,

---

**Renee Higgs (Research Student)**

---

**Dr. C. Korporaal (Supervisor)**

# **APPENDIX 5**

## **Case History**

**DURBAN UNIVERSITY OF TECHNOLOGY**  
**CHIROPRACTIC DAY CLINIC**  
**CASE HISTORY**

Participant: \_\_\_\_\_ 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:



**Intern's Case History:**

**1. Source of History:**

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

**3. Present Illness:**

	<b>Complaint 1</b>	<b>Complaint 2</b>
< 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. xrays
- < 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 6**

**Physical Examination**

**Durban University of Technology**  
**PHYSICAL EXAMINATION: SENIOR**

Participant Name : \_\_\_\_\_ File no : \_\_\_\_\_ Date : \_\_\_\_\_  
 Student : \_\_\_\_\_ Signature : \_\_\_\_\_

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

<b>GENERAL EXAMINATION:</b>	
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 7**

**Lumbar Regional**

**REGIONAL EXAMINATION - LUMBAR SPINE AND PELVIS**

Participant: \_\_\_\_\_ 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 (8cm)  
 Bony and Soft Tissue Contours

**GAIT:**

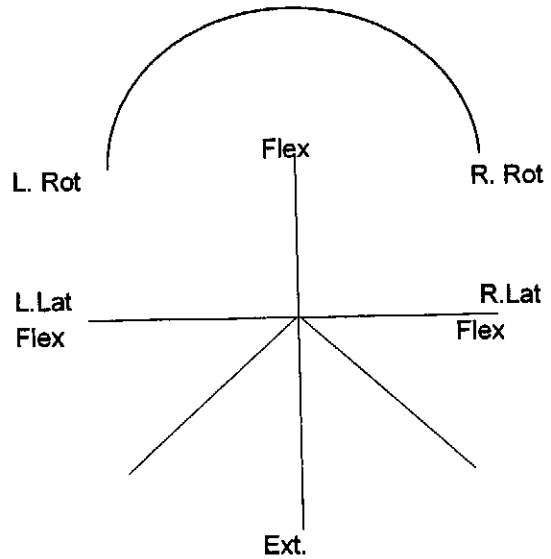
Normal walking  
 Toe walking  
 Heel walking  
 Half squat

**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

SLR	Degree	LBP?	Location	Leg pain	Buttock	Thigh	Calf	Heel	Foot	Braggard

	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		

**SITTING:**

Spinous Percussion  
 Valsalva

Lhermitte

TRIPOD	Degree	LBP?	Location	Leg pain	Buttock	Thigh	Calf	Heel	Foot	Braggard
SI, +, ++										

Slump 7 test										

**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			

**NON ORGANIC SIGNS:**

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				Proprioception		
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 8**

## **SOAPE Note**

DURBAN UNIVERSITY OF TECHNOLOGY

Participant Name: \_\_\_\_\_ File #: \_\_\_\_\_ Page: \_\_\_\_\_

Date: \_\_\_\_\_ Visit: \_\_\_\_\_ Intern: \_\_\_\_\_  
Attending Clinician: \_\_\_\_\_ Signature: \_\_\_\_\_

S: Numerical Pain Rating Scale (Participant) 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 (Participant) 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 (Participant) 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 9**

## **Numerical Pain Rating Scale**

**NRS Pain Rating Scale**

**Participant 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

**APPENDIX 10**

**Data Capture Sheet**

**Participant Name**.....

**Algometer Readings:**

	Date	Location			
Reading 1					
Reading 2					
Reading 3					

**Stabiliser Readings:**

	Date	Readings	Ave. Reading	Time
Reading 1				
Reading 2				
Reading 3				

**EMG Readings:**

	Date	Location	Reading
Reading 1			
Reading 2			
Reading 3			

## **APPENDIX 11**

### **Participant Diary**



## Participant Diary

**Participant Name:**.....

### WEEK 1

	Date	Sign	Date	Sign	Date	Sign
<b>Exercise 1</b>						
<b>Exercise 2</b>						
<b>Exercise 3</b>						
<b>Exercise 4</b>						

