


# The immediate effect of thoraco-lumbar spinal manipulation compared to lower lumbar spinal manipulation on core muscle endurance and activity in patients with mechanical low back pain.

By

Stuart M Murray


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

I, Stuart Michael Murray, do declare that this dissertation is representative of my own work in both conception and execution.

  
\_\_\_\_\_

Date: 22/7/9

APPROVED FOR FINAL SUBMISSION:

  
\_\_\_\_\_

Date 22/7/9

Supervisor: Dr. A. Jones M.Dip:Chiropractic (DUT), MMed Sci: sports medicine (UKZN)

## **Dedication**

*I dedicate this dissertation to my family:*

*Thank you for having the belief in me and giving me the drive to raise myself to new heights.*

*Mom, thank you for the sacrifices you have had to make for me; you are an amazing woman.*

*Dad, thanks for always being so proud of me and for all the help through my student days; it would have been a lot harder without you.*

*Ivor, you have always been there for me and I am eternally grateful for your generosity and kindness.*

## **Acknowledgements**

*Dr Jones, thank you for all your assistance with this project and all the chats about cycling; they will be missed.*

*Kate, you have been the most patient and loving person in my life. Thank you for everything you have contributed in my life.*

*The Wiseman's; thank you for being my family away from home; you are incredibly kind and I will always be grateful.*

*Thanks to Pat and Linda for their patience with me in the Clinic and all their hard work.*

*Thank you to all the clinicians for their valuable input.*

*Uncle Fred, thank you for your help with the proof reading, it's very much appreciated.*

*The department of Human Sciences, especially Mr Walters, thank you for allowing me to use your equipment, it would not have been possible without your help.*

*Thank you to all people that participated in the research, you where fantastic.*

*To my lecturers; it was a long hard road, but it was a good learning experience throughout, thank you.*

*To all the true friends I've made in Chiro; this has been great, but all great things must come to an end; I look forward to all our future adventures together. I will miss you all.*

# **Abstract**

## **Background**

Through the literature review it has become apparent that low back pain is a very real problem in most societies. It has been suggested that there is enough evidence to prove the relationship between low back pain and local muscle dysfunction and that focus in management of these patients should be the rehabilitation of these muscles by exercise. Literature suggests that optimal core muscle strength, control and endurance working synergistically with the rest of the neuromusculoskeletal system is necessary for lumbar spine stability .

Arthrogenic Muscle Inhibition is caused by distension and/or damage of a joint and is thought to disable the muscle from contracting all its muscle fibres. When a joint is injured it is thought that AMI causes muscle weakness, which in turn hampers the rehabilitation process of that joint despite complete muscle integrity. Spinal manipulative therapy has been shown to alter the excitability of spinal muscle motor neurons due to the stimulation of mechanoreceptors in the joint capsules suggesting that SMT could be a means to remove this inhibitory action. The literature supports the hypothesis that a decrease in the neurological deficit caused by AMI may result in a faster recovery rate.

## **Aims**

The aim of this study is to determine the immediate effect of thoraco-lumbar spinal manipulation compared to lower lumbar spinal manipulation on core muscle endurance and activity in patients with mechanical low back pain by assessing the correlation between the objective and subjective measures.

## **Method**

A prospective, convenience sample with purpose allocation (pre /post) clinical trial was used as the sampling method. Thirty participants were placed in two groups, group one and group two, of fifteen people each. Group one underwent spinal

manipulative therapy between L4 and S1 spinal levels. Group two underwent spinal manipulative therapy in between T8 and L1 spinal levels. The objective and subjective testing was done pre- and post-intervention. The objective data was that of a surface EMG attached bilaterally over the internal oblique as well as a prone abdominal draw in biofeedback test. The subjective data included a pain numerical rating scale (0-100).

### **Results**

The results showed to partially favour group two (thoraco-lumbar), in both increased endurance time that would prove that AMI does in fact inhibit the transversus abdominis and obliques internus, thus it would hinder the rehabilitative process. Some of the statistics were not in favour of the aims, as there was no difference in the effect of group one or two on the NRS, as both improved consistently. It would be recommended that use be made of fine-wire EMG for testing the activity in both the obliques internus and the transversus abdominis, which would allow for more consistent readings, thus adding strength to the research.

<b><u>Table of Contents</u></b>	<b><u>Page</u></b>
<b><u>Chapter 1 - Introduction</u></b>	
1.1 Introduction	1
1.2 Aims and hypotheses	3
1.3 Rationale	4
<b><u>Chapter 2 - Review of related literature</u></b>	
2.1 Introduction	6
2.2 Low back pain	6
2.2.1 Epidemiology	6
2.2.2 Aetiology and classification	7
2.3 Spinal instability	9
2.3.1 Lumbar segmental stability	10
2.3.2 Dysfunction of the transversus abdominis	14
2.4 Relevant neurophysiology	14
2.4.1 Joint and joint receptors	14
2.4.2 Transmission to the spinal cord	16
2.4.3 Ascending pathways	17
2.4.4 Descending pathways	17
2.4.4.1 Corticospinal tract	18
2.4.4.2 Vestibulospinal tract	18
2.4.4.3 Rubrospinal tract	18
2.4.5 The interneuron	18
2.5 Arthrogenic Muscle Inhibition	20
2.5.1 Definition	20
2.5.2 Causes of AMI	20
2.5.3 Clinical implications of AMI	21
2.5.4 Measurement of AMI	22
2.5.5 Therapeutic interventions that reduce AMI	22
2.5.6 The proposed neurophysiological mechanism of spinal manipulation on AMI	23
2.5.7 Effect of spinal manipulative therapy on trunk muscles	23
2.6 Relevant anatomy	24

2.6.1 Bony anatomy	24
2.6.1.1 Thoracic spine	24
2.6.1.2 Lumbar spine	25
2.6.2 Relevant musculature	25
2.6.2.1 Transversus abdominis	25
2.6.2.2 Internal oblique	26
<b>2.7 Summary</b>	<b>27</b>

## **Chapter 3 - Materials and methods used**

<b>3.1 The study design</b>	<b>29</b>
3.1.1 Research design	29
3.1.2 Sampling allocation	29
3.1.3 Sampling size	29
3.1.4 Sampling method	29
3.1.5 Participant screening	30
3.1.6 Inclusion and exclusion criteria	30
3.1.6.1 Inclusion criteria	30
3.1.6.2 Exclusion criteria	31
<b>3.2 Intervention method</b>	<b>32</b>
<b>3.3 Data collection and materials used</b>	<b>34</b>
3.3.1 Objective data	34
3.3.1.1 Pressure biofeedback unit	34
3.3.1.2 Surface electromyograph	36
3.3.2 Subjective data	36
3.3.2.1 Pain numerical rating scale (NRS)	36
<b>3.4 Statistical analysis</b>	<b>37</b>

## **Chapter 4 - Statistical methodology and results**

<b>4.1 Statistical methodology</b>	<b>38</b>
<b>4.2 Results</b>	<b>38</b>
4.2.1 Demographic and baseline comparison between treatment groups	38
4.2.2 Test endurance (time)	40
4.2.3 Test endurance (mmHg)	41
4.2.4 Left EMG	42
4.2.5 Right EMG	43

4.2.6 NRS	44
4.2.7 Lumbar group	46
4.2.8 Thoraco-Lumbar group	46
<b>4.3 Conclusion</b>	<b>47</b>
<b><u>Chapter 5 - Discussion</u></b>	
<b>5.1 Introduction</b>	<b>48</b>
<b>5.2 Discussion</b>	<b>48</b>
5.2.1 Demographics	48
5.2.2 Inter-group analysis	49
5.2.2.1 Test muscle endurance	49
5.2.2.2 EMG data	50
5.2.2.3 NRS	51
5.2.3 Intra-group analysis	51
5.2.3.1 Lumbar group	51
5.2.3.2 Thoraco-lumbar group	51
<b><u>Chapter 6 - Conclusions and recommendations</u></b>	
<b>6.1 Conclusions</b>	<b>53</b>
<b>6.2 Study limitations</b>	<b>54</b>
<b>6.3 Recommendations</b>	<b>54</b>
<b><u>References</u></b>	<b>56</b>



## **Table of Tables**

Page

### **Chapter 2**

**Table 2.1:** Parts and distinctive characteristics of the thoracic vertebrae 24

**Table 2.2:** Parts and distinctive characteristics of the lumbar vertebrae 25

### **Chapter 4**

**Table 4.1:** Occupations of the sample ( $n = 30$ ) 39

**Table 4.2:** Comparison of demographic and baseline variables between the two groups 40

**Table 4.3:** Within and between subjects effects for test endurance (time) 41

**Table 4.4:** Within and between subjects effects for test endurance (mmHg) 42

**Table 4.5:** Within and between subjects effects for Left EMG 43

**Table 4.6:** Within and between subjects effects for Right EMG 44

**Table 4.7:** Within and between subjects effects for pain (NRS) 45

**Table 4.8:** Pearson's correlations between changes in outcomes in the Lumbar group 46

**Table 4.9:** Pearson's correlations between changes in outcomes in the Thoraco-lumbar group 47

## **Table of Figures**

Page

### **Chapter 2**

<b>Figure 2.1</b> Factors that require consideration within a biopsychosocial framework, for the diagnosis and classification of CLBP disorders.	9
<b>Figure 2.2:</b> An adaptation of the model, conceptualised by Panjabi (1992), showing the relationship between the 3 sub-systems	13
<b>Figure 2.3:</b> The injury paradigm	21
<b>Figure 2.4:</b> An illustration of the transversus abdominis	26
<b>Figure 2.5:</b> An illustration of the obliques externus muscle	27

### **Chapter 4**

<b>Figure 4.1:</b> Profile plot of mean test endurance (time) by time and group	41
<b>Figure 4.2:</b> Profile plot of mean test endurance (mmHg) by time and group	42
<b>Figure 4.3:</b> Profile plot of mean left EMG by time and group	43
<b>Figure 4.4:</b> Profile plot of mean right EMG by time and group	44
<b>Figure 4.5:</b> Profile plot of mean NRS by time and group	45

**Appendices****Page**

Appendix A – Letter of information and consent	66
Appendix B – Case history	68
Appendix C – Physical examination	73
Appendix D – Thoracic regional	74
Appendix E – Lumbar regional	77
Appendix F – SOAPE note	81
Appendix G – Pain numerical rating scale	82
Appendix H – Data Capture Sheet	83
Appendix I – Prone biofeedback abdominal draw in test procedure	84
Appendix J – Advertisement	86
Appendix K – Letter of statistics approval	87
Appendix L – Four point kneeling	88
Appendix M – Ethics clearance certificate	89

**Definitions****Motion Palpation**

Bergmann *et al.* (1993) defines palpation as the application of variable manual pressure through the surface of the body for the purpose of determining the shape, size, consistency, position, inherent motility, and health of the tissues beneath.

**Thoraco-Lumbar Spine**

For the purpose of this study this will be defined as segmental levels T8 through to L1.

**Lumbar Spine**

For the purpose of this study this will be defined as segmental levels L4 through to S1.

## **Chapter 1**

### **Introduction**

#### **1.1 Introduction**

Low back pain (LBP) dysfunction is becoming an increasing problem in the Western industrialized world and is putting a large strain on the health budget (Indahl *et al.*, 1995). The major issue is that, of those affected 5-10% become disabled and these result in 75-90% of the cost accrued due to LBP (Indahl *et al.*, 1995). For persons younger than 45 years, LBP represents the most common cause of disability and it is the third most common cause of disability in persons older than 45 years (Hills, 2006). Research indicates that mechanical back pain is largely influenced by mechanical instability of the low back (Kirkady - Willis, 1988; Nachemson, 1984).

“Arthrogenic muscle inhibition (AMI) is a presynaptic, ongoing reflex inhibition of musculature surrounding a joint following distension or damage to structures of that joint” (Hopkins and Ingersoll, 2000). AMI is a natural response designed to protect the joint from further damage (Suter *et al.*, 1999; Hopkins and Ingersoll, 2000). AMI in turn would form a perpetuating cycle of muscle inhibition leading to decreased core stability and thus increased instability which would lead to further joint dysfunction. This leads to further inhibition of muscle contraction (Hopkins and Ingersoll, 2000). Suter *et al.* (2000) stated that AMI may limit the functional recovery of the joint and muscle complex after injury, and that the early goals of treatment should be to reduce muscle inhibition to gain full recovery.

Hodges and Richardson (1996) found that core stabilisation was diminished in patients with mechanical low back pain due to insufficient muscular stabilisation of the lumbar spine by the transversus abdominis muscle. AMI gives a hypothesized explanation to these findings. The local muscles, lumbar multifidus and transversus abdominis, are responsible for segmental stabilisation (Stanford, 2002). In mechanical low back pain sufferers, it was shown that there was a decreased

endurance in the transversus abdominis muscle (Richardson and Jull, 1994; Evans and Oldrieve, 2000), which could possibly lead to further mechanical low back pain due to the inability of the muscular subsystem to stabilise the spine.

AMI can be eliminated or diminished by removing, masking, overriding or otherwise altering inhibitory interneuron activity (Ingersoll, Palmieri and Hopkins, 2003). Anything that may alter, slow or compete with mechanoreceptor feedback may be a candidate (Ingersoll, Palmieri and Hopkins, 2003). Spinal Manipulative Therapy (SMT) has been shown to alter the excitability of spinal muscle motor neurons due to the stimulation of mechanoreceptors in the joint capsules (Indahl *et al.*, 1995), suggesting that SMT could be a means to remove this inhibitory action.

Spinal mobilisation was shown to have a stimulatory effect on the obliques externus and internus when it was applied to the L5/S1 region (Ferreira *et al.*, 2007). In the same study there was no increase in the activity of the transversus abdominis muscle (Ferreira *et al.*, 2007). Boden (2002), in an unpublished study, stated that, "... retraining the core stability did have an effect on the endurance of the transversus abdominis muscle but it was not sufficient to conclude that a combined core stabilisation and manipulation program was more effective than manipulation alone." Both studies' deficits could be attributed to AMI, as innervation of the transversus abdominis arises from thoracic spinal nerves T8-T12, iliohypogastric nerve, and ilioinguinal nerve which are branches off the L1 spinal nerve (Simons, Travell and Simons, 1999). AMI may thus still occur if the SMT was not applied at the aforementioned levels. No other published studies have addressed this issue in the core muscles.

The obliques internus muscle shares its innervation with the transversus abdominis muscle, the branches of the eighth through twelfth intercostal nerves as well as branches of the iliohypogastric and ilioinguinal nerves from the first lumbar nerve (Simons, Travell and Simons, 1999). It follows that, if AMI affects the transversus abdominis, it would also do so to the obliques internus muscle. The study by Ferreira *et al.* (2007) attributed the activation of the obliques externus and internus to the torque applied to them via the side posture technique. This could be addressed by

using drop table thrusting techniques as it was found to be effective for patients with neuromusculoskeletal problems such as facet syndrome (Haldeman *et al.*, 1993 as cited in Gatterman *et al.*, 2001).

If AMI is proven to be present in patients with mechanical low back pain, it would add credence to Panjabi's theory (1992) on the three subsystems explaining the link of low back pain and weak transversus abdominis muscle (Hodges and Richardson, 1996).

## **1.2 Aims and hypotheses**

The aim of this study is to determine the immediate effect of thoraco-lumbar spinal manipulation compared to lower lumbar spinal manipulation on core muscle endurance and activity in patients with mechanical low back pain by assessing the correlation between the objective and subjective measures.

1. To evaluate the relative effectiveness of thoraco-lumbar spinal manipulative therapy compared to that of lower lumbar spinal manipulative therapy, in terms of objective findings (a test for endurance of the transversus abdominis muscle will be done via a biofeedback unit and a test of the obliques internus muscle will be done via an electromyographic apparatus).

The following Null hypothesis was set: there will be no difference in the objective outcomes of the study in either the group receiving lumbar manipulation or the group receiving thoraco-lumbar manipulation.

2. To evaluate the relative effectiveness of thoraco-lumbar spinal manipulative therapy compared to that of lower lumbar spinal manipulative therapy, in terms of subjective findings (NRS).

The following Null hypothesis was set: there will be no difference in the subjective outcomes of the study in either the group receiving lumbar manipulation or the group receiving thoraco-lumbar manipulation.

3. To compare the correlation of trends in terms of objective and subjective findings in participants with mechanical low back pain.

The following Null hypothesis will be set: there will be no correlation in the objective and subjective outcomes of the study in either the group receiving lumbar manipulation or the group receiving thoraco-lumbar manipulation.

### **1.3 Rationale**

The lack of core stability is commonly associated with mechanical low back pain as a cause, and subsequently it is thought that an increase in core strength assists in reducing low back pain. Core stability is inversely related to mechanical low back pain (Richardson *et al.*, 1996), but no study has assessed the effect of SMT, at the correct level of innervation, on the transversus abdominis and the obliques internus as this may have a stimulatory effect on the core muscles.

There has been evidence to support AMI of the lower limb and also that manipulative therapy is effective in increasing muscle strength in muscles innervated from these levels (Hillermann *et al.*, 2003; Matkovich, 2004). The level of SMT may affect the stimulatory effect on the core muscles; this study will attempt to show that the level manipulated affects the muscles innervated from that level.

Muscle inhibition due to pain is not considered to be a part of the concept of AMI (Stokes *et al.*, 1984; Young *et al.*, 1993). Contradictory to this, it has been stated that pain inhibition does play a part in AMI (Eriksson, 1981). AMI and pain may have correlating trends and this study will attempt to show whether there is, in fact, a correlation.



In the remaining chapters, the researcher will review the literature on acute LBP (Chapter 2); describe in detail the methodology of this study (Chapter 3) and present the statistics and relevant results (Chapter 4); discuss the results (Chapter 5) and present the subsequent conclusions and recommendations (Chapter 6). Thereafter, recommendations will be made for suggested improvements in the management of acute LBP.

## **Chapter 2**

### **Review of Related Literature**

#### **2.1 Introduction**

The following review of current literature relating to this research will focus on epidemiology of low back pain, core stability and its relation to low back pain, and the possible role of Arthroscopic Muscle Inhibition. Included will be a description of the relevant neurology and anatomy.

#### **2.2 Low back pain**

##### **2.2.1 Epidemiology**

Incidence is defined as the rate that healthy people in a given population develop a disease or symptom over a specific time period. Prevalence is defined as the number of people in a specific population group that develops a disease or symptom at a specific time (Borenstein *et al.*, 1995). The lifetime incidence of LBP, suggested by Koes (1991), in the Western world, is said to be between 60 and 80 percent; this is also in line with Indahl *et al.* (1995) findings of a lifetime incidence of 80%. According to Burton and Cassidy (1992), low back pain has a lifetime incidence of between 60% and 90% for any population, supporting the findings suggested by Koes (1991) and Indahl *et al.* (1995).

Although 80-90% of LBP episodes subside over a 2-3 month period, there is still a high reoccurrence of LBP (Indahl *et al.*, 1995). Every year, 3-4% of the population is temporarily disabled, and 1% of the working-age population is disabled totally and permanently because of LBP (Wheeler, 2007). LBP is second only to the common cold as a cause of lost work time; it is the fifth most frequent cause for hospitalization and the third most common reason to undergo a surgical procedure. Wheeler (2007)

showed that productivity losses from chronic LBP approach \$28 billion annually in the United States.

Indahl *et al.* (1995) estimated that the 5-10% of the patients who suffer from Chronic LBP account for 75-90% of the total cost; that accounts for \$21-25.2 billion of Wheeler's (2007) estimates. Another author suggests that a total indirect and direct cost of LBP may cost the U.S government \$100 Billion a year (Hills, 2006). This is a major drain on the health care systems of the U.S.A. South Africa's lifetime incidence of low back pain in Indian and Coloured communities was in keeping with Koes' (1991) findings, where it was found to be 78.2% and 76.6% respectively, and the prevalence was 45% and 32,6% respectively (Docrat, 1999). The effect of LBP on the South African economy has not been stated, but if one compares the figures mentioned above it would appear that it is highly likely that there would be a marked similarity.

## **2.2.2 Aetiology and classification**

Schaefer and Faye (1989:195) characterised LBP into the following types of syndromes:

1. Lumbar facet syndrome
2. Sacroiliac syndrome
3. Lumbar radicular syndrome, discogenic or mechanical in nature

The cause of these may be due to:

- Sprain/strain
- Overuse
- Poor posture
- Disuse
- Joint dysfunction, fixation or hypermobility
- Developmental anomalies
- Degenerative changes
- A combination of the above

Schaefer and Faye (1989:195)

According to Kirkaldy-Willis (1988) there are three further aspects to take into account when studying the origin of LBP.

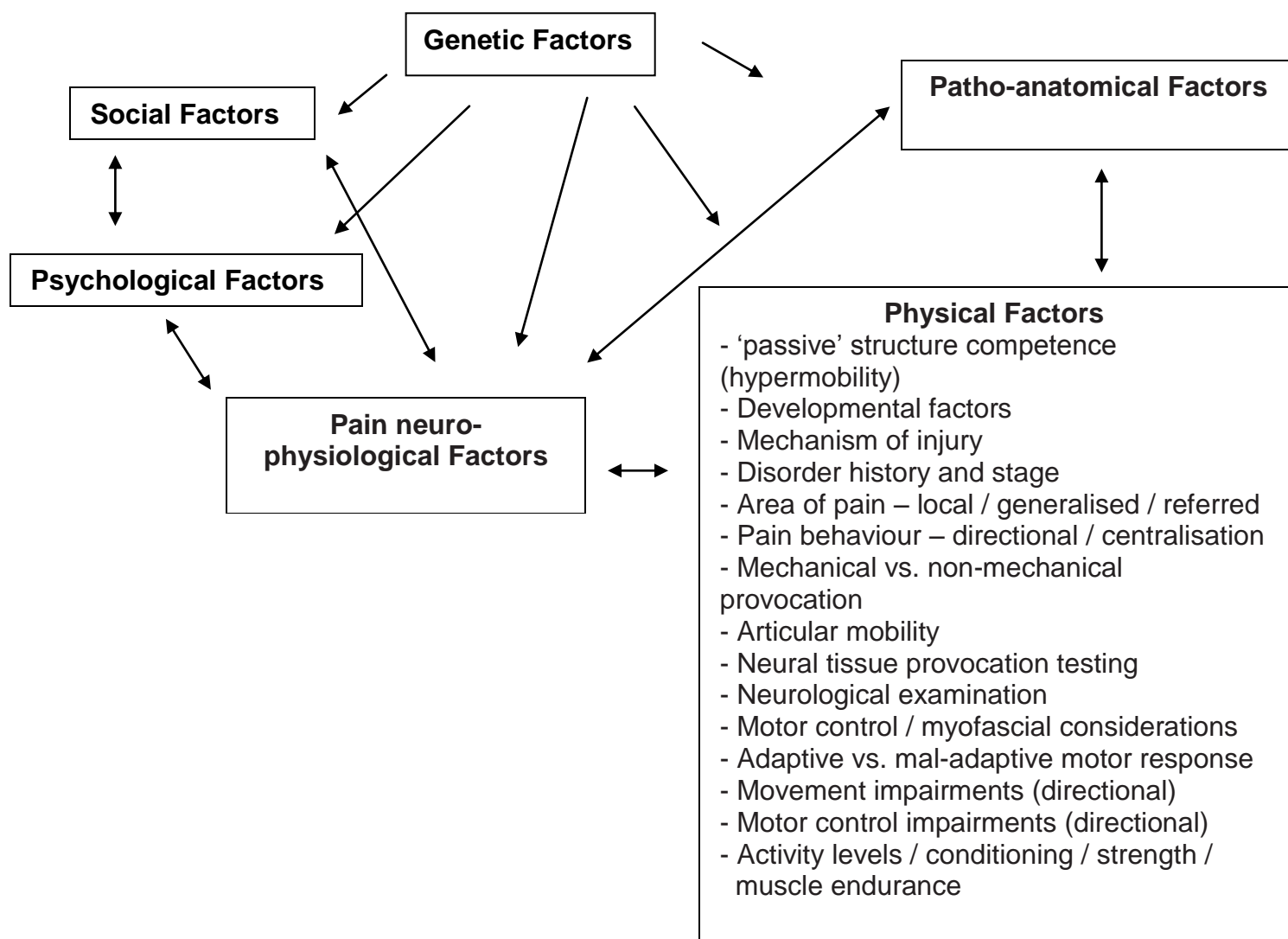
These include:

1. Emotional factors – anxiety, depression, fear, tension.
2. Changes in muscle – impaired local circulation, sustained muscle contraction, vasoconstriction, structural muscle changes and abnormal contraction.
3. Changes in the three joint complex – strains, synovitis, facet joint syndrome, degeneration and disc degeneration

With many pathological causes of low back pain understood, it is still a difficult procedure to classify and diagnose those ideas that are not pathological. The fact is that in most cases (85%) a definitive diagnosis is difficult to make (Waddel, 1995). This group is categorised, very loosely, into 'non-specific low back pain' (Dillingham, 1995).

In recent times classification systems have been researched, and a view that the diagnosis and classification of chronic low back pain (CLBP) may be the result of many factors within a biopsychosocial model, all having an interconnected effect on one another with genetic factors affecting each aspect of the model, has been adopted (O'Sullivan, 2005:242-255).

O'Sullivan's (2005) model tries to incorporate all aspects that may affect low back pain, and place them into sub-groups allowing for classification of low back pain and in turn the correct treatment. A major cause of problems arises due to physical problems, which have been expanded on in Figure 2.1.



**Figure 2.1** Factors requiring consideration within a biopsychosocial framework, for the diagnosis and classification of CLBP disorders. (Adapted from O'Sullivan, 2005)

### 2.3 Spinal instability

Segmental instability of the lumbar spine is defined as abnormal motion between two or more vertebrae (Rydevik, 1992). Nachemson (1984) suggested that special attention should be paid to a sub-group, namely that of clinical instability.

### 2.3.1 Lumbar segmental stability

The core, as described by Akuthola and Nadler (2004), is box shaped with the abdominals in the front, the gluteals and paraspinals in the back, pelvic floor and hip girdle musculature the bottom and the diaphragm the top.

As such, the core acts as a muscular corset to work as a unit to stabilise the pelvis and the body. Core stability results from highly coordinated muscle activation patterns involving many muscles, which provide support and control of the joints, and that the recruitment patterns must continually change, depending on the task (Jull *et al.*, 1993; McGill, 2003).

Lee and Vleeming (2003) created a model that explained the interactions between all the various aspects that affect spinal stability, namely 'the integrated model of function'. This system describes four subgroups that affect the lumbopelvic stability, each equally as important as the next. The four groups were:

1. Form closure (structure)
2. Force closure (forces created by myofascial action)
3. Motor control (specific timing of muscle action/inaction during loading)
4. Emotions

(Lee and Vleeming, 2003)

The model proposes that joint mechanics may be influenced by a multitude of factors, and that all these factors require attention in patient management (Lee and Vleeming, 2003). This statement is supported by Panjabi's theory on spinal stability, that many factors, passive, active and neurological affect the stabilizing system of the spine (Panjabi, 2003).

- Form closure

The term 'form closure' was first used by Vleeming *et al.* (1990) and then by Snijders *et al.* (1993) and it refers to how the joints structure, orientation and shape assist in stability and mobility (Lee and Vleeming, 2003).

- Force closure

The structures responsible for force closure are the ligaments, fascia and musculature. If the surfaces in form closure were completely and constantly compressed together, it would allow for no motion. So with force closure, a compressive force is applied at the moment of loading, relevant to the load, thus allowing for movement across the joint. The amount of force closure required depends on the individual's form closure and the quantity of the load applied (Lee and Vleeming, 2003).

The ligaments are responsible for force closure when the joint is in a closed pack position (Lee and Vleeming, 2003); the conundrum is that stability is required throughout the range of motion. The muscles that provide motion and stability in the lumbar spine can be divided into global and local muscle systems (Bergmark, 1989; Stevens *et al.*, 2006). The local muscles maintain intersegmental stabilization and the global muscles allow trunk movement and regional stabilization between thorax and the pelvis (Lee and Vleeming, 2003).

There is a difference in when these muscle groups fire. Research by Hodges and Richardson (1997), Hodges (1999) and Moseley *et al.* (2002) show that the local muscles fire in anticipation of movement and that it is not direction dependant. Whilst research by Radebold *et al.* (2000) and Hodges (2003) show that global muscles fire later than the local muscles and the contraction is direction dependant.

The muscles that fit the characteristics of local in the lumbopelvic area are the deep multifidus, transversus abdominis, diaphragm and pelvic floor muscles. The transversus abdominis was shown to be the first muscle to contract in anticipation to movement (Hodges and Richardson, 1997).

Global muscles have been described as being part of a sling system. Lee and Vleeming (2003) describes four such slings :

- posterior oblique sling contains connections between the latissimus dorsi and the gluteus maximus through the thoracodorsal fascia

- anterior oblique sling, which contain connections between the external oblique muscle, the anterior abdominal fascia, the contra lateral internal oblique abdominal muscle and the adductor of the thigh;
- longitudinal sling connects the peroneii muscles, bicep femoris, sacrotuberous ligament, the deep lamina of the thoracodorsal fascia and the erector spinae;
- lateral sling contains the primary stabilizers of the hip joint (gluteus medius/minimus and tensor fascia latae and lateral stabilizers of the thoracopelvic region).

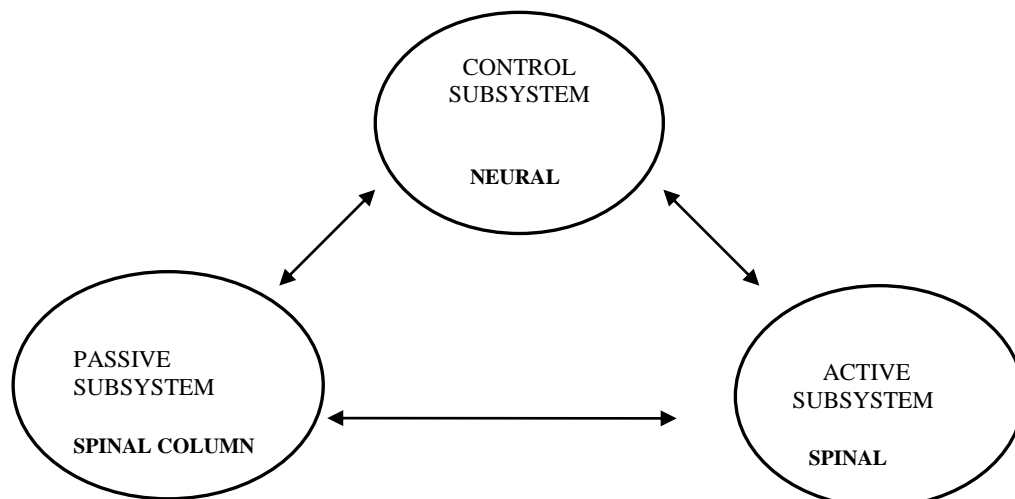
Through these slings, forces that are produced by one muscle can be transmitted to another muscle via fascia, muscle, bone, ligament and capsules. The global muscle system is essentially an integrated sling system, composed of several muscles, which provide forces. These integrated slings help to transfer load and if there is muscle dysfunction it may lead to poor load transfer (Lee and Vleeming, 2003).

- Motor control and Emotion

The co-ordination of motor control is essential in movement, as stability must be maintained but simultaneously motion must be controlled and not restrained. The coordination of the global and local muscle systems that control the lumbopelvic area bring stability but do not restrict movement (Lee and Vleeming, 2003).

Another model was conceptualized by Panjabi (1992); it divided the causes for clinical instability into three groups: namely the passive, active and neural subsystems. The passive subsystem – vertebrae, intervertebral discs, spinal ligaments, joint capsules and the passive properties of the muscles; the active subsystem – active properties of muscles and tendons; the neural subsystem – proprioceptors and other neural control components (Panjabi, 1992).





**Figure 2.2:** An adaptation of the model, conceptualised by Panjabi (1992), showing the relationship between the three sub-systems.

The three subsystems complement each other and work together to achieve stability. The passive subsystem provides stability at the end of ROM while acting as monitoring mechanoreceptors at mid range. The neural subsystem receives data from the passive subsystem and other receptors in the spine, integrates it, and activates the active subsystem to stabilize the spine. Panjabi (1992) thought the most sensitive area for degeneration and injury to be that of the 'neutral zone'. The neutral zone is explained as a small range of movement of the spine over which the passive sub-system has little restraint. This small area of movement was thought to increase with any degeneration, injury and muscle weakness (Panjabi, 1992). It was also maintained by Crisco and Panjabi (1990) that the best way to bring stability to this segment was by means of the local muscles.

Jull and Richardson (1995) explained that the most important muscles to retrain in the lumbar spine were the lumbar multifidus and the transversus abdominis, as these were both local muscles. The lumbar multifidus attaches directly onto the lumbar spine and, according to Wilke *et al.* (1995), accounts for two thirds of the segmental stability in the neutral zone. The transversus abdominis, as mentioned above, assists in increasing the intra abdominal pressure, preparing the trunk for movement

(Cresswell *et al.*, 1992). According to Lee and Vleeming (2003), an increase of the intra abdominal pressure has a positive effect in stabilising the lumbar segments.

### **2.3.2 Dysfunction of the transversus abdominis muscle**

Richardson and Jull (1995) suggested that there was enough evidence to prove the relationship between low back pain and local muscle dysfunction and that the focus in management of these patients should be the rehabilitation of these muscles by exercise. There is a definitive relationship between dysfunction of transversus abdominis and low back pain (Hodges and Richardson, 1997). In a study of patients with chronic low back pain, a timing delay or absence was found in which the transversus abdominis muscle failed to anticipate the initiation of arm or leg motion (Hodge and Richardson, 1999; Hodges, 2003). Delayed activation of transversus abdominis muscle means that the thoracodorsal fascia is not pre-tensed; the joints of the low back and pelvis are not compressed in preparation for external loading and are thus potentially vulnerable to losing intrinsic stability. The possible cause of the transversus abdominis' dysfunction may be due to disuse (Richardson and Jull, 1994) and/or to reflex and pain inhibition associated with lumbar pain and instability (Baugher *et al.*, 1984). This dissertation is dedicated to investigating reflex inhibition and its association to dysfunction of the transversus abdominis muscle.

## **2.4. Relevant neurophysiology**

### **2.4.1 Joint and joint receptors**

The spinal cord consists of a complex system of channels relaying information in electronic form from several parts of the body. The central and peripheral nervous systems work in conjunction to gather, transmit and process information from many different neurophysiological systems in order to co-ordinate movement (Hopkins and Ingersoll, 2000).

The co-ordination requires substantial information from the joint itself. The joint transmits electrical data to the supra-spinal structures concerning the environment,

position and movement of the joint (Levangie and Norkin, 2001). Data is captured via receptors in the ligaments, capsules and tendons and sent via afferent pathways to the supra-spinal structures (Levangie and Norkin, 2001). The receptors are known as mechanoreceptors. Mechanoreceptors are those receptors that respond to physical or mechanical stimuli and are able to transduce that energy into a specific nerve signal (Hopkins and Ingersoll, 2000). These mechanoreceptors can also act as proprioceptors. Hopkins and Ingersoll (2000) concluded that joint receptors have two major functions:

- To provide position sense or information about the relative configuration of body segments
- To initiate protective reflex mechanisms that protect and helps stabilize the joint

According to McLain and Pickar (1998) there are type one, type two and type three mechanoreceptors found in the facet joints of the lumbar spine. According to Freeman and Wyke (1967) and Gatterman (1995), these are characterised as follows:

#### 1. The Ruffini endings

Gatterman (1995) stated that these receptors were sensitive, static and dynamic mechanoreceptors that fire constantly due to continual joint motion. These were described as slow adapting receptors with a low threshold; this means that they respond to the slightest stimuli but, because of the slow adaptation of the receptors, are suited for prolonged discharge. These receptors are often located in the joint capsule, and so respond to changes in capsular pressure, often associated with joint effusions. Ruffini endings also give information on joint limitations and proximity (Hopkins and Ingersoll, 2000).

#### 2. Pacinian corpuscles

Gatterman (1995) stated that these receptors were less sensitive and that they only fire during movement. These dynamic mechanoreceptors are found mainly in the

fibrous periosteum near articular attachments (Jones, 1999:119). The response to stimuli is quick; therefore any movement causes stimulation of these receptors (Hopkins and Ingersoll, 2000). They are active during acceleration and deceleration and are inactive in immobile joints and joints that are moving at a constant velocity (Freiwald, Reuter and Engelhardt, 1999:83).

### 3. Golgi-like bodies

Gatterman (1995) suggested that these were slow reacting mechanoreceptors. These receptors resemble tendon organs and are commonly found in ligaments around the joint (Jones, 1999:119). During the initiation of movement they fire rapidly, then reduce to a slow, steady, discharge. These receptors play an important role in joint position sense (Freiwald, Reuter and Engelhardt, 1999:83-84).

When the receptor is stimulated it depolarizes, creating an action potential; this travels along the dendrite until it reaches the cell body of the nerve which is found in the dorsal horn of the spinal cord (Hopkins and Ingersoll, 2000).

#### **2.4.2 Transmission to the spinal cord**

The capsules of lumbar facet joints receive afferent innervation from the medial branch of the dorsal ramus from the two segmental levels that make up the joint. Thus the L2-L3 facet joint is innervated by the L1 and L2 spinal nerves. The medial branch from the superior segment divides and sends a descending branch along the lamina to innervate the superior portion of the facet capsule. The medial branch from the inferior segment divides, sending branches to the inferior portion of the capsule (Haldeman *et al.*, 2005:237). The afferent fibres bodies are found in the dorsal root ganglia, which appear as small enlargements on the dorsal roots, near the convergence with the ventral roots at the entrance to the intervertebral foramina. Once the impulse passes the intervertebral foramen, it enters the spinal cord (Crossman and Neary, 1995:40).

### 2.4.3 Ascending pathways

The sensory neurons terminate in the grey matter but predominantly in the dorsal horns. The grey matter of the spinal cord is divided into ten zones, known as the Rexed's laminae (Crossman and Neary, 2005:72). Laminae I –III, known as the substantia gelatinosa, receives collaterals from the smallest myelinated (group A delta) and unmyelinated (group C) afferents that are associated to nociception. These decussate at the segment entered and form the spinothalamic and spinoreticular tracts. The information ascends to the pain control centres in the somatosensory cortex via the thalamus (Crossman and Neary, 2005:78).

Fibres from the specialized mechanoreceptors entering the spinal cord divide almost immediately into medial and lateral branches. The medial branches enter the dorsal column and rise up all the way into the brain. The lateral branch enters the lateral horn and then divides many times to provide terminals that synapse with intermediate and anterior portions of the cord gray matter (Guyton and Hall, 2006). These are distributed as follows:

1. A major portion enters the dorsal column and ascends to the brain
2. Many of them are very short and synapse locally to elicit local spinal cord reflexes
3. Others enter the spinocerebellar tracts

The fibres that ascend to the sensory cortex via the thalamus first decussate in the medulla (Guyton and Hall, 2006)

### 2.4.4 Descending pathways

The information from the supraspinal centres is transmitted via descending pathways. The information is conveyed along the following specific spinal tracts (Hopkins and Ingersoll, 2000).

- Corticospinal
- Vestibulospinal
- Rubrospinal

#### **2.4.4.1 Corticospinal tract**

The tract consists of about one million nerve fibres. All these neurons appear to be excitatory. The fibres synapse on the dendrites of alpha and gamma motor neurons, notably of the limbs (Fitzgerald, Gruener and Mtui, 2007:193) as well as the interneurons (Hopkins and Ingersoll, 2000). The tract plays a role in governing the force of muscle contraction that is generated (Porter and Wilkinson, 1997:248).

There are conflicting reports on the role of the corticospinal tract and inhibition; at this stage its role in arthrogenic muscle inhibition is not completely understood (Hopkins and Ingersoll, 2000).

#### **2.4.4.2 Vestibulospinal tract**

The lateral and medial vestibulospinal tracts function to control the anti-gravity (extensor) muscles that function to maintain the upright posture (Guyton and Hall, 2005); they do this through projections to the interneurons and the motor neurons (Hopkins and Ingersoll, 2000). The anti-gravity muscles remain tonically active to maintain upright posture and, prior to movement, postural reflexes change. These postural changes are mediated at the interneuron by the vestibular system and the cerebral cortex (Hopkins and Ingersoll, 2000).

#### **2.4.4.3 Rubrospinal tract**

It originates from the red nucleus and is said to have an inhibitory effect on interneurons (Hopkins and Ingersoll, 2000).

#### **2.4.5 The interneuron**

The vast majority of neurones found within the central nervous system are interneurons (Crossman and Neary, 2005). Ingersoll, Palmieri and Hopkins (2003) suggested that these interneurons were responsible for the development of Arthrogenic Muscle Inhibition.

Once the sensory fibres enter the dorsal horn of the spinal cord, it usually branches to synapse on several interneurons. An interneuron can be described as a neuron receiving information from one neuron and transmitting it to other neurons (Hopkins and Ingersoll, 2000). The network of interneurons and the incredible amount of information from sensory fibres and supraspinal centres travelling through these interneurons make this network difficult to comprehend completely. With this in mind, a general explanation of the role of interneurons in the spinal cord follows.

Interneurons are the intermediates of pathways to  $\alpha$ - and  $\gamma$ -motorneurons and autonomic efferent neurons and to ascending pathways. They receive projections from sensory afferent fibres, descending fibres and other interneurons (Hopkins and Ingersoll, 2000). It would seem that interneurons are merely relay stations, but according to Jankowska and Lundberg (1981:230-233) they have an important integrative function. The net effect of all information arriving at the interneuron is expressed either as inhibitory or excitatory response of the motor neuron pool (Hopkins and Ingersoll, 2000).

The interneurons responsible could be classified into two types.

- The Ia inhibitory interneuron and
- The Ib inhibitory or excitatory interneuron

(Hopkins and Ingersoll, 2000).

Hopkins and Ingersoll (2000) suggested that Renshaw cells would cause the inhibition of the Inhibitory Ia interneurons; this is known as disinhibition. It also receives inputs from the corticospinal, rubrospinal and vestibulospinal tracts which results in excitation. Thus the net effect of the Ia inhibitory interneuron depends on the spatial facilitation between these convergent systems.

Hopkins and Ingersoll (2000) suggest the Ib interneurons receive nerve supplies from:

1. Golgi tendons
2. Joint and cutaneous efferents
3. Inhibitory Ia interneurons

#### 4. Descending tracts from the brain stem

Injury of the joint seems to stimulate the Ib inhibitory; this in turn inhibits the large type A  $\alpha$  motor neurons which are responsible for force contraction of skeletal muscle. It is proposed that interneuron activity was responsible for the development of arthrogenic muscle inhibition (Matkovich, 2004).

## **2.5 Arthrogenic muscle inhibition**

### **2.5.1 Definition**

Arthrogenic muscle inhibition (AMI) is a presynaptic, ongoing reflex inhibition of muscles surrounding a joint after distension or damage to structures of that joint.

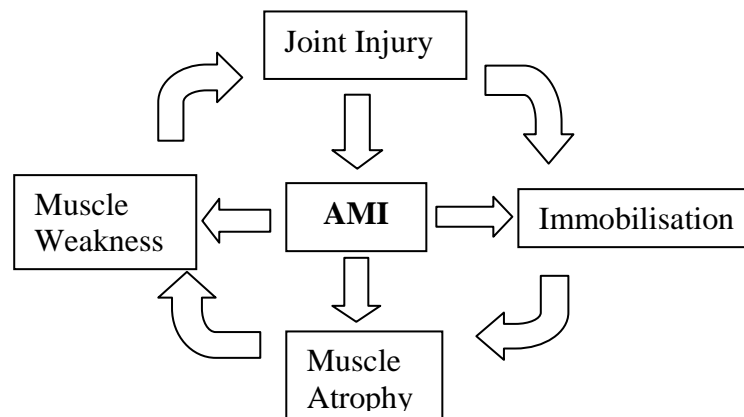
AMI results from the activity of many different joint receptors, which act on inhibitory interneurons synapsing on the motoneuron pool of joint muscular (Hopkins and Ingersoll, 2000). The AMI is said to reduce the ability of a muscle to utilize all motor units of its muscle group to their full extent during a maximum effort voluntary muscle contraction (Suter *et al.*, 2000).

### **2.5.2 Causes of arthrogenic muscle inhibition**

Arthrogenic muscle inhibition is caused by activity from many different joint receptors, which act on inhibitory interneurons synapsing on the motoneuron pool of joint musculature (Hopkins and Ingersoll, 2000). The information from inhibitory interneurons impedes the recruitment within the motoneuron pool, decreasing the force of any contraction originating from that motoneuron pool. Free nerve endings and specialised nociceptors may play a role in inhibition, but the primary effect seems to be as a result of mechanoreceptor activity (Ingersoll, Palmieri and Hopkins, 2003). This does well to explain the action that leads up to the effect of AMI, but the actual causative factors, or aetiology, has a wide range. The potential aetiologies of AMI are traumatic injuries/damage to joint structures (Hurley *et al.*, 1994; Hopkins *et al.*, 2002), joint effusion and pain (Hopkins *et al.*, 2002), immobilization (Reid,



1992:49) and osteoarthritis (Arokoski *et al.*, 2002). The joint injury paradigm from Stokes and Young (1984) supports these causes and shows that many of these factors are related to AMI and also play a part in maintaining it; this leading to sustained muscle weakness.



**Figure 2.3:** The injury paradigm  
(Adapted from Hopkins and Ingersoll, 2000)

### 2.5.3 Clinical implications of AMI

If muscle atrophy is enhanced due to AMI, there is a possibility that it may cause a hindrance in the rehabilitation of the affected muscle groups (Hurley, Jones and Newham, 1994; 305). This is also supported by Suter *et al.*, (2000) who maintain that AMI may limit the functional recovery of the joint and muscle complex after injury and that early goals of treatment should be to reduce the muscle inhibition.

Suter *et al.* (2000) maintained that after the joint is injured the resultant AMI causes muscle weakness, which in turn hampers the rehabilitation process of that joint despite complete muscle integrity. With this in mind, a relationship with low back pain and local muscle dysfunction has been shown (Jull and Richardson, 1995). It is also possible that there is a link between AMI and the local muscle dysfunction.

The removal of AMI allows the patient to maintain or increase activity levels causing a decreased rehabilitation time, a quicker return to activity and a reduction of the

adverse affects of AMI on tissues (source). The prolonged presence of AMI could lead to damage to muscles, bone, ligaments and nerves (Hopkins and Ingersoll, 2000)

#### **2.5.4 Measurement of AMI**

AMI is simply a reduction in the motoneuron pool recruitment, which can clinically be seen as a decrease in the force of contraction of the affected muscle (Suter *et al.*, 2000). The presence of AMI can be measured in at least two ways:

1. The voluntary force output of that motoneuron pool
2. The product of neuromuscular recruitment of the motoneuron pool

Voluntary measurements can be taken using a dynamometer or electromyography. These measurements will give an indication into the force of contraction used by the muscle, which is an indirect evaluation of the muscle's motor neuron recruitment (Perrin, 1993:213).

Involuntary measurements of motoneuron recruitment can be taken by careful stimulation of sensory fibres and the measurement of the reflexive twitch contraction using the Hoffman reflex (Hopkins and Ingersoll, 2000).

#### **2.5.5 Therapeutic interventions that reduce AMI**

AMI can be eliminated or diminished by removing, masking, overriding or otherwise altering inhibitory interneuron activity (Ingersoll, Palmieri and Hopkins, 2003). Investigations into various techniques of reducing AMI have been performed, including:

- Cryotherapy (Hopkins *et al.*, 2002)
- Lidocaine injections (Hopkins *et al.*, 2002)
- Transcutaneous electrical stimulation (TENS) (Ingersoll, Palmieri and Hopkins, 2003)

But, anything that may alter, slow or compete with mechanoreceptor feedback may be a candidate (Ingersoll, Palmieri and Hopkins, 2003). Spinal Manipulative Therapy has been shown to alter the excitability of spinal muscle motor neurons due to the stimulation of mechanoreceptors in the joint capsules (Indahl *et al.*, 1995), suggesting that SMT could be a means to remove this inhibitory action.

### **2.5.6 The proposed neurophysiological mechanism of spinal manipulation on AMI**

Suter *et al.* (1999) investigated the effect of sacroiliac manipulation on quadriceps strength in patients suffering from anterior knee pain, and a follow up study by Suter *et al.* (2000) showed that there was a marked increase in the quadriceps' strength.

It is proposed that a manipulation applied in the form of a high velocity, low amplitude thrust, results in the activation of mechanoreceptors and proprioceptors in and around the manipulated joint which causes an altered afferent input to the motoneuron pool (Suter *et al.*, 2000). This, in turn, causes a change in motoneuron excitability, causing an increase in motor neuron recruitment and thus a decrease in AMI (Suter *et al.*, 2000). Decreased muscle inhibition is demonstrated by the increase in the muscle's strength. Spinal mobilisation was shown to have a stimulatory effect on the obliques externus and internus when it was applied to the L5/S1 region (Ferreira *et al.*, 2007). In the same study there was no increase in the activity of the transversus abdominis (Ferreira *et al.*, 2007).

### **2.5.7 Effect of spinal manipulative therapy on trunk muscles**

It has been shown that spinal mobilisation at L5 /S1 has stimulatory effect on the obliques externus and internus but there was no increase in the activity of the transversus abdominis (Ferreira *et al.*, 2007). Boden (2002), in an unpublished study, stated that, "... Retraining the core stability did have an effect on the endurance of the transversus abdominis muscle but it was not sufficient to conclude that a combined core stabilisation and manipulation program was more effective than manipulation alone." Both studies' deficits could be attributed to AMI, as innervation of the

transversus abdominis muscle arises from thoracic spinal nerves T8-T12, iliohypogastric nerve, and ilioinguinal nerve which are branches off L1 spinal nerve (Simons, Travell and Simons, 1999). As the aforementioned spinal levels were not treated, there would have been no effect on the transversus abdominis muscle, if the hypothesis of AMI stands.

## **2.6 Relevant anatomy**

### **2.6.1 Bony anatomy**

#### **2.6.1.1 Thoracic spine**

The vertebrae in the thoracic segment of the spine have the following characteristic features:

**Table 2.1:** Parts and distinctive characteristics of the thoracic vertebrae  
(Adapted from Moore, 1992)

Part	Distinctive Characteristics
Body	Heart-shaped; has one or two costal facets for articulation with head of rib
Vertebral Foramen	Circular and smaller than those of the cervical and lumbar vertebrae
Transverse Process	Long and strong and extended posterolaterally; length dimensions from T1-T12 (T1- T10 have transverse costal facets for articulation with tubercle of the rib)
Articular Processes	Superior facets directed posteriorly and slightly laterally ; inferior facets directed anteriorly and slightly medially; plane of facets lies on an arc centered about vertebral body
Spinous Processes	Long and slopes posteroinferiorly; tip extends to level of vertebral body beneath

### 2.6.1.2 Lumbar spine

The vertebrae in the lumbar segment of the spine have the following characteristic features:

**Table 2.2:** Parts and distinctive characteristics of the lumbar vertebrae (Adapted from Moore, 1992)

Part	Distinctive Characteristics
Body	Massive; kidney-shaped when viewed superiorly
Vertebral Foramen	Triangular; larger than in thoracic vertebrae and smaller than in cervical vertebrae
Transverse Processes	Long and slender; accessory process on posterior surface of base of each process
Articular Processes	Superior facets directed posteromedially (or medially); inferior facets directed anterolaterally(or laterally); mamillary process on posterior surface of each superior articular process
Spinous Processes	Short and sturdy; thick; broad and hatchet-shaped

## 2.6.2 Relevant Musculature

### 2.6.2.1 Transversus abdominis muscle

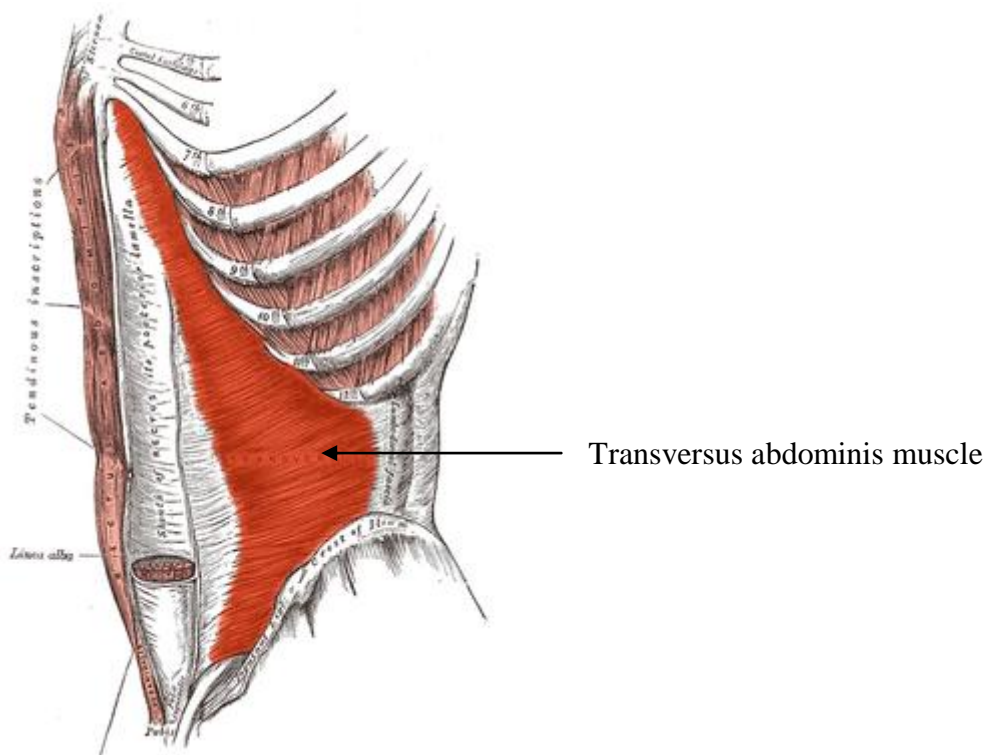
The transversus abdominis muscle is the innermost flat muscle of the anterolateral abdominal wall. Its fibres, except for the most inferior ones, run horizontally.

Its origin is the internal surfaces of the seventh to twelfth costal cartilages, thoracolumbar fascia, iliac crest and the lateral third of the inguinal ligament.

The insertion is at the linea alba with the aponeurosis of the internal oblique muscle, pubic crest and pectin pubis via the conjoint tendon.

The function of this muscle is to compress and support the abdominal viscera. (Moore and Agur, 1995: 82, 83).

Innervation of the transversus abdominis muscle is derived from thoracic spinal nerves T8-T12, 7<sup>th</sup> intercostals nerve as well as iliohypogastric and ilioinguinal nerves which stem from the L1 spinal nerves (Simons, Travell, Simons, 1999).



**Figure 2.4:** An illustration of the transversus abdominis muscle ([http://en.wikipedia.org/wiki/Transversus\\_abdominis\\_muscle](http://en.wikipedia.org/wiki/Transversus_abdominis_muscle))

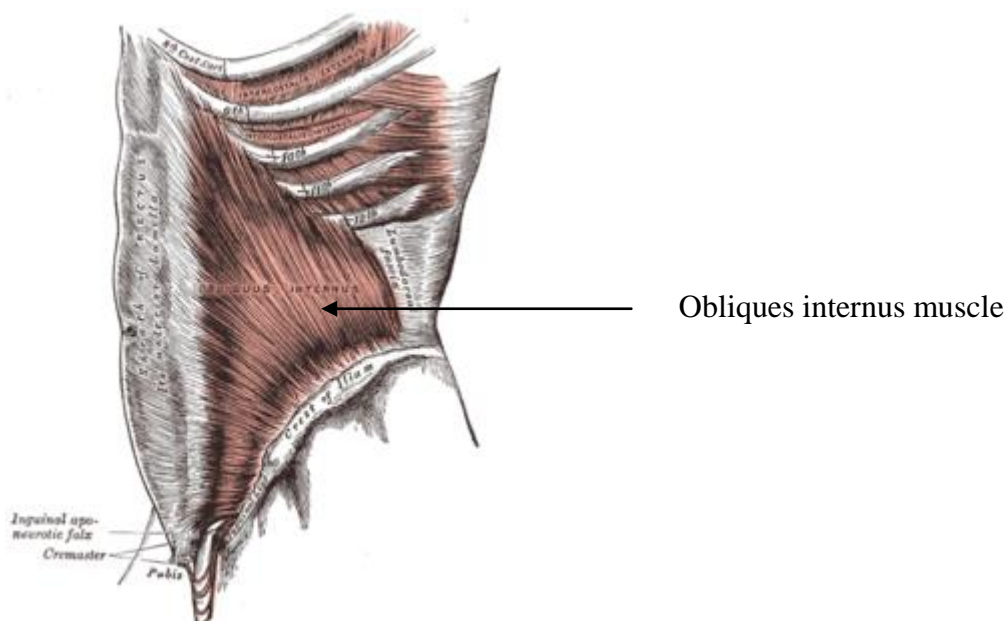
### 2.6.2.2 Internal oblique muscle

The internal oblique, as seen in Figure 2.5, is an intermediate flat muscle, the fibres of which run at right angles to the external oblique. The origin of this muscle is at the thoracolumbar fascia, the anterior two-thirds of the iliac crest and the lateral half of

the inguinal ligament. The insertion of the internal oblique is at the inferior borders of the tenth to twelfth ribs, the linea alba and the pubis via the conjoint tendon.

The action of the internal oblique muscle is to compress and support the abdominal viscera, as well as to flex and rotate the trunk (Moore and Agur, 1995: 82, 83).

Innervation of the obliques internus muscle arises from thoracic spinal nerves T8-T12 as well as iliohypogastric and ilioinguinal nerves which stem from the L1 spinal nerve. This is identical to the innervation of transversus abdominis muscle, excluding the 7th intercostal nerve which is absent in the obliques internus muscle (Simons, Travell, Simons, 1999).



**Figure 2.5:** An illustration of the obliques internus muscle ([http://en.wikipedia.org/wiki/Internal\\_oblique\\_muscle](http://en.wikipedia.org/wiki/Internal_oblique_muscle))

## **2.7 Summary**

Through the literature review it has become apparent that LBP is a very real problem in most societies. Transversus abdominis muscle is part of the local system of muscles as described by Bergmark (1989) and Stevens *et al.* (2006). These local muscles serve to stabilise the lumbar spine. Richardson and Jull (1995) suggested that there was enough evidence to prove the relationship between low back pain and

local muscle dysfunction and that focus in management of these patients should be the rehabilitation of these muscles by exercise.

Literature suggests that optimal core muscle strength, control and endurance working synergistically with the rest of the neuromusculoskeletal system are necessary for lumbar spine stability (Panjabi, 1992; Jull and Richardson, 2000; Arakoski, 2001; McGill, 2003; Akuthota, 2004).

AMI is caused by distension and/or damage of a joint (Hopkins and Ingersoll, 2000). If muscle atrophy is enhanced due to AMI, there is a possibility that it may cause a hindrance in the rehabilitation of the affected muscle groups (Hurley, Jones and Newham, 1994; 305). Suter *et al.* (2000) maintained that after the joint is injured the resultant AMI causes muscle weakness, which in turn hampers the rehabilitation process of that joint despite complete muscle integrity.

Spinal Manipulative Therapy has been shown to alter the excitability of spinal muscle motor neurons due to the stimulation of mechanoreceptors in the joint capsules (Indahl *et al.*, 1995), suggesting that SMT could be a means to remove this inhibitory action.

The literature supports the hypothesis that a decrease in the neurological deficit caused by AMI may result in a faster recovery rate. Therefore this research aimed to test the hypothesis, based upon the literature, by determining the immediate effects of thoraco-lumbar spinal manipulation versus that of lower lumbar spinal manipulation on core muscle endurance and activity in 30 patients with mechanical low back pain.



## **Chapter 3**

### **Materials and methods used**

#### **3.1 The Study Design**

##### **3.1.1 Research design**

A prospective, convenience sample with purpose allocation (pre /post) clinical trial was used to investigate the immediate effects of thoraco-lumbar spinal manipulation compared to lower lumbar spinal manipulation on core muscle endurance and activity in patients with mechanical low back pain. The study received ethical clearance from the Faculty of Health Sciences Research Committee (ethics clearance number 046/08 as found in appendix M), in accordance with the declaration of Helsinki (1964).

##### **3.1.2 Sampling allocation**

A total of 30 participants were allocated to two groups of 15 each. Group one received SMT of the lower lumbar region and group two received SMT of the thoraco-lumbar junction.

##### **3.1.3 Sampling size**

The 30 participants fitting the inclusion criteria were included.

##### **3.1.4 Sampling method**

Low back pain sufferers, acute or chronic, were used. This is due to AMI being based on the premise that it is caused by joint distension or damage to components of the joint (Hopkins and Ingersoll, 2000). It stands to reason that all mechanical low back pain sufferers fall into this category. Advertising was displayed around Durban

University of Technology, sports clubs, gyms and other general notice boards within the greater Ethekewini municipal area (appendix J). If the prospective participant was interested they were able to contact the researcher via telephone. Participation in the study was completely voluntary and no coercion was used.

### **3.1.5 Participant Screening**

Participant screening commenced when the researcher was contacted, telephonically or otherwise. A cursory discussion between the participant and researcher ruled out participants who did not fit the inclusion or exclusion criteria of the study.

Questions included:

- What is your current age?
- Where is your pain?
- On a level of 0 to 100, 100 being the worst pain you've ever experienced and 0 being no pain at all, how would you rate your pain?
- Have you received manual or medicinal intervention in the last 48hrs?

If the participant met the cursory interview outlines, the individuals were then invited to an initial consultation at Durban University of Technology Chiropractic Day Clinic. There the participants were given an information document detailing the methodology of the study (Appendix A) and a letter of informed consent which they were asked to sign (appendix A). This letter informed the participants that they were able to withdraw from the study at any time without reason.

### **3.1.6 Inclusion and Exclusion Criteria**

#### **3.1.6.1 Inclusion Criteria**

- Participants between the ages of 18 (to avoid parent / guardian consent complexities) and 45 years, to avoid and reduce the chance of sacroiliac and / or spinal ankylosis (Kirkaldy-Willis, 1992:418), were included.

- Participants needed to have a minimum of one restriction in lumbar segments L4-S1 and/or a minimum of one restriction between T8-L1.
- Participants accepted needed to have a pain rating scale on the NRS greater than 40 and less than 80. This improved the sample homogeneity (Mouton, 1996).
- Participants who were currently receiving manual or medicinal intervention within 48 hours prior to the onset of the study had to comply with a three-day washout period as proposed by Poul *et al.* (1993).
- Both male and female participants were accepted into the study.

### 3.1.6.2 Exclusion Criteria

- Participants were excluded from the study according to the following contraindications to spinal manipulation treatment (SMT) (Bergmann *et al.*, 1993):
  - Marked osteoporosis that was previously diagnosed.
  - Ankylosing Spondylitis.
  - The presence of fever, tumours, tuberculosis or any infectious diseases.
  - Spinal fusion or spinal surgery.
  - Acute disc herniation.
  - Abdominal aortic aneurysm.
- Participants who had extreme discomfort on contraction of the abdominal muscles.
- Participants who required further clinical testing to confirm the diagnosis were excluded.
- Participants who presented with neurological signs and symptoms such as
  - Presence of parasthesias
  - Presence of neurological deficit.
  - Presence of root tension signs.
  - Presence of hip, buttock, or back pain on performing the straight leg raise test.(Plaughner, 1993)

### **3.2 Intervention method**

All participants then underwent a case history (appendix B), physical examination (appendix C) and lumbar regional examination if in group one or both a thoracic and a lumbar regional, if in group two (appendices D and E respectively). The treatment was then noted in the SOAPE note (appendix F).

- Group **ONE** were asked to rate their pain via the numerical rating scale (NRS) the results of which were recorded (appendix G).
- The researcher then motion palpated the participant, focusing on finding fixations between L4 and S1 segments (as per the method in Schaefer and Faye, 1989). The patient was then re-motion palpated by the on duty clinician, so as to confirm levels of fixation/s. If there was no consensus a discussion and re-evaluation were performed until a level was agreed upon by both parties.
- The fixated facets were marked on the patient with a crayon pencil.
- The participant then underwent training on the recruitment of the transversus abdominis muscle, by the four point kneeling method (Oldrieve and Evans, 2000).
- Electromyography (EMG) electrodes were placed over the left and right internal oblique muscles, two on each side. The participant was asked to lie supine and expose the anterior superior iliac crest bilaterally, where the electrodes were placed one centimeter lateral to the anterior superior iliac crest (Ng *et al.*, 1998).
- The participant then underwent the prone abdominal draw in test with a pressure biofeedback unit (appendix I).

- The participant was then asked to be seated and connected to the leads that supply the Power Lab EMG unit.
- The researcher then approached the participant from behind and braced the upper body.
- The participants were then asked to perform a right rotation of their trunk at the maximum of their ability for 2 seconds and repeated that contraction 3 consecutive times, with no rest period between contractions. The EMG readings were taken from the left electrode.
- The participant were then asked to repeat the rotation, but to the left side and the readings were taken from the right electrode.
- The participant then underwent SMT at the fixated level/s between L4-S1, using a prone drop piece method. The participants were motion palpated again; if there was an increase in passive range of motion the SMT was be deemed successful.
- A pain numerical rating scale (NRS) score was taken again (appendix G).
- The participant was then asked to perform a right rotation of the trunk at the maximum of their ability for 2 seconds and repeat the contraction 3 consecutive times, with no rest period between contractions. The EMG readings were taken from the left electrode.
- The participant was then asked to repeat the rotation, but to the left side and the readings were taken from the right electrode.
- The participant then underwent the prone abdominal draw in test with a pressure biofeedback unit (appendix I).
- The EMG pads were finally removed

All above information was captured on appendix H.

Group **TWO** underwent the same procedures as group one, with differences as follow:

- The researcher motion palpated the participant, focusing on finding fixations between T8-L1 segments. The patient was then motion palpated by the on duty clinician, so as to confirm levels of fixation/s. If the findings were refuted a discussion and re-evaluation were held until a level was agreed upon by both parties.
- The participant then underwent SMT at the fixated level/s between T8-L1, using a prone drop piece method. The participants were motion palpated again; if there was an increase in passive range of motion the SMT was be deemed successful.

Again, all above information was captured on appendix H

### **3.3 Data collection and materials used**

#### **3.3.1 Objective data**

##### **3.3.1.1 Pressure biofeedback unit (appendix I)**

Endurance testing of the transverse abdominal muscle was done by using the stabilizer biofeedback device (Stabilizer Manual Chattanooga Group Inc., 4717 Adams Road, Hixson TN 37343, USA). This was done using the prone abdominal draw in test for transverse abdominis.

This was used as a method of measuring the endurance of the transversus abdominis muscle, and was timed using a stopwatch.

Richardson *et al.* (1999) developed the prone abdominal draw in test that is effectively used to measure the endurance of the transversus abdominis muscle. These findings were supported by research by Jull and Richardson (1995) and Evans and Oldrieve (2000) and thus it was deemed that it was a reliable test to use in this study.

The prone abdominal draw in test, as described below, tests both the transversus abdominis muscle and the obliques internus muscle, which is also suited to the study.

Before the formal testing commenced, the participants were taught four-point kneeling, in accordance with Richardson *et al.* (1999). This position allowed for a facilitated deep stretch of the abdominals resulting from a forward drift of the abdominal contents. This stretch led to an inhibitory effect on the superficial muscles, particularly rectos abdominis (Richardson & Jull 1995).

#### The prone abdominal draw in test for transversus abdominis and internal oblique muscles:

A 3-chamber pressure cell was placed centrally under the abdomen, with the umbilicus in the centre of the inflatable sleeve, and inflated to a baseline of 70mmHg. The subject was then instructed to draw the abdominal wall upward and inward without moving the spine or pelvis. The pressure reading should have decreased by 6-10 mmHg.

A variation of 2 mmHg was allowed for normal breathing pattern. A measurement was taken of the time at which the patient could no longer hold the contraction at the baseline level (70mmHg – 6 to 10 mmHg). Once the training was complete the participant was told to contract as taught; the time was then recorded. If unable to contract and reproduce the pressures that was required, they were asked to do it to the best of their ability.

### **3.3.1.2 Surface electromyograph (EMG)**

The Power Lab unit (ADInstruments LTD. 7 Bond Street, Consultancy house, Dunedin, New Zealand) was used to take the EMG readings. It is an advanced multi channel EMG device, which measures the muscle activity in millivolts per second (mV.s). The data was captured on Chart 5 version 5.5 09 (November 2006 Copyright ADInstruments 1994-2006). The surface EMG is known for its sensitivity, being responsive not only to muscle activity but also to sound. So care was taken to make sure the environment was as conducive to accurate readings as possible. Research addressing the functional use of surface EMG, states that it is used successfully in assessing patients with low back pain (Pullman *et al*, 2000). Fine-wire EMG is very accurate but due to its invasive nature it was not used. EMG is the most common tool used in research done on AMI; its use ensures validity to the study (Hopkins and Ingersoll, 2000).

The participants were seated on the chiropractic table with their feet securely placed on the floor. The participant was then asked to cross their arms and hold their opposite shoulder. The researcher then proceeded to brace the torso by embracing it from a posterior aspect, so as to offer resistance to the maximal voluntary contraction (MVC). The MVC is described as being one of the best ways to test AMI (Hopkins and Ingersoll, 2000) and as a result was employed in this research program. To increase reliability, the MVC was repeated three times with no rest in between contractions; the mean was taken between the three and was entered as the MVC for that specific round of testing (pre or post manipulation).

### **3.3.2 Subjective data**

#### **3.3.2.1 Pain numerical rating scale (NRS) (appendix G)**

A pain rating score was taken before and after the SMT procedure, to ascertain if the procedure had an effect on the low back pain. The participant was asked to indicate on a line what their pain rating was, on a scale of 0-100; zero (0) being no pain and one hundred (100) being the most excruciating pain they had ever experienced. This



method is known to be the most practical of the pain rating scales in patients with low back pain (Mannion *et al.*, 2007).

### **3.4 Statistical analysis**

The data was captured using Microsoft Excel and then exported to SPSS version 15, where it was analyzed statistically.

Baseline outcome values were compared between groups to assess whether equivalence was achieved, by means of independent samples t-test for quantitative normally distributed variables.

Repeated measures ANOVA testing was used to compare the change in all quantitative outcomes over time between the two groups. Profile plots were generated to visually compare trends in the two groups. Crossing over profiles or non parallel lines indicated the presence of a trend of an effect of treatment. The direction of this trend was reported and discussed. A statistically significant ( $p < 0.05$ ) time\*group interaction effect indicated a significant intervention effect.

Intra-group correlation analyses between changes in outcomes over time were achieved using Pearson's correlation analysis. A  $p$  value  $< 0.05$  and a correlation coefficient  $> 0.5$  were considered as statistically and clinically important.

## **Chapter 4**

### **Statistical Methodology and Results**

#### **4.1 Statistical methodology**

SPSS version 15.0 (SPSS Inc., Chicago, Illinois, USA) was used to analyze the data. Demographic and baseline outcome values were compared between groups to assess whether equivalence was achieved through randomization by means of Independent samples t-test for quantitative normally distributed variables and Pearson's chi square tests for categorical variables.

Repeated measures ANOVA testing was used to compare the change in all quantitative outcomes over time between the two groups. Profile plots were generated to visually compare trends in the two groups. Crossing over profiles or non parallel lines indicated the presence of a trend of an effect of treatment. The direction of this trend was reported and discussed. A statistically significant ( $p < 0.05$ ) time\*group interaction effect indicated a significant intervention effect.

Intra-group correlation analyses between changes in outcomes over time was achieved using Pearson's correlation analysis. A  $p$  value  $< 0.05$  and a correlation coefficient  $> 0.5$  was considered as statistically and clinically important.

#### **4.2 Results**

##### **4.2.1 Demographic and baseline comparison between treatment groups**

Thirty participants were randomized into two equal groups. The mean age of the sample overall was 25.4 years (standard deviation 4.4 years) and the ages ranged from 20 to 41 years. Male participants made up 60% of the sample whilst females

made up the remaining 40%. The vast majority were Caucasian (86.7%) and their occupations are shown in Table 4.1.

**Table 4.1:** Occupations of the sample ( $n = 30$ )

	Frequency	Percent
Sales Rep	4	13.3
Chiropractor	4	13.3
Psychology student	3	10.0
Chiropractic Student	3	10.0
Unemployed	2	6.7
Marketing	2	6.7
Shop manager	1	3.3
Supplement Consultant	1	3.3
Biokineticist	1	3.3
General manager	1	3.3
Waitress	1	3.3
Property evaluator	1	3.3
Bachelor of commerce student	1	3.3
Homoeopath	1	3.3
Plumber	1	3.3
Production manager	1	3.3
Photography student	1	3.3
Chef	1	3.3
Total	30	100.0

On comparison of the demographics and baseline outcomes between the two groups, it can be seen from Table 4.2 that there was a statistically significant difference in terms of right EMG between the groups ( $p = 0.035$ ). The thoraco-lumbar group started with a higher mean value for this variable than the lumbar group. Otherwise there were no significant differences ( $p > 0.05$ ) in other variables between the groups.

**Table 4.2:** Comparison of demographic and baseline variables between the two groups

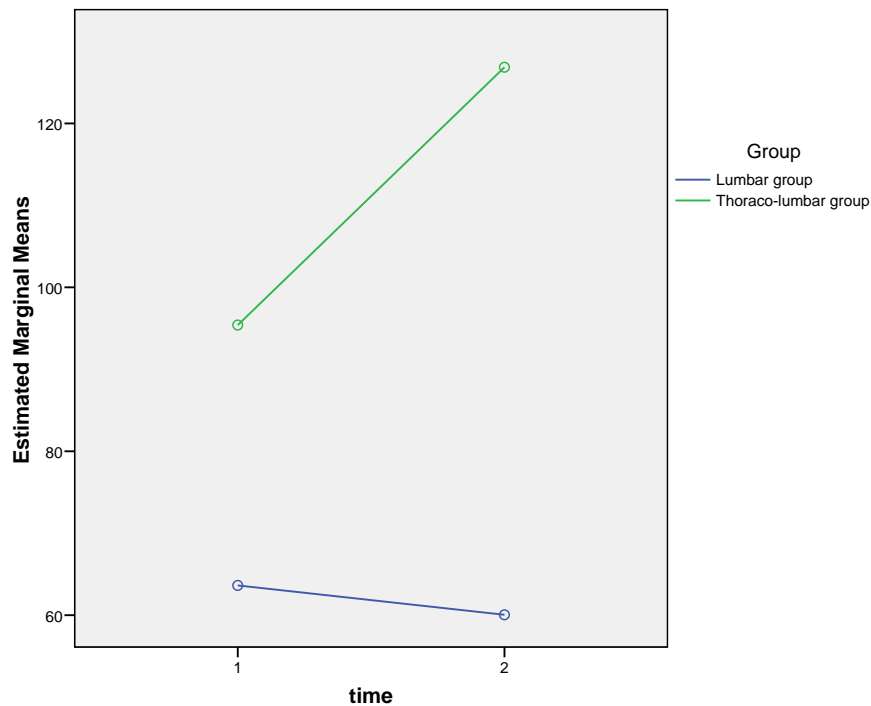
		Group				p value
		Lumbar group		Thoraco-lumbar group		
		Count	%	Count	%	
Race	Caucasian	13	86.7%	13	86.7%	0.135
	Black	0	.0%	2	13.3%	
	Indian	2	13.3%	0	.0%	
Side	Left	7	46.7%	2	13.3%	0.136
	Right	4	26.7%	6	40.0%	
	Bilaterally	4	26.7%	7	46.7%	
Age: mean (SD)		24.0	2.3	26.9	5.5	0.078
Time pre: mean (SD)		63.6	45.3	95.4	85.8	0.215
mmHg pre: mean (SD)		7.3	2.8	6.1	2.7	0.239
Left EMG pre: mean (SD)		32.2	15.8	43.2	20.5	0.109
Right EMG pre: mean (SD)		31.9	11.6	46.3	22.3	0.035
NRS pre: mean (SD)		56.3	9.8	50.1	9.0	0.083

**Aim 1. To evaluate the relative effectiveness of thoraco-lumbar spinal manipulative therapy compared to that of lower lumbar spinal manipulative therapy, in terms of objective findings (test endurance of transversus abdominis muscle via biofeedback unit and EMG readings of obliques internus muscle).**

#### 4.2.2 Test endurance (Time)

Table 4.3 shows that there was a statistically significant time\*group interaction for this outcome ( $p = 0.007$ ). This means that the effect of time was different in each treatment group, and this is borne out by Figure 4.1 which shows that the lumbar group showed a mean decrease in endurance over time while the thoraco-lumbar group showed a steep increase in endurance in the same time period. Therefore

the thoraco-lumbar intervention produced a more favourable measurement than the lumbar intervention for this factor.



**Figure 4.1:** Profile plot of mean test endurance (time) by time and group

**Table 4.3:** Within and between subjects effects for test endurance (time)

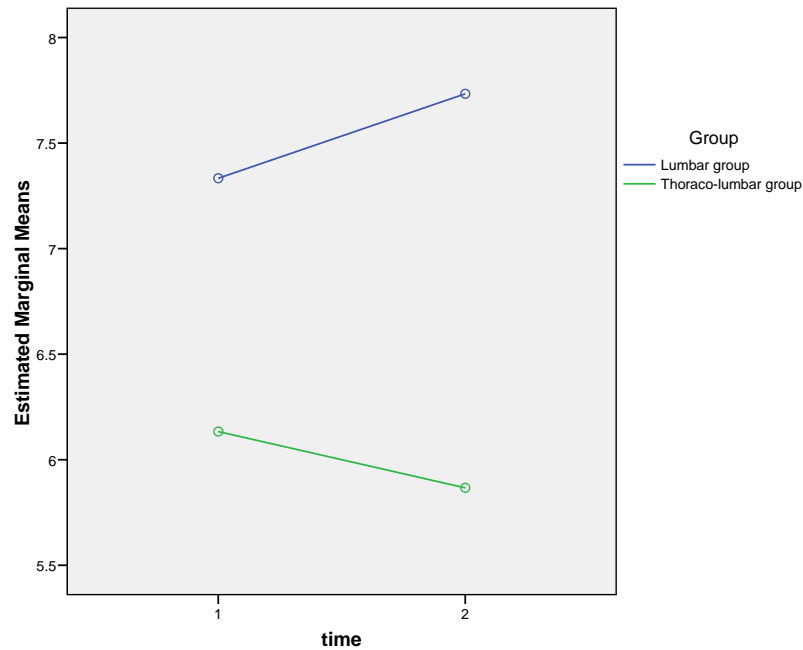
Effect	Statistic	<i>p</i> value
Time	Wilk's lambda = 0.839	0.028
Time*group	Wilk's lambda = 0.768	0.007
Group	F = 2.675	0.113

#### 4.2.3 Test endurance (mmHG)

Table 4.4 shows that there was no significant intervention effect for this outcome ( $p = 0.239$ ). Therefore both treatments were equally effective for this outcome. Figure 4.2 shows that there was a slight trend in that the thoraco-lumbar group showed a slight decrease in mean value over time while the lumbar group increased slightly.

**Table 4.4:** Within and between subjects effects for test endurance (mmHg)

Effect	Statistic	<i>p</i> value
Time	Wilk's lambda = 0.998	0.812
Time*group	Wilk's lambda = 0.951	0.239
Group	F = 3.130	0.088

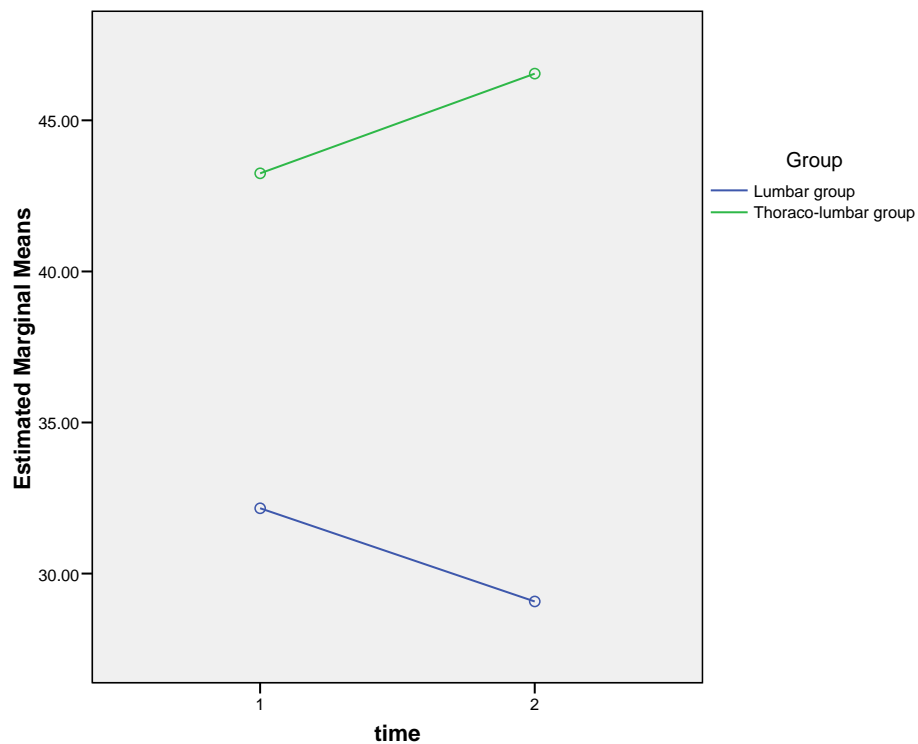
**Figure 4.2:** Profile plot of mean test endurance (mmHg) by time and group

#### 4.2.4 Left EMG

There was a statistically significant interaction effect for this outcome ( $p = 0.030$ ) meaning that there was a significant effect of the intervention and the two groups did not react the same over time. Figure 4.3 shows that the effect in the lumbar group was to decrease over time while the thoraco-lumbar group increased over time.

**Table 4.5:** Within and between subjects effects for Left EMG

Effect	Statistic	<i>p</i> value
Time	Wilk's lambda = 1.000	0.939
Time*group	Wilk's lambda = 0.843	0.030
Group	F = 4.54	0.042

**Figure 4.3:** Profile plot of mean left EMG by time and group

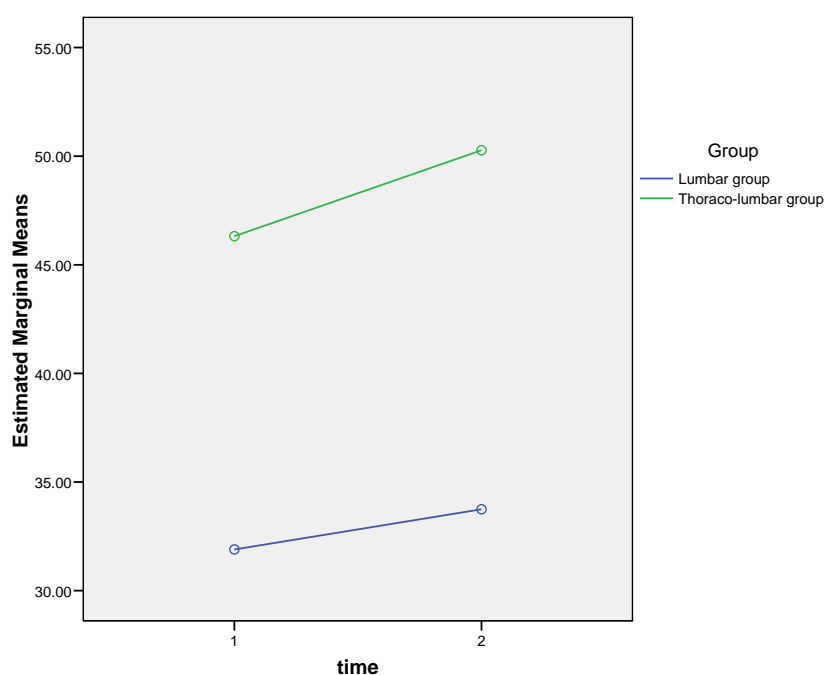
#### 4.2.5 Right EMG

There was no intervention effect in terms of this outcome ( $p = 0.473$ ). There was a statistically significant group effect ( $P=0.024$ ) and an almost significant time effect ( $0.055$ ), meaning that the two groups were significantly different with regard to this outcome at both time points and that there was a general increase over time, but this increase over time was the same in both groups. Figure 4.4 confirms this by showing

that the profiles of the two groups were parallel over time. Thus, it did not matter which treatment was received for this outcome, both treatments had the same effect.

**Table 4.6:** Within and between subjects effects for Right EMG

Effect	Statistic	p value
Time	Wilk's lambda = 0.875	0.055
Time*group	Wilk's lambda = 0.981	0.473
Group	F = 5.66	0.024



**Figure 4.4:** Profile plot of mean right EMG by time and group

**Aim 2.** *To evaluate the relative effectiveness of thoraco-lumbar spinal manipulative therapy compared to that of lower lumbar spinal manipulative therapy, in terms of subjective findings (NRS).*

#### 4.2.6 NRS

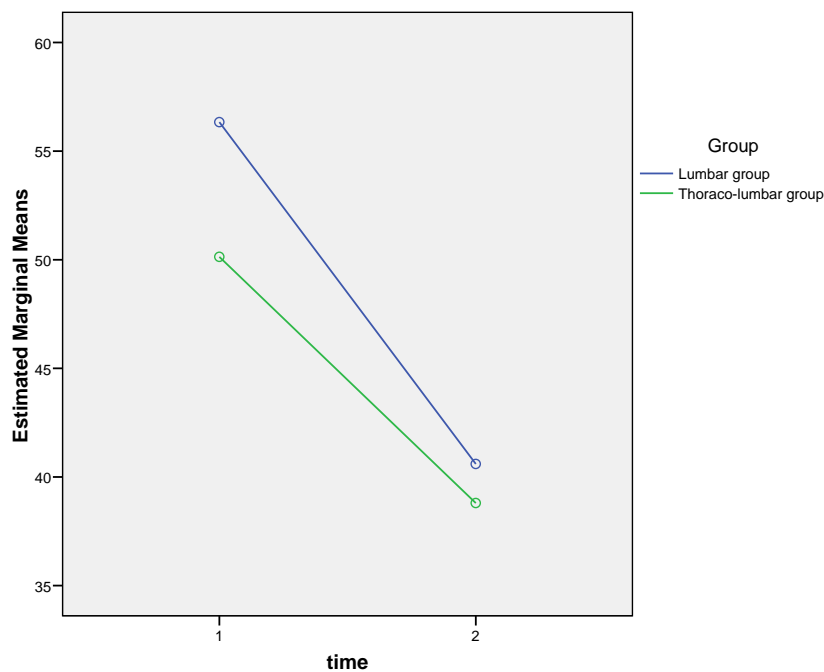
Table 4.7 shows that while the overall effect of time was highly significant in both groups ( $p < 0.001$ ) there was no differential effect of the intervention ( $p = 0.373$ ).



Figure 4.5 shows that both groups pain decreased markedly over time and almost to the same extent, i.e. the profiles were nearly parallel. Therefore, in terms of pain, both treatments worked equally well.

**Table 4.7:** Within and between subjects effects for pain (NRS)

Effect	Statistic	<i>p</i> value
Time	Wilk's lambda = 0.474	< 0.001
Time*group	Wilk's lambda = 0.972	0.373
Group	F = 1.20	0.283



**Figure 4.5:** Profile plot of mean NRS by time and group

**Aim 3. To compare the correlation of trends in terms of objective and subjective findings in participants with mechanical low back pain.**

Correlations were assessed intra-group.

### 4.2.7 Lumbar group

The only significant correlation found in this treatment group was between change in right EMG and change in NRS. This was a negative correlation ( $r = -0.531$ , Table 4.8) which means that as the one increased so the other decreased. Therefore as right EMG values increased, so pain decreased.

**Table 4.8:** Pearson's correlations between changes in outcomes in the Lumbar group (n=15)

		Change in time	Change in pressure	Change in left EMG	Change in right EMG	Change in NRS
Change in time	Pearson Correlation	1	-.020	.473	.194	-.122
	Sig. (2-tailed)		.945	.075	.488	.665
	N	15	15	15	15	15
Change in pressure	Pearson Correlation	-.020	1	.213	-.173	.214
	Sig. (2-tailed)	.945		.446	.537	.444
	N	15	15	15	15	15
Change in left EMG	Pearson Correlation	.473	.213	1	.245	-.359
	Sig. (2-tailed)	.075	.446		.378	.188
	N	15	15	15	15	15
Change in right EMG	Pearson Correlation	.194	-.173	.245	1	-.531(*)
	Sig. (2-tailed)	.488	.537	.378		.042
	N	15	15	15	15	15
Change in NRS	Pearson Correlation	-.122	.214	-.359	-.531(*)	1
	Sig. (2-tailed)	.665	.444	.188	.042	
	N	15	15	15	15	15

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

### 4.2.8 Thoraco-lumbar group

In the thoraco-lumbar group there were two significant correlations. The first was between change in NRS and change in endurance (time). This was a negative correlation ( $r = -0.617$ ) therefore, as time increased, so pain decreased. The next correlation was between change in left EMG and change in endurance (mmHg). This was also a negative correlation ( $r = -0.534$ ) which indicated that as pressure decreased, left EMG values increased.

**Table 4.9:** Pearson's correlations between changes in outcomes in the Thoraco-lumbar group (n=15)

		Change in time	Change in pressure	Change in left EMG	Change in right EMG	Change in NRS
Change in time	Pearson Correlation	1	-.304	-.003	.018	-.617(*)
	Sig. (2-tailed)		.271	.992	.950	.014
	N	15	15	15	15	15
Change in pressure	Pearson Correlation	-.304	1	-.534(*)	-.027	.296
	Sig. (2-tailed)	.271		.040	.923	.283
	N	15	15	15	15	15
Change in left EMG	Pearson Correlation	-.003	-.534(*)	1	-.022	-.074
	Sig. (2-tailed)	.992	.040		.938	.793
	N	15	15	15	15	15
Change in right EMG	Pearson Correlation	.018	-.027	-.022	1	.145
	Sig. (2-tailed)	.950	.923	.938		.606
	N	15	15	15	15	15
Change in NRS	Pearson Correlation	-.617(*)	.296	-.074	.145	1
	Sig. (2-tailed)	.014	.283	.793	.606	
	N	15	15	15	15	15

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

### 4.3 Conclusion

There was therefore, a significant difference between the two treatment groups in terms of endurance measured in seconds, and left EMG measurements. The thoraco-lumbar group was found to show superior outcomes to the lumbar group in terms of these outcomes. For the remainder of the outcomes the effects in the two groups were similar. Patients receiving thoraco-lumbar manipulation can expect to see their endurance increase as their pain decreases.

## **Chapter 5**

### **Discussion**

#### **5.1 Introduction**

This chapter of the dissertation will be based on the discussion of the research data captured. All the data collection was done pre- and post-intervention for all individuals.

#### **5.2 Discussion**

##### **5.2.1 Demographics**

The demographics revealed a 3:2 ratio of male and females respectively, with males making up 60% and females 40 % of the total intake. The lifetime prevalence was shown to be more significant in females (56.4%) as opposed to for men (48.4%) (van der Meulen, 1997); this was not supported by the findings in the current research. This may be correlated to the small sample size used, which may not represent the true demographics for patients with lower back pain in South Africa. The mean age fell within the Kirkaldy-Willis' (1988) recommended range of 18-45 years. The ages ranged from 20 - 41 years with a mean of 25.4. 86.7% of the participants were Caucasian, 6.65% Indian and 6.65% Black; this does not fall into the South African population split, but as Jayson (1992) reported, racial differences in the frequency of low back pain had not been adequately studied, supported by van der Meulen (1997) and Docrat (1999). There was a large diversity of occupations; the individuals who were physically active may have given better EMG reading as a trend was seen that a decrease in body fat content gave better EMG readings. As the percentage body fat was not analyzed it is difficult to obtain any specific conclusions in this parameter. The male - female ratios were identical in both groups, 3:2 males and females respectively. This allowed for increased homogeneity. The only other similar study by

Ferreira *et al.* (2007) had 20 participants and a mean age of 33 and 28 in each of the groups; in comparison, this study comprised 30 patients and had mean ages of 24 and 26.9. The demographics, as shown in table 4.2, were similar to Ferreira *et al.* (2007), which shows good comparability between the two studies.

## 5.2.2 Inter-group analysis

### 5.2.2.1 Test muscle endurance

The time\*group interaction (Table 4.3) showed a statistically significant interaction ( $p = 0.007$ ), whilst the Time ( $p = 0.028$ ) and Group ( $p = 0.113$ ) did not. This equates to a difference in the two groups' endurance times which are markedly different. Figure 4.1 shows these differences, with the lumbar group showing a mean decrease in endurance time whilst the thoraco-lumbar group shows a steep increase in the same time. Thus the thoraco-lumbar intervention showed to be the more favourable intervention. The increase in the thoraco-lumbar group may be due to AMI being removed by the manipulation of the levels that innervate that muscle (Hopkins and Ingersoll, 2000). The decrease of the endurance time in the lumbar group may be due to the incorrect level of manipulation; thus the AMI would still be present and the inhibiting function would still be taking affect over the muscles required for the endurance test. This supports the links discussed in chapter 2, and supports the hypothesis of AMI.

The  $p$  values for Time ( $p = 0.812$ ), Time\*Group ( $p = 0.239$ ) and Group ( $p = 0.088$ ) were not statistically significant and there was no significant intervention effect for this outcome. There was a slight trend, Figure 4.2, which showed a slight increase in the lumbar group and a slight decrease in the thoraco-lumbar group. This shows that the lumbar group showed a slight increase in the ability to drop the pressure within the biofeedback apparatus, whilst in the thoraco-lumbar group it decreased. Jull *et al* (1993) suggest that there is evidence emerging to show that the oblique abdominals and transversus abdominis muscles may not always be optimally recruited or may fatigue in their normal stabilising role even in normal, currently asymptomatic individuals. In the testing phase, the focus was not on the maximal amount of

decrease in pressure but that the abdominal draw in reduced the pressure by 6-10 mmHg; this Richardson *et al* (1999) suggested being a successful abdominal draw in test. The statistics, therefore, do not allow for any conclusions to be drawn.

#### 5.2.2.2 EMG data

The left and right EMG readings showed different trends, with the left showing the only statistical effect ( $p = 0.030$ ). Figure 4.3 showed that there was an increase of the EMG readings in the thoraco-lumbar group but a decrease in the lumbar group. The reason for a decrease in the lumbar group could be associated with a further increase to the inhibitory effect of AMI at the thoraco-lumbar segment, that supplies the muscle being tested. The increase may be due to a biomechanical effect occurring when the lower lumbar joints were moved, aggravating the thoraco-lumbar joint, causing an increased AMI. The increase found could be explained by the theory that if the AMI was removed, which would decrease the inhibitory interneurons from firing on their synapses with the motor-neuron pool, it could allow an increase in the muscle activity (Hopkins and Ingersoll, 2000)

The right EMG reading showed no intervention effect ( $p = 0.473$ ). It did, however, show a statistically significant group effect and an almost statistically significant time effect. Both treatments were equally effective, showing an equal increase over time, but the time points were significantly different. This is illustrated in figure 4.4 with parallel lines.

An explanation for the effect only taking place on the left hand side could be attributed to two things. The left side was adjusted by the right hand of the researcher; as the researcher is right side dominant, there may have been a larger velocity created when moving joints on the left side, thus stimulating the interneuron more and intern removing AMI more successfully. With regards to the participants, a larger baseline EMG measurement was taken on the contralateral side to dominant side i.e. right handed participants showed larger baseline EMG measurement from left EMG electrodes. The participants' side of dominance was not noted and as a result this factor was not statistically analysed. This may have influenced the above

results. If AMI were present it is possible that a greater effect may have been shown on the non-dominant side as the dominant side would have been more active, in terms of the EMG, and may thus not have had the same magnitude of response. This may play a role in the increased in EMG recording on the left.

### **5.2.2.3 NRS**

There was a significant overall effect of time ( $p < 0.001$ ). It showed a decrease of pain over time in both groups, it is statistically significant but not a clinically important result. Both the treatments were effective in removing pain from the participants which could be attributed to tactile stimulation during motion palpation and spinal manipulative therapy, which inhibits the pain fibres, as explained by Melzack and Wall in their gate control theory (Cramer and Darby, 1995:34).

## **5.2.3 Intra-group analysis**

### **5.2.3.1 Lumbar group**

The singular significant finding is that as the right EMG increased the NRS decreased, which is a negative correlation ( $r = -0.531$ ) shown in table 4.8. Also noted was the fact there was a correlation, between left EMG increase and NRS decrease, but it was determined not to be statistically viable. The increase of muscle activity may be as a result of the effect of spinal manipulative therapy of the lumbar spine that causes increased functional ability of the patient, reduces pain and relaxes hypertonic muscles (Herzog *et al.*, 1999).

### **5.2.3.2 Thoraco-lumbar group**

Within this group two significant correlations were found. The first was between the change in NRS and change in endurance (time), which was a negative correlation ( $r = -0.617$ ). This showed that a decrease in pain allowed for an increase in endurance time; this means that pain could be related to the inability of an individual to contract

the core muscles. With the intervention at the thoraco-lumbar junction, it may have removed the AMI (Hopkins and Ingersoll, 2000) as well as providing pain suppression (Melzack and Wall in Cramer and Darby, 1995:34), which when combined may have provided the significant intervention statistics in thoraco-lumbar group compared to that of the lumbar group.

The second correlation was that of the change in left EMG to that of a change in endurance (mmHg). The negative correlation ( $r = 0.534$ ) meant that as left EMG increased the pressure produced decreased. In the testing phase, the focus was not on the maximal amount of decrease in pressure but that the abdominal draw in reduced the pressure by 6-10 mmHg; this Richardson *et al.* (1999) suggested to be a successful abdominal draw in test. The statistics would, therefore, not allow for any conclusions to be drawn from it.



## **Chapter 6**

### **Conclusions and Recommendations**

#### **6.1 Conclusions**

The premise of the research was to test the effect of thoraco-lumbar manipulation versus that of lumbar manipulation on the transversus abdominis and internal oblique muscles.

There was a significant difference between the two treatment groups in terms of endurance measured in seconds, and left EMG measurements, thus rejecting the first Null hypothesis must be rejected. The thoraco-lumbar group was found to show superior outcomes to the lumbar group in terms of these outcomes. The other outcomes that showed no difference in trends, may be as a result of intervention methods.

This shows that manipulating restrictions at the level from which muscles receive their innervation, actually stimulates the muscles involved. In this case the thoraco-lumbar group showed that when manipulation the level of spinal innervation occurred it stimulated the transversus abdominus and internal obliques, in endurance and EMG readings on the left. The transversus abdominus and obliques internus muscles share the same innervation, so by extension, an increase in activity (Millivolts) in the obliques internus would also mean an increased activity in the transversus abdominus. This is also supported by the finding that endurance time was increased during prone biofeedback testing, which may be attributed to a decreased inhibition of the motorneuron pool that allowed for complete recruitment of the muscle. This also supports the hypothesis of AMI, as it can be shown that the removal AMI by manipulation lead to increased muscle activity and endurance.

For the other outcomes the effects in the two groups were similar. Patients receiving thoraco-lumbar manipulation can expect to see their endurance increase as their pain decreases.

As there was a decrease of pain in both groups it is difficult to determine if AMI is in fact linked to pain inhibition or not. The second Null hypothesis was accepted.

A correlation between increased EMG and increased muscle endurance (mmhg and time) should have been seen. This would have proven that there was an increase in both the transversus abdominis and obliques internus muscles. Even though there are correlations proving that there are connections, these are not significant enough to draw any confident conclusions.

## **6.2 Study limitations**

1. The biofeedback unit demonstrated variations from person to person, the main problem being the ability to isolate the transversus abdominis and obliques internus muscles and exclude the other global muscles. This lead to inconsistencies.
2. The surface EMG unit also gave varying results; the amount of resistance played a large role. The cause of this is unknown, but a negative correlation of increased body fat to decreased EMG readings was observed by the researcher.

## **6.3 Recommendations**

1. The surface EMG should be excluded from the testing and the more specific fine-wire EMG should be used; this would allow for the transversus abdominis muscle to be tested directly as well as the obliques internus muscle to be tested more accurately.
2. The participants should be allowed to enter the study only if they meet a certain body fat percentage, thus increasing the homogeneity.

3. The testing of the muscles involved should be expanded to incorporate the multifidus muscle, as it is also a local stabilising muscle which has been linked to low back pain .
4. The testing procedure should be limited to one side; this may lead to fewer variables and more constancy through intervention and testing, thus increasing homogeneity.
5. To increase homogeneity the participants should all be related to one sport/lifestyle type; this will allow for a more consistent outcome of the results.
6. Future research should be done to evaluate the effect of the treatment over a longer period, to ascertain the long term effects.
7. The allocation method for placing participants into testing groups should be altered to improve the power of the study.

## **References**

Akuthola, V., Nadler, S.F. 2004. Archives of Physical Medical Rehabilitation. 85(3): S86-92. Department of Rehabilitation Medicine, University of Colorado, Denver, USA.

Arokoski, M. H., Arokoski, J. P. A., Haara, M., Kankaanpaa, M., Vesterinen, M., Niemitukia, L. H., Helminen, H. J. 2002. Hip muscle strength and muscle cross sectional area in men with and without hip osteoarthritis. The Journal of Rheumatology, 29(9): 2185 –2195.

Baughner, W.H., Warren, R.F., Marshall, J.L., Joseph, A. 1984. Quadriceps atrophy in the anterior cruciate insufficient knee. American Journal of Sports Medicine, 12(3):192-5.

Bergmark, a. 1989. Stability of the lumbar spine: a study in mechanical engineering. Acta Orthopaedica Scandinavica Supplementum. 230:1-54.

Bergman, T. F., Peterson, B. H., Lawrence, D. J. 1993. Principles and procedures. Chiropractic Technique. Churchill Livingstone. Pp113. ISBN 0-44305-752-0.

Boden, N.L. 2002. The effectiveness of spinal manipulative therapy versus spinal manipulative therapy in conjunction with core stabilisation exercises in the treatment of mechanical low back pain. Masters degree in Technology: Chiropractic. Dissertation. Technikon Natal, Berea, Durban, South Africa.

Borenstein, D.G. Wiesel, S.W. and Boden, S.D. 1995. Epidemiology of low back pain and sciatica. In Borenstein, D.G., Wiesel, S.W. and Boden, S.D. ed. Low back pain: Medical diagnosis and comprehensive management. 2<sup>nd</sup> ed. 22 – 27p. Philadelphia: W.B. Saunders Company. 732p. ISBN 0-7216-5411-8.

Burton, C.V., and Cassidy, J.D. 1992. Economics, epidemiology and risk factors. In Kirkaldy-Willis, W.H. Managing Low Back Pain. 3<sup>rd</sup> ed. pp. 1 – 3. New York: Churchill Livingstone Inc. 419p. ISBN 0-443-08789-X.

Cramer, G. D. and Darby, S. A. 1995. Basic and Clinical Anatomy of the Spine, Spinal Cord and ANS. Mosby. ISBN 0-8016-6467- 5.

Cresswell, A.G., Thorstensson, A. 1992. Dynamic responses of intra-abdominal pressure and abdominal muscle activity during trunk loading. Journal of Biomechanics.25(7):738

Crisco, J. J., Panjabi, M. M. 1990. Euler stability of the ligamentous (Part I) and muscular (Part II) lumbar spine. Journal of Biomechanics 23 7): 723

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

Crossman, A.R., Neary, D. 2005. Neuroanatomy: an illustrated colour text.2<sup>nd</sup> Edition. Churchill Livingstone. ISBN 0-443-06216-1.

Dillingham, T. 1995. Evaluation and management of low back pain: and overview. State of the art reviews 9(3):559-574

Docrat, A. 1999. A comparison of the epidemiology of low-back pain in Indian and Coloured communities in South Africa. Masters degree in Technology: Chiropractic. Dissertation. Technikon Natal, Berea, Durban, South Africa.

Eriksson, E. 1991. Rehabilitation of muscle function after sports injury- major problem in sports medicine. International Journal of Sports Medicine, 20:1-6.

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

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

Fitzgerald, M. J. T., Greuner, G. and Mtui, E. 2007. Clinical Neuroanatomy and Neuroscience. 5<sup>th</sup> ed. Elsevier: 193. ISBN 978-1-4160-3445-2

Freeman MA, Wyke B. 1967. Articular reflexes at the ankle joint: an electromyographic study of normal and abnormal influences of ankle-joint mechanoreceptors upon reflex activity in the leg muscles. British Journal of Surgery, 54(12):990-1001

Freiwald, J., Reuter, I. Engelhardt, 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.

Gatterman, M.I. 1995. Subluxation: The articular lesion. In Gatterman, M.I. Foundations of chiropractic subluxation. ed. pp. 11 – 12. Boston, New York. Mosby. 487p. ISBN 0815135432.

Gatterman, M.I. et al. 2001. Rating specific chiropractic technique procedures for common low back conditions. Journal of Manipulative and Physiological Therapeutics, 24(7): 449-456

Guyton, A. C., Hall, J. E. 2005. Textbook of Medical Physiology. 11th Ed. Elsevier. ISBN 0-7216-0240-1.

Haldeman, S. 2005. Principles and Practice of Chiropractic. Third Edition. The McGraw-Hill Medical Publishing Division. ISBN 0-07-137534-1.

Herzog, W; Scheele, D; Conway, P.J. 1999. Electromyographic Responses of Back and Limb Muscles Associated with Spinal Manipulative Therapy. Spine. 24(2): 146-152.

Hills, E.C. 2006. Mechanical Low Back Pain. eMedicine.com, Medscape

Hodges, P.W. 1999. Is there a role for transversus abdominis in lumbo-pelvic stability? Manual Therapy. 4(2):74-86

Hodges, P. W. 2003. Core stability exercise in chronic low back pain. Orthopaedic clinics of North America 34:245

Hodges, P.W. and Richardson, C.A. 1996. Inefficient Muscular Stabilization of the Lumbar Spine Associated with Low Back Pain: A motor control evaluation of transversus abdominis. Spine, 21(22):2640-50

Hodges, P.W., Richardson, C.A. 1997. Contraction of the abdominal muscles associated with movement of the lower limb. Physical Therapy 77:132

Hodges, P.W., Richardson, C.A.1999. Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds. Archives of Physical Medicine, 80(9):1005-12

Hopkins, J.T., Ingersoll, C. D. 2000. AMI the limiting factor. Journal of Sport Rehabilitation, 9(2), 135-159

Hopkins, J.T., Ingersoll, C.D., Edwards, J., Klootwyk, T.E. 2002. Cryotherapy and transcutaneous electric neuromuscular stimulation decrease arthrogenic muscle inhibition of the Vastus Medialis after knee joint effusion. Journal of Athletic Training, 37(1):25-31.

Hurley, M. V., Jones, D. W., Newham, D. J. 1994. Arthrogenic quadriceps

inhibition and rehabilitation of patients with extensive traumatic knee injuries.

Clinical Science, 86:305 – 310.

Indahl, A., Velund, L. and Reikeras, O. 1995. Good prognosis for low back pain when left untampered. Spine 20(4):473-477.

Ingersoll, C.D., Palmieri, R.M., Hopkins, J. T. 2003. A Joint Dilemma [on-line].  
Los Angeles: The Interdisciplinary Journal of Rehabilitation.

Jankowska, E., Lundberg, A. 1981. Interneurons in the spinal cord. Trends in Neuroscience. 230-233.

Jayson, M.I.V. 1992. The Lumbar Spine and Back Pain. 4<sup>th</sup> edition. United Kingdom: Churchill Livingstone. 637p.

Jones, L. A. 1999. Somatic Senses 3: Proprioception. In: Cohen, H. (ed.)  
Neuroscience for Rehabilitation. 2nd ed. Philadelphia: Lippincott, Williams and  
Wilkins. ISBN 0 – 397 – 55465 – 6.

Jull, G., Richardson, C., Toppenberg, R., Comerford, M., Bui, B. 1993. Towards a measurement of active muscle control for lumbar stabilisation. Australian Journal of Physiotherapy 39(3), 187–193

Jull, G.; Richardson, C. and Richardson, B. 1995. Relationship between lower back pain and Test: a dysfunction of deep abdomen muscle exists in lower back pain patients. In: Proceedings World Confederation of Physical Therapists. Washington. pg932.

Jull, G.A., Richardson, C.A. 2000. Motor control problems in patients with spinal pain: a new direction for therapeutic exercise. Journal of Manipulative and Physiological Therapeutics, 23(2):115-7.



Kapandji, I.A., The Physiology of the Joints - Annotated diagrams of the mechanics of the human joints - Volume Three: The Trunk and the Vertebral Column. 2nd ed. Vol. 3. 1974, Edinburgh: Churchill Livingstone.

Koes, B.W., Bouter, L.M., Beckerman *et al.* Physiotherapy exercise and back pain: A blinded review. British Medical Journal. 1991; 30 2: 1572 – 6.

Kirkaldy-Willis, W.H. 1988. Managing Low Back Pain. 2<sup>nd</sup> ed. New York. Churchill and Livingstone. pg 259, 403. ISBN 0-443-08535-8.

Kirkaldy-Willis, W.H., Burton, C.V. And Cassidy, J.D. 1992. *The sight and nature of the lesion*. In: Kirkaldy-Willis, W.H. *Managing low-back pain*. 3<sup>rd</sup> ed. New York: Churchill Livingstone. ISBN 044308789

Lee, D.G. and Vleeming, A. 2003. Principles of the Integrated Model of Function and its Application to the Lumbopelvic-hip Region. The Pelvic Girdle. 3<sup>rd</sup> edition. Elsevier. 5: 1-16.

Levangie, P.K. and Norkin, C.C. 2001. Joint structure and function. 3<sup>rd</sup> edition. Philadelphia: F.A. Davies Company.

Mannion, A.F., Balagué, F., Pellisé, F. and Cedraschi, C. 2007. Pain measurement in patients with low back pain. Nature Clinical Practice Rheumatology (2007) 3, 610-618

Matkovich, G.2004. The immediate effect of sacroiliac manipulation on hip strength in patients suffering from chronic sacroiliac syndrome. Masters degree in Technology: Chiropractic. Dissertation. Technikon Natal, Berea, Durban, South Africa.

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

McLain, R.F., Pickar, J.G. 1998. Mechanoreceptor Endings in Human Thoracic and Lumbar Facet Joints. Spine 23:2 168-173

Moore, K.L. and Agur, A.M.R. 1995. Essential Clinical Anatomy. Baltimore: Lippencott Williams and Wilkins. Pp 82, 83.

Moore, K.L. 1992. Clinically Oriented Anatomy. 3rd edition. Williams and Wilkins. ISBN 0-683-06133-X

Moseley, G.L., Hodges, P.W., Gandevia, S.C. 2002. Deep and superficial fibres of the lumbar multifidus muscle are differentially active during voluntary arm movements. Spine 27(2): 29-36

Mouton, J. Understanding social research. 1996. Pretoria : Van Schaik,. ISBN 0627021638.

Nachemson, A. L., Terenius, L., Bigos, S. J., Maruta, T. and Doxey N. C. S. 1984. Acute and sub acute back pain. Pain. 18:1 96

Ng, J.K., Kippers, V. and Richardson, C.A. 1998. Muscle fibre orientation of abdominal muscles and suggested EMG electrode positions. Electromyographic and Clinical Neurophysiology. 38:51-8

O'Sullivan, P. 2005. Diagnosis and classification of chronic low back pain disorders: Maladaptive movement and motor control impairments as underlying mechanism. Manual Therapy 10 (2005) 242-255

Panjabi, M.M. 1992. The stabilizing system of the spine, Part 1: Neutral zone and instability hypothesis. Journal of Spinal Disorder. 5(4) 383 – 389.

Panjabi, M.M. 1992. The stabilizing system of the spine, Part 2: Neutral zone and instability hypothesis. Journal of Spinal Disorders. 5(4): 390 – 397.

Panjabi, M.M. 2003. Clinical spinal instability and low back pain. Journal of Electromyography and Kinesiology. 13:371-379.

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

Perrin, D. H. 1993. Isokinetic Exercise and Assessment. Champaign: Human Kinetics Publishers. 212p. ISBN 0-87322-464-7.

Porter, J.L., 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.

Pullman,S.L., Good,D.S.,Marquenez, A.I., Tabbal, S., Rubin, M.2000. Clinical Utility of surface EMG. Neurology 55: 171-177

Radebold A, Cholewicki J, Panjabi MM, Patel TC. 2000. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. Spine. 25(8):947-54.

Reid, D. C. 1992. Sports Injury Assessment and Rehabilitation. United States of America. Churchill Livingstone. 606p – 610p. ISBN 0-443-08663-1.

Richardson, C. and Jull, G. 1994. Concepts of assessments and rehabilitation for active lumbar activity. In: Boyling, J.D., Palastanga, N. (eds). Grieve's modern therapy. Manual therapy: The vertebral column. 2<sup>nd</sup> ed. Churchill Livingstone, Edinburgh. Pg 705-720.

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

Rydevik, B. L. 1992. The effects of compression on the physiology of nerve roots. Journal of manipulative and physiological therapeutics.15 (1):62-6.

Schaefer, R.C., Faye, L.J. Principles of Dynamic Chiropractic. Motion Palpation and Chiropractic Technique. 1989, 1<sup>st</sup> ed. pp. 211- 216, 256 – 259.

Simons, D.G., Travell, J.G. and Simons, L.S. 1999. Myofascial Pain and Dysfunction: The Trigger Point. Edition 2:949

Snijders C J, Vleeming A, Stoeckart R 1993a Transfer of lumbosacral load to iliac bones and legs.1: Biomechanics of self- bracing of the sacroiliac joints and its significance for treatment and exercise. Clinical Biomechanics 8:285

Snijders C J, Vleeming A, Stoeckart R 1993b Transfer of lumbosacral load to iliac bones and legs. 2: Loading of the sacroiliac joints when lifting in a stooped posture. Clinical Biomechanics 8:295

Stanford, M.E. 2002. *Effectiveness of specific lumbar stabilization exercises: A single case study*. Journal of Manual and Manipulative Therapy, 10(1): 40 – 46.

Stevens, V.K., Coorevits, P.L., Bouche, K.G., Mahieh, N.N., Vanderstraeten, G.G. and Danneels, L.A. 2006. The Influence of Specific Training on Trunk Muscle Recruitment Patterns in Healthy Subjects during Stabilization Exercises. Manual Therapy. 7: 1-9.

Stokes, M., Young, A. 1984. Investigations of quadriceps inhibition: implications for clinical practice. Physiotherapy, 69:425-432.

Suter, E., McMorland, G., Herzog, W., Bray, R. 1999. Decrease in quadriceps

inhibition after sacroiliac manipulation in patients with anterior knee pain. Journal of Manipulative and Physiological Therapeutics, 23(3):149-153.

Suter, E., McMorland, G., Herzog, W. and Bray, R. 2000. Conservative lower back treatment reduces inhibition in knee-extensor muscles: A randomised controlled trial. Journal of Manipulative and Physiological Therapeutics, 22(3): 149-153.

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: Chiropractic Dissertation. Technikon Natal, Berea, Durban, South Africa.

Vleeming, A., Volkers, A. C. W., Snijders, C. J., Stoeckart R. 1990. Relation between form and function in the sacroiliac joint. 2: Biomechanical aspects. Spine 15(2):133

Waddel, G. 1995. Modern management of spinal disorders. Journal of Manipulative and Physiological Therapeutics 18(9): 590-596

Wheeler, A.H., 2007. Pathophysiology of Chronic Back Pain. eMedicine Neurology, Medscape

Wilke, H.J., Wolf, S., Claes, L.E., Arand, M., Wiesend, A. 1995. Stability of the lumbar spine with different muscle groups: A biomechanical In Vitro study. Spine; 20(2): 192 – 198.

## APPENDIX A

### Letter of Information and Consent

**Title of the Research Study:**

The immediate effects of thoraco-lumbar spinal manipulation compared to lower lumbar spinal manipulation on core muscle endurance and activity in patients with mechanical low back pain.

**Principle Investigator/s:**

**Stuart Murray**

Contact number 083 554 9536

**Co-Investigator/s:**

**Dr. Andrew Jones**

Contact number 031 903 4467

[MTech-Chiropractic]

**Brief Introduction and Purpose of the Study:**

You have been selected to take part in a study investigating the immediate effects of thoraco-lumbar spinal manipulation compared to lower lumbar spinal manipulation on core muscle endurance and activity in patients with mechanical low back pain.

**Outline of the Procedures:**

All participants, including you, will be randomly split into two equal groups. Each of the groups will receive a standard clinical treatment with core muscle training and non invasive testing of core muscles, as well as spinal manipulative therapy.

**Risks or Discomforts to the Subject:**

The treatment is safe and is unlikely to cause any adverse side effects, other than transient tenderness and stiffness that is common in spinal manipulative therapy. Patients may experience post exercise soreness, however this will be transient and the patients are not expected to have prolonged pain / soreness.

**Benefits:**

There will be no remuneration of any sort to the patient.

**Reason/s why the Subject May Be Withdrawn from the Study:**

If subject does not meet inclusion criteria or infringes on the exclusion criteria.

**Remuneration:**

Treatment for the duration of the research process will be free of charge. Subjects taking part in the study will not be offered any other form of remuneration for taking part. Upon completion of the research process, the normal cost of consultations will be charged for those patients wanting further treatment.

**Costs of the Study:**

You will not be liable to any costs what so ever.

**Confidentiality:**

All patient information will be kept confidential and will be stored in the Chiropractic Day Clinic for 5yrs, after which it will be shredded.

All the results of the study will be made available in the Durban University of Technology library in the form of a mini-dissertation, but no personal information will be included.

Please don't hesitate to ask questions on any aspect of this study.

**Research-related Injury:**

No compensation will be made to subject by the researcher, supervisors, DUT Chiropractic Day Clinic or Durban University of Technology.

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

Should you wish you can contact my research supervisor at the above details or alternatively you could contact the Faculty of Health Sciences Research and Ethics Committee as per Mr. Vikesh Singh (031) 2042701.

**Statement of Agreement to Participate in the Research Study:**

(I,.....subject's full name, ID number....., have read this document in its entirety and understand its contents. Where I have had any questions or queries, these have been explained to me by .....to my satisfaction. Furthermore, I fully understand that I may withdraw from this study at any stage without any adverse consequences and my future health care will not be compromised. I, therefore, voluntarily agree to participate in this study.

Subject's name (print) .....Subject's signature:.....

Date:.....

Researcher's name (print): ..... Researcher's signature:.....Date:.....

Witness name (print) signature: .....Witness signature: .....Date:.....

Supervisor's name (print):.....Supervisor's signature: .....Date:.....

**Appendix B**

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

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

File #: \_\_\_\_\_ Age: \_\_\_\_\_

Sex : \_\_\_\_\_ Occupation: \_\_\_\_\_

Intern: \_\_\_\_\_

Signature

**FOR CLINICIANS USE ONLY:**

Initial visit

Clinician: \_\_\_\_\_ Signature : \_\_\_\_\_

**Case History:**

Examination:

Previous: \_\_\_\_\_ Current: \_\_\_\_\_

X-Ray Studies:

Previous: \_\_\_\_\_ Current: \_\_\_\_\_

Clinical Path. lab:

Previous: \_\_\_\_\_ Current: \_\_\_\_\_

**CASE STATUS:**

PTT: \_\_\_\_\_ Signature: \_\_\_\_\_ Date: \_\_\_\_\_

**CONDITIONAL:**

Reason for Conditional:

.....

.....

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



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

**Intern's Case History:**

1. **Source of History:**
2. **Chief Complaint : (patient's own words):**
3. **Present Illness:**

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

4. **Other Complaints:**
5. **Past Medical History:**
  - < General Health Status
  - < Childhood Illnesses

- < Adult Illnesses
- < Psychiatric Illnesses
- < Accidents/Injuries
- < Surgery
- < Hospitalisations

**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 C

Durban Institute of Technology

**PHYSICAL EXAMINATION: SENIOR**

Patient Name : \_\_\_\_\_

File no : \_\_\_\_\_

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

Student : \_\_\_\_\_

Signature : \_\_\_\_\_

**VITALS:**

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

**GENERAL EXAMINATION:**

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

**SYSTEM SPECIFIC EXAMINATION:**

CARDIOVASCULAR EXAMINATION

RESPIRATORY EXAMINATION

ABDOMINAL EXAMINATION

NEUROLOGICAL EXAMINATION

COMMENTS

Clinician: \_\_\_\_\_

Signature : \_\_\_\_\_

## APPENDIX D

## THORACIC SPINE REGIONAL EXAMINATION

Patient: \_\_\_\_\_ File: \_\_\_\_\_ Date: \_\_\_\_\_

Intern: \_\_\_\_\_ Signature: \_\_\_\_\_

Clinician: \_\_\_\_\_ Signature: \_\_\_\_\_

**STANDING:**

 Posture ( incl. L/S & C/S )  
 Muscle tone

 Scars  
 Chest deformity

Skyline view – Scoliosis

(pigeon, funnel, barrel)

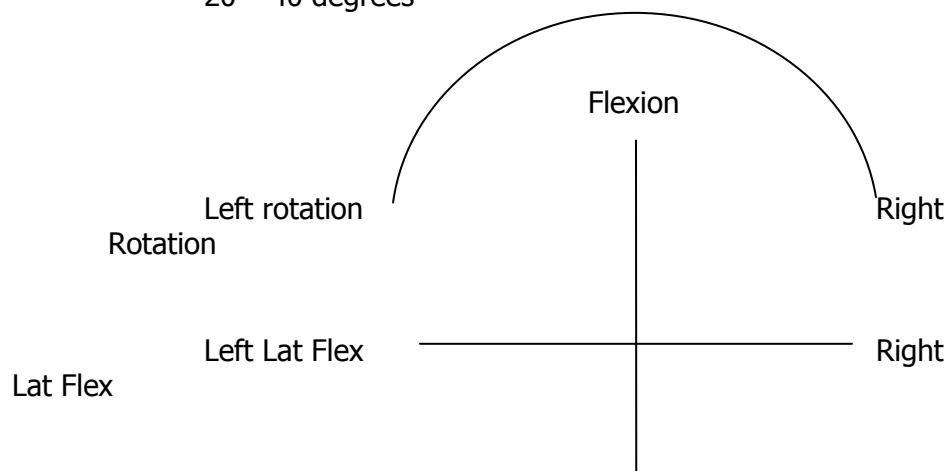
Spinous Percussion

Breathing (quality, rate, rhythm, effort)

Deep Inspiration

**RANGE OF MOTION:**

Forward Flexion	20 – 45 degrees (15cm from floor)
Extention	25 – 45 degrees
L/R Rotation	35 – 50 degrees
L/R Lat Flex	20 – 40 degrees





Reflexes	
----------	--

**KEMP'S TEST:****MOTION PALPATION:**

		Right	Left
Thoracic Spine			
Ribs	Calliper (Costo-transverse joints)		
	Bucket Handle	Opening	
		Closing	
Lumbar Spine			
Cervical Spine			

BASIC EXAM	History	ROM	Neuro/Ortho
LUMBAR			
CERVICAL			

**APPENDIX E**



**REGIONAL EXAMINATION - LUMBAR SPINE AND PELVIS**

Patient: \_\_\_\_\_ File#: \_\_\_\_\_ Date: \_\_\_/\_\_\_/\_\_\_

Intern\Resident: \_\_\_\_\_ Clinician: \_\_\_\_\_

**STANDING:**

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

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

**GAIT:**

Normal walking  
 Toe walking  
 Heel Walking  
 Half squat

R. Rot

**ROM:**

**Forward Flexion = 40-60° (15 cm from floor)**

Extension = 20-35°

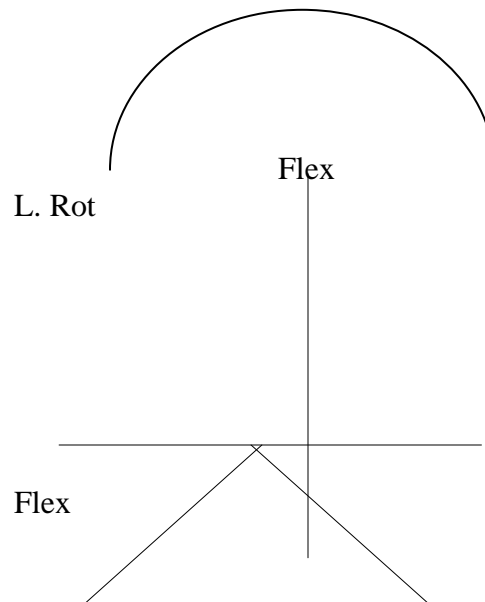
L/R Rotation = 3-18°

L.Lat

R.Lat

L/R Lateral Flexion = 15-20°

Flex



**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

Ext.

<b>SLR</b>		Degre e	LBP?	Locati on	Leg pain	Butto ck	Thigh	Calf	Heel	Foot	Bragg ard
------------	--	------------	------	--------------	-------------	-------------	-------	------	------	------	--------------

	<b>L</b>										
	<b>R</b>										

	<b>L</b>	<b>R</b>
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

<b>TRIPOD</b>		Degree	<b>LBP?</b>	<b>Location</b>	<b>Leg pain</b>	<b>Buttock</b>	<b>Thigh</b>	<b>Calf</b>	<b>Heel</b>	<b>Foot</b>	<b>Braggard</b>
		<b>L</b>									
	<b>R</b>										
SI, +, ++											

Slump 7 test											
		<b>L</b>									
	<b>R</b>										

**LATERAL RECUMBENT:****L****R**

	<b>L</b>	<b>R</b>
<b>Ober's</b>		
<b>Femoral n. stretch</b>		
SI Compression		

**PRONE:****L****R**

	<b>L</b>	<b>R</b>
Gluteal skyline		

Skin rolling		
Iliac crest compression		
Facet joint challenge		
SI tenderness		
SI compression		
Erichson's		
Pheasant's		

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

**NON ORGANIC SIGNS:**

Pin point pain  
 Burn's Bench test  
 Hoover's test

Axial compression  
 Trunk rotation  
 Flip Test  
 Ankle dorsiflexion 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						

Repeat Pin point test

MYOTOMES						
Action	Muscles	Levels	L	R		
Lateral Flexion spine	Muscle QL	T12-L4				
Hip flexion	Psoas, Rectus femoris	L1,2,3,4				5+ Full strength
Hip extension	Hamstring, glutes	L4,5;S1. 2				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,	L4,5;S1				
Knee extension	Quad	L2,3,4				W – wasting
Ankle plantarflex	Gastroc, soleus	S1,2				
Ankle dorsiflexion	Tibialis anterior	L4,5				
Inversion	Tibialis anterior	S1				
Eversion	Peroneus longus	L4				
Great toe extens	EHL	L5				

### **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 F**

DURBAN UNIVERSITY OF TECHNOLOGY

**Patient Name:**

**File #:**

**Page:**

<i>Date:</i>	<i>Visit:</i>	<i>Intern:</i>	
<i>Attending Clinician:</i>			<i>Signature:</i>
<i>S: Numerical Pain Rating Scale (Patient)</i>		<i>Intern Rating</i>	<i>A:</i>
<i>Least 0 1 2 3 4 5 6 7 8 9 10 Worst</i>			
<i>O:</i>			<i>P:</i>
<i>E:</i>			
<i>Special attention to:</i>		<i>Next appointment:</i>	
<i>Date:</i>	<i>Visit:</i>	<i>Intern:</i>	
<i>Attending Clinician:</i>			<i>Signature:</i>
<i>S: Numerical Pain Rating Scale (Patient)</i>		<i>Intern Rating</i>	<i>A:</i>
<i>Least 0 1 2 3 4 5 6 7 8 9 10 Worst</i>			
<i>O:</i>			<i>P:</i>
<i>E:</i>			
<i>Special attention to:</i>		<i>Next appointment:</i>	
<i>Date:</i>	<i>Visit:</i>	<i>Intern:</i>	
<i>Attending Clinician:</i>			<i>Signature</i>

## APPENDIX G

### Pain Rating

**Patient Name:**

**Date:**

**Pain Numerical Rating Scale:**

Please indicate on the line below the number between 0 and 100 that best describes your pain. A zero (0) would mean 'no pain' and a one hundred (100) would mean 'pain as bad as it could be'. Please write only one number.

No pain \_\_\_\_\_ Excruciating pain  
0 100  
(Mannion *et al*, 2007)

## APPENDIX H

Patient Name:  
Race:

Date of visit:

Occupation:

Age:

File no:

**Prone test for transversus abdominis and internal oblique:**

Reading	Visit	Time	mmHg
1	1		
2	1		

**NRS Pain Rating Scale**

Reading	Value	Reading	Value
1		2	

**EMG Readings of Obliques Internus**

Reading	Millivolts		Reading	Millivolts	
	Left	Right		Left	Right
1	1.	1.	2	1.	1.
	2.	2.		2.	2.
	3.	3.		3.	3.

Ave.

**APPENDIX I****Core stability assessment tests: The Stabilizer Biofeedback Device**

### **1. Testing for the presence of core stability activation:**

In accordance with Richardson *et al.* (1999), before formal testing begins participants were taught to recruit transversus abdominis in four-point kneeling. This position provided a facilitated stretch to the deep abdominals resulting from the forward drift of the abdominal contents. This stretch leads to an inhibitory effect on the superficial muscles, particularly rectus abdominis (Richardson & Jull 1995).

When this ability was recognized to be present, participants were then instructed to lie prone on a chiropractic table with their head turned to one side. The Stabilizer Biofeedback Device was placed under their abdomen, with the centre at the navel and the distal edge at the anterior superior iliac spine (ASIS). It was then inflated to the baseline pressure of 70 mmHg.

Participants were then examined as to whether they could initiate transversus abdominis activation in this prone position. A drop in pressure of 6-8 mmHg was seen with a correct contraction.

This test was performed at the initial consultation. It was noted yes/no, for statistical purposes, as to whether the subject could perform a correct activation of transversus abdominis.

If the subject could not do this, the subject was retrained in the four point kneeling and prone positions to perform this activation satisfactorily, prior to taking the quantitative time-based readings.

If the subject still could not manage a satisfactory activation, the subject was instructed to perform a contraction of transversus abdominis, as trained by the researcher, to the best of their ability and a time-based reading of this contraction was taken for the prone and supine positions.

### **2. The prone abdominal draw in test test for transversus abdominis and internal oblique:**



- A 3-chamber pressure cell was placed centrally under the abdomen, with the umbilicus in the centre of the inflatable sleeve, and inflated to a baseline of 70 mmHg.
- The subject was then instructed to draw the abdominal wall up and in without moving the spine or pelvis.
- The pressure reading should have decreased by 6-10 mmHg.
- A variation of 2 mmHg was allowed for normal breathing pattern.
- A measurement was taken of the time at which the patient could no longer hold the contraction at the baseline level (70mmHg – 6 to 10 mmHg).

## APPENDIX J

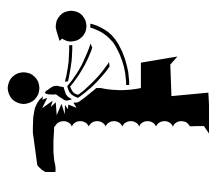
DO YOU SUFFER FROM

# LOW BACK PAIN

and are between the ages of 18-45

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 further information contact

Stuart on:  
3732205 / 2512  
or 083 554 9536

**APPENDIX K**

52 Klooflands Rd  
Kloof  
3610

The Faculty of Health Sciences  
Research and Ethics Committee  
Durban University of Technology  
Steve Biko Campus  
Durban  
4 September 2008

**Re: Stuart Murray's Research**

Dear Committee members

I have been consulted by Mr Murray regarding required sample size for his study. Since this is the first study of this specific intervention in this population, there is no information from previous studies available on which to base sample size calculations. Thus no formal sample size calculations were performed for this study. This is an exploratory study with objectives to assess trends in efficacy using the proposed intervention compared with the standard of care.

Therefore Mr Murray proposes to use 30 participants in total based on the sample sizes other studies have used. Time and logistical constraints also limit the sample size to this number. It is not guaranteed that statistical significance will be achieved with this sample size, but based on reasons given previously this is the most feasible sample size which would allow Mr Murray to achieve his objectives.

Yours sincerely



Tonya Esterhuizen (Mrs)

## APPENDIX L

### **Four point kneeling: training of the transversus abdominis**

As indicated by Evans and Oldrieve (2000), as described by Jull (1995).

The patient was positioned with their shoulder directly over the hands and the hips over the knees. The examiner's hand was placed under the lower abdomen and the following was asked of the patient, "As you breathe out, gently draw your lower abdomen off my hand and maintain this position while breathing normally." This position was used, as the forward shift of the abdominal contents provides a facilitatory stretch of the deep abdominals, but provides an inhibitory effect for the superficial muscle, the rectus abdominis (Jull, 1995)

## APPENDIX M

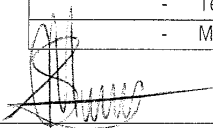
### ETHICS CLEARANCE CERTIFICATE

Student Name	<b>Stuart Murray</b>	Student No	<b>20300550</b>
Ethics	FHSEC 046/08	Date of FRC Approval	
Research Title:	The immediate effects of thoraco-lumbar spinal manipulation compared to lower lumbar spinal manipulation on core muscle endurance and activity in patients with mechanical low back pain.		

*In terms of the ethical considerations for the conduct of research in the Faculty of Health Sciences, Durban University of Technology, this proposal meets with Institutional requirements and confirms the following ethical obligations:*

1. The researcher has read and understood the research ethics policy and procedures as endorsed by the Durban University of Technology, has sufficiently answered all questions pertaining to ethics in the DUT 186 and agrees to comply with them.
2. The researcher will report any serious adverse events pertaining to the research to the Faculty of Health Sciences Research Ethics Committee.
3. The researcher will submit any major additions or changes to the research proposal after approval has been granted to the Faculty of Health Sciences Research Committee for consideration.
4. The researcher, with the supervisor and co-researchers will take full responsibility in ensuring that the protocol is adhered to.
5. **The following section must be completed if the research involves human participants:**


	YES	NO	N/A
❖ Provision has been made to obtain informed consent of the participants	X		
❖ Potential psychological and physical risks have been considered and minimised	X		
❖ Provision has been made to avoid undue intrusion with regard to participants and community	X		
❖ Rights of participants will be safe-guarded in relation to:	X		
- Measures for the protection of anonymity and the maintenance of Confidentiality.			
- Access to research information and findings.	X		
- Termination of involvement without compromise	X		
- Misleading promises regarding benefits of the research	X		

  
SIGNATURE OF STUDENT/RESEARCHER

31/10/2008  
DATE

  
SIGNATURE OF SUPERVISOR/S

31/10/2008  
DATE

  
SIGNATURE OF HEAD OF DEPARTMENT

28/10/08  
DATE

  
SIGNATURE: CHAIRPERSON OF RESEARCH ETHICS COMMITTEE

28-10-08  
DATE