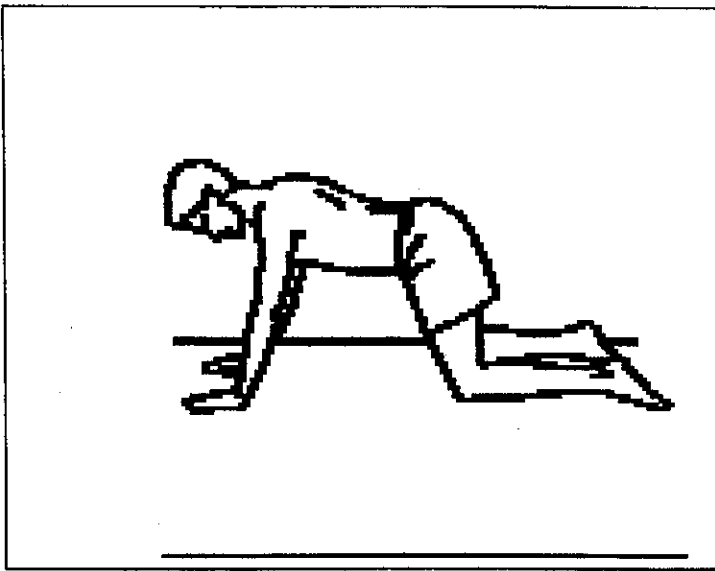
### WEEK 2

	Date	Sign	Date	Sign	Date	Sign
<b>Exercise 1</b>						
<b>Exercise 2</b>						
<b>Exercise 3</b>						
<b>Exercise 4</b>						

## **APPENDIX 12**

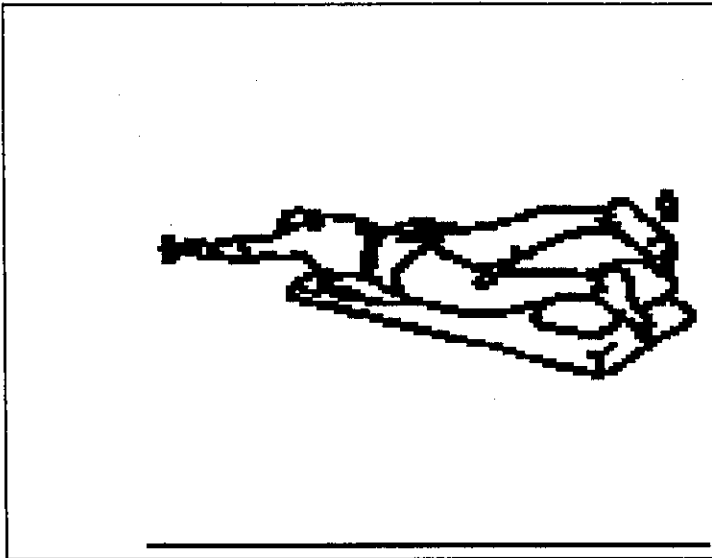
### **Participants Exercise Sheet**

### Exercise 1



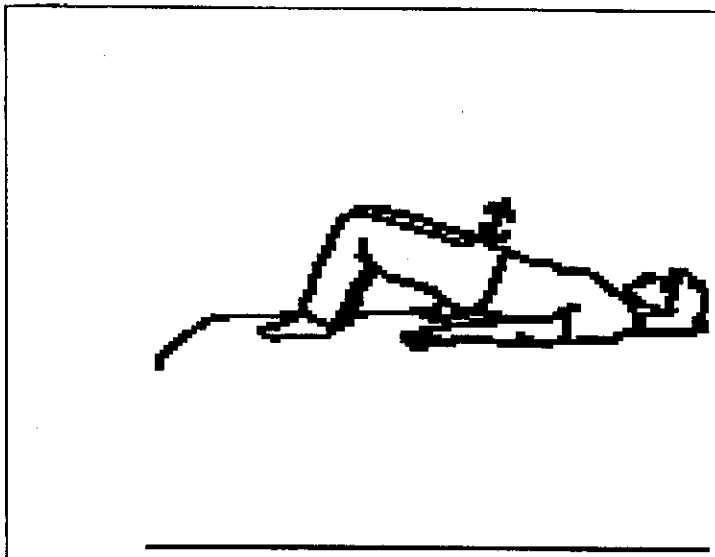
- Crawling position.
- Pull your stomach in.
- Hold for 5 seconds.
- Repeat 10 times morning and night

### **Exercise 2:**



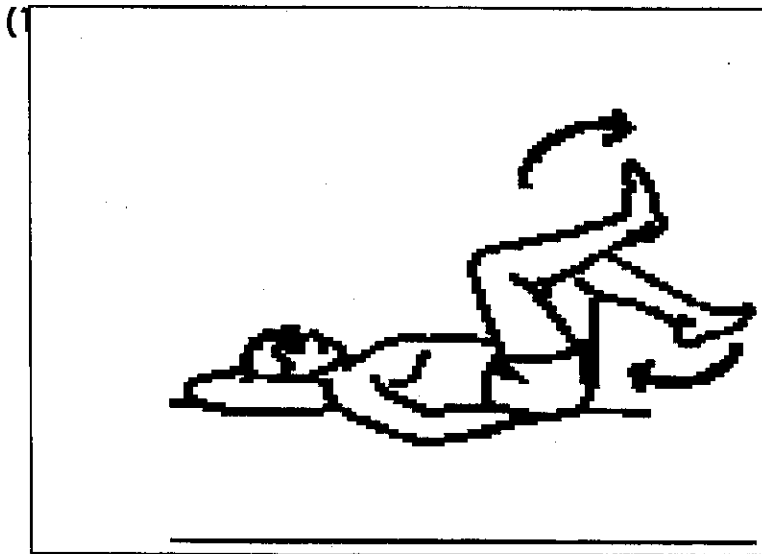
- Lying face down with your arms above your head on the floor (you can have a pillow under your stomach and one under your ankles)
- Lift opposite arm and leg approximately 20cm off the floor and stretch.
- Hold the stretching for approximately 5 seconds – relax, and repeat with other side.
- Repeat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

### **Exercise 3:**



- 
- Lying on your back with 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 morning and night

### **Exercise 4:**



#### **A)**

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

#### **B)**

- Lying on your back with hands supporting your pelvis.
- Make a cycling movement with both your legs for 1 minute-morning and night (Physio Tools Ltd)

## **APPENDIX 13**

### **Core Stability Assessment Techniques**

## Core stability assessment techniques using the Stabiliser Biofeedback Device

### The prone test for transversus abdominus 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.
  - Participant must attempt to maintain this contraction for the set time period for this test, which is 40 seconds.
  - Measurement of time at which the participant can no longer hold the contraction at the baseline level (70mmHg – 6 to 10 mmHg), within the set time period for the test (40 seconds).
  - Measurement of the change in mmHg from the baseline level (70mmHg – 6 to 10mmHg) up to the end of the set time period (40 seconds).
- Measurement of the rate at which the pressure changes from the time at which it begins to change up to the end of the set time period of the test (40 seconds).

### Supine position for training transversus abdominus

- Place 3-chamber pressure cell under the lumbar spine and inflate to a baseline of 40 mmHg.
- Draw in the abdominal wall without moving the spine or pelvis.
- Pressure should remain at 40 mmHg; i.e. no movement of the spine.
- Hold for 10 seconds; breathe normally.
- Participant must attempt to maintain this contraction for the set time period for this test, which is 40 seconds.
- Measurement of time at which the participant can no longer hold the contraction at the baseline level (40 mmHg), within the set time period for the test (40 seconds).
- Measurement of the change in mmHg from the baseline level (40 mmHg) up to the end of the set time period (40 seconds).
- Measurement of the rate at which the pressure changes from the point at which it begins to change up to the end of the set time period of the test (40 seconds).

### 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:**

- Participant relaxes abdominal wall (which examiner can feel by gently palpating abdomen).
- Participant 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.
- Participant 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 14**

### **Roland Morris Low Back Pain and Disability Questionnaire**

## 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 its quality